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**AN EVALUATION OF THE  
EFFECTIVENESS OF  
CASTELLATED LARYNGOFISSURE  
AND BILATERAL  
ARYTENOID LATERALISATION  
FOR THE RELIEF OF LARYNGEAL  
PARALYSIS IN DOGS**

**A Thesis presented for  
The Degree of Master of Veterinary Science  
at Massey University**

**by**

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## ABSTRACT

In recent years, laryngeal paralysis in dogs, has become a frequently recognised disorder. Various surgical procedures have been advocated to relieve the consequential laryngeal obstruction, but few critical examinations of the effectiveness of these procedures have been attempted. The aim of this study was to determine and compare the efficacy of two of the more commonly used surgical methods (i) castellated laryngofissure with vocal fold resection and (ii) bilateral arytenoid lateralisation in providing an adequate laryngeal airway in dogs after surgically induced laryngeal paralysis.

The laryngeal function of ten adult healthy experimental dogs was assessed by clinical examination, laryngoscopy, blood gas and tidal breathing flow-volume loop analyses prior to and after bilateral recurrent laryngeal nerve resection. Five dogs then underwent a castellated laryngofissure with vocal fold resection and the remaining five, bilateral arytenoid lateralisation. Six weeks later, the laryngeal function of the ten dogs was reassessed using the same investigative techniques. Radiographs of the chest were taken at the start and completion of the experiment on the live dogs to help detect the presence of any co-existent lower respiratory tract disease. An autopsy was performed on each dog, six weeks after surgery, and details of visible changes in the laryngeal structure recorded.

After neurectomy, all of the dogs had inspiratory stridor, a hoarse bark and reduced exercise tolerance. Medial displacement of the arytenoid cartilages and vocal cords caused narrowing of the *rima glottidis*. No abduction of these structures occurred during inspiration. There was a significant fall in arterial oxygen tension and inspiratory airflow recorded on blood gas analyses and tidal breathing flow-volume loop studies, respectively.

The degree of relief from the laryngeal obstruction in the neurectomised dogs after castellated laryngofissure with vocal fold resection was variable. Some of the animals still had signs of respiratory embarrassment. The size of the *rima glottidis* achieved was either slightly narrower or similar to that seen in the non-abducted, pre-neurectomised larynx of the experimental dogs. No significant improvement in blood gas or tidal breathing flow-volume loop analyses were recorded following the surgical technique.

In contrast, bilateral arytenoid lateralisation consistently alleviated the clinical signs of bilateral recurrent laryngeal neurectomy. The *rima glottidis* width was increased to either the non-abducted, pre-neurectomised size or greater, depending on the position of the lateralisation suture. Furthermore, blood gas and tidal breathing flow-volume analyses recorded a significant increase in arterial oxygen tension and inspiratory airflow, respectively.

Although bilateral arytenoid lateralisation was more efficient than castellated laryngofissure with vocal fold resection in alleviating the laryngeal obstruction caused by bilateral recurrent laryngeal neurectomy, it did have some inadequacies. The fixed abduction of the arytenoid cartilages destroyed the expiratory braking mechanism of the larynx. Furthermore, execution of the surgical technique requires intimate knowledge of the anatomy of the larynx and the surrounding structures and, since the position of the lateralisation suture is important, best results are likely to be achieved by surgeons experienced with the procedure.

## TABLE OF CONTENTS

	Page
<b>Acknowledgements</b>	i
<b>Abstract</b>	ii
<b>Table of Contents</b>	iv
<b>List of Tables</b>	vii
<b>List of Figures</b>	viii
<b>Chapter I.    <u>INTRODUCTION</u></b>	1
I <sub>1</sub> . <u>Laryngeal Paralysis - the disease</u>	1
i)       Clinical signs	
ii)      Aetiology	
I <sub>2</sub> . <u>Anatomy</u>	4
i)       Cartilages	
ii)      Laryngeal mucosa and associated structures	
iii)     Laryngeal muscles	
iv)      Laryngeal nerves	
I <sub>3</sub> . <u>Physiology</u>	17
i)       Action during respiration	
ii)      Action during swallowing	
iii)     Action during phonation	
I <sub>3a</sub> .      i)       The paralysed larynx	20
I <sub>4</sub> . <u>Laryngeal Paralysis - diagnostic techniques</u>	23
i)       Laryngoscopy	
ii)      Blood gas analysis	
iii)     Tidal breathing flow-volume loops	
iv)      Upper airway pressure flow curves	
v)       Electromyography	
vi)      Palpation	
vii)     Radiography	
viii)    Histopathology	
ix)      Histochemistry	
x)       Nerve transmission studies	

	<b>Page</b>
I <sub>5</sub> . <u>Laryngeal Paralysis - a review of surgical techniques</u>	30
i)      Partial laryngectomy	
ii)     Arytenoid lateralisation	
iii)    Castellated laryngofissure and vocal fold resection	
iv)    Modified castellated laryngofissure	
v)    Neuromuscular pedicle graft	
<b>Chapter II.      <u>MATERIALS AND METHODS</u></b>	<b>34</b>
II <sub>1</sub> . <u>Investigative Procedures</u>	34
i)      Radiography	
ii)     Laryngoscopy	
iii)    Blood gas analysis	
iv)    Tidal breathing flow-volume loop analysis	
II <sub>2</sub> . <u>Surgical Procedures</u>	39
i)      Bilateral recurrent laryngeal nerve resection	
ii)     Castellated laryngofissure with vocal fold resection	
iii)    Bilateral arytenoid lateralisation	
II <sub>3</sub> . <u>Experimental Procedure</u>	58
II <sub>4</sub> . <u>Statistical Analysis</u>	59
<b>Chapter III.      <u>RESULTS</u></b>	<b>60</b>
III <sub>1</sub> . <u>Clinical Assessment</u>	60
III <sub>2</sub> . <u>Radiography</u>	61
III <sub>3</sub> . <u>Laryngoscopy</u>	61
III <sub>4</sub> . <u>Blood Gas Analysis</u>	69
III <sub>5</sub> . <u>Tidal Breathing Flow-volume Loop Analysis</u>	72
III <sub>6</sub> . <u>Autopsy Examination</u>	89

	<b>Page</b>
<b>Chapter IV.     <u>DISCUSSION</u></b>	<b>94</b>
IV <sub>1</sub> . <u>The effects of bilateral recurrent laryngeal nerve resection</u>	94
IV <sub>2</sub> . <u>The efficacy of castellated laryngofissure with vocal fold resection in alleviating the effects of experimentally induced laryngeal paralysis</u>	97
IV <sub>3</sub> . <u>The efficacy of bilateral arytenoid lateralisation in alleviating the effects of experimentally induced laryngeal paralysis</u>	98
IV <sub>4</sub> . <u>Evaluation of the diagnostic techniques used</u>	99
i)     Laryngoscopy	
ii)    Radiography	
iii)   Blood gas Analysis	
iv)    Tidal breathing flow-volume loop analysis	
v)     Autopsy examination	
IV <sub>5</sub> . <u>Critical evaluation of the experiment</u>	103
<b><u>Chapter V.     CONCLUSION</u></b>	<b>105</b>
<b><u>REFERENCES</u></b>	<b>106</b>

## LIST OF TABLES

	<b>Page</b>
<b>Table 1.</b> Result of blood gas analyses from dogs undergoing castellated laryngofissure and vocal fold resection.	70
<b>Table 2.</b> Result of blood gas analyses from dogs undergoing bilateral arytenoid lateralisation.	71
<b>Table 3.</b> Mean values for tidal breathing flow-volume loops from dogs after castellated laryngofissure and vocal fold resection.	74
<b>Table 4.</b> Indices of tidal breathing flow-volume loops from dogs after castellated laryngofissure and vocal fold resection.	75
<b>Table 5.</b> Mean values for tidal breathing flow-volume loops from dogs after bilateral arytenoid lateralisation.	76
<b>Table 6.</b> Indices of tidal breathing flow-volume loop from dogs after bilateral arytenoid lateralisation.	77

## LIST OF FIGURES

	Page
<b>Figure 1.</b> Lateral view of the laryngeal cartilages.	5
<b>Figure 2.</b> Sagittal section through the larynx showing cartilages and their associated structures.	7
<b>Figure 3.</b> Cranial view of the normal larynx.	8
<b>Figure 4.</b> Dorsal aspect of the larynx showing the intrinsic laryngeal muscles.	10
<b>Figure 5.</b> Lateral aspect of the larynx with the left lamina of the thyroid cartilage, cut and reflected, to show the intrinsic laryngeal muscles.	11
<b>Figure 6.</b> Schematic drawing of medulla with <i>nucleus ambiguus</i> and its efferent neurons, the cervical spinal cord with external branch of accessory nerve and the vagus.	14
<b>Figure 7.</b> Lateral aspect of larynx showing path of cranial laryngeal and recurrent laryngeal nerves.	15
<b>Figure 8.</b> Dorsal aspect of larynx showing path of cranial laryngeal and recurrent laryngeal nerves.	16
<b>Figure 9.</b> Diagrammatic representation of A) the normal larynx, and B) the effects of left recurrent laryngeal nerve paralysis and C) the effects of left cranial laryngeal and recurrent laryngeal nerve paralysis.	21
<b>Figure 10.</b> Maximum flow-volume loop of a healthy adult human showing typical expiratory and inspiratory tracings.	25
<b>Figure 11.</b> Characteristic flow-volume loops produced by major airway lesions A) a fixed lesion B) a variable extrathoracic lesion and C) a variable intrathoracic lesion.	25
<b>Figure 12.</b> Characteristic tidal breathing flow-volume loops of a healthy dog showing peak expiratory flow (PEF), peak inspiratory flow (PIF), midtidal expiratory flow ( $EF_{50}$ ), midtidal inspiratory flow ( $IF_{50}$ ), and expiratory and inspiratory flow at 25% of the tidal volume remaining to be exhaled ( $EF_{25}$ and $IF_{25}$ respectively).	26

	<b>Page</b>
<b>Figure 13.</b> Photograph of one of the experimental dogs with rubber mask and pneumotachograph placed over the muzzle.	36
<b>Figure 14.</b> Photograph of multichannel recorder displaying the flow and volume signals.	36
<b>Figure 15.</b> Photograph of X-Y plotter displaying tidal breathing flow-volume loops.	37
<b>Figure 16.</b> Experimental arrangement used to obtain tidal breathing flow-volume loops in conscious dogs.	38
<b>Figures 17 - 24a</b> Sequential views of surgical procedure for castellated laryngofissure and vocal fold resection.	42-47
<b>Figures 25 - 35</b> Diagrams showing surgical procedure of bilateral arytenoid lateralisation.	49-56
<b>Figures 36 - 45</b> Photographs of larynges taken at endoscopic examination of the ten experimental dogs.	62-68
<b>Figures 46 - 55</b> Tidal breathing flow-volume loops of the ten experimental dogs.	78-88
<b>Figure 56</b> Photograph of larynx from cadaver after castellated laryngofissure demonstrating asymmetry of the <i>rima glottidis</i> . The ventral portion of the laryngeal opening is deviated to the left.	91
<b>Figure 57</b> Photographs of two trachea rings illustrating the effects of tracheostomy on the tracheal shape. A) the site of the tracheostomy shows narrowing and change in shape when compared to the normal tracheal lumen, B) immediately distal to the tracheostomy site.	91

		<b>Page</b>
<b>Figure 58</b>	Photographs of oral view of larynx from cadaver after bilateral arytenoid lateralisation showing symmetrically positioned arytenoid cartilages.	92
<b>Figure 59</b>	Photograph of lateral view from cadaver after bilateral arytenoid lateralisation showing caudal position of lateralisation suture (arrow).	92
<b>Figure 60</b>	Photograph of larynx from cadaver after bilateral arytenoid lateralisation showing asymmetry of the <i>rima glottidis</i> .	93
<b>Figure 61</b>	Photograph of larynx from cadaver after bilateral arytenoid lateralisation showing rostral position of lateralisation suture (arrow).	93

## CHAPTER I. INTRODUCTION

Within the last decade, the number of reported cases of laryngeal paralysis in dogs has increased markedly. This may be due to an actual rise in the incidence of this condition or, more likely, due to greater recognition of the disease by veterinarians. The symptoms of laryngeal paralysis make the condition easy to confuse with lower respiratory tract or cardiac disease. In its mild form, many dogs, particularly those that are sedentary, show few symptoms and the diagnosis of laryngeal paralysis may be overlooked by all, but the most discerning practitioner.

Co-inciding with the recent increase in interest in laryngeal paralysis, has been the development of a number of operations designed to alleviate the laryngeal obstruction. There have been few critical analyses of the effectiveness of these procedures. The purpose of this study is to determine and compare the efficacy of two of the more common surgical techniques, in use at this time.

### I<sub>1</sub>. LARYNGEAL PARALYSIS - THE DISEASE

The recognition that laryngeal paralysis occurred in dogs, began with the description of the clinical features of vocal cord paresis (Cook, 1964; Leonard, 1971; Baker, 1972). These symptoms consisted of inspiratory dyspnoea, low exercise tolerance, altered bark and episodes of collapse. Theories postulated for the cause of this syndrome were flaccidity of the epiglottis (Cook, 1964) and trauma (Baker, 1972). The first report correlating neurogenic laryngeal muscle atrophy in dogs suffering from vocal cord paresis was by O'Brien, Harvey, Kelly and Tucker (1973).

Laryngeal paralysis in dogs is now known to have both an hereditary and an acquired form. The hereditary form exists in Bouvier des Flandres (Venker-van Haagen, Bouw, and Hartman, 1981) and is presumed to exist in Siberian Huskies (O'Brien and Hendricks, 1986) and Dalmations (Braund, Steinberg, Shores, Steiss, Mehta, Toivio-Kinnucan and Amling, 1989) as either a unilateral or bilateral disease. Males appear to be more commonly affected (Venker-van Haagen, Hartman and Goedegebuure, 1978) and the age of onset varies between three months and three years.

The acquired form of laryngeal paralysis is usually found in dogs aged between seven and 10 years (Harvey and O'Brien, 1982; Gaber, Amis and Le Couteur, 1985; Greenfield, 1987). Large dogs, such as St. Bernards, Labrador retrievers, and Afghan hounds appear to be more susceptible, but the disease has also been reported in mixed and medium to small breeds (Harvey and O'Brien, 1982; Gaber *et al*, 1985). Initially, it was reported that laryngeal

paralysis affected both male and female dogs in equal proportions (Harvey and O'Brien, 1982), but later reports (Gaber *et al*, 1985; Greenfield, 1987; White, 1989) showed that males were approximately three times more likely to be affected by the condition than females.

i) Clinical Signs

Early clinical signs of laryngeal paralysis include an altered bark, gagging, coughing, and vomiting (O'Brien *et al*, 1973; Venker-van Haagen *et al*, 1978; Harvey, Irby, Watrous, 1983). Most dogs, however, are presented late in the course of the disease and have a respiratory distress syndrome characterised by loss of endurance, laryngeal stridor and inspiratory dyspnoea (Baker, 1972; O'Brien *et al*, 1973; Harvey and Venker-van Haagen, 1975; White, 1989). The dog, if stressed, can become cyanotic and even collapse with complete airway obstruction. There is often an increased respiratory rate at rest and referred upper airway sounds can be auscultated over the thoracic region (Gaber *et al*, 1985). In the acquired form of the disease, the symptoms appear to be more gradual in onset and progression (Greenfield, 1987). Working and racing dogs may present with reduced racing performance in unilateral disease and airway obstruction in bilateral disease (O'Brien and Harvey 1983). Pet dogs appear to be able to cope with some laryngeal dysfunction unless it is severe or they are stressed by circumstances such as exercise, excitement or hyperthermia (Harvey *et al*, 1983).

It has been reported that some dogs with hereditary and acquired forms of laryngeal paralysis also develop pelvic limb weakness and "foot drop" associated with bilateral cranial tibial muscle denervation (O'Brien, *et al*, 1973; Venker-van Haagen *et al*, 1978; Gaber *et al*, 1985). More recently, laryngeal paralysis has been described as a clinical manifestation of a more generalised polyneuropathy (Braund *et al*, 1989) with generalised weakness in all limbs and myotatic hyporeflexia. Other abnormalities, reported to occur in association with laryngeal paralysis, are megaesophagus, bronchopneumonia, narrowing or dilatation of the extrathoracic trachea (Gaber *et al*, 1985), gastro-oesophageal reflux and hiatus hernia (Burnie, Simpson and Corcoran, 1989).

ii) Aetiology

The hereditary form of laryngeal paralysis in Bouvier des Flandres has been studied in some detail by Venker-van Haagen and his co-workers (1981) who, on the basis of breeding experiments, demonstrated that the disease is transmitted by an autosomal dominant gene(s). Venker-van Haagen *et al* (1978) also described histological evidence of neurogenic atrophy in one or both dorsal cricoarytenoid muscles in affected animals and demonstrated axonal degeneration of right and left recurrent laryngeal nerves. These changes were accompanied by an increase in endoneural connective tissue and number of Schwann cells,

which were present throughout the entire length of the nerves, suggesting that the primary lesion was neuronal in origin. Further studies related the syndrome to an inherited degenerative process in the *nucleus ambiguus* (Venker-van Haagen, 1980).

The exact cause of the acquired form of laryngeal paralysis is unknown. Biopsies of intrinsic laryngeal muscles affected have shown evidence of neurogenic atrophy (O'Brien *et al*, 1973; Reinke and Suter, 1978; Love, Waterman and Lane, 1987). Furthermore, electromyographic studies of the dorsal cricoarytenoid muscle demonstrated denervation potentials (Harvey and O'Brien, 1982), indicating that the recurrent laryngeal nerves are probably involved in the disease process.

Hypothyroidism has been considered to play a role in the aetiology of this form of laryngeal paralysis but further evidence is required to link the two diseases. Harvey *et al* (1983) in one report cited nine out of ten affected dogs studied had low serum thyroxine levels. A later report by Gaber *et al* (1985), however, found only three out of 11 dogs with laryngeal paralysis to have primary hypothyroidism on the basis of a low thyroid stimulating hormone (TSH) response or low serum thyroxine level. Since the latter tests have been shown to be relatively insensitive for a diagnosis of canine hypothyroidism (Jacobs, Lumsden and Willet, 1987), the relationship between hypothyroidism and laryngeal paralysis must remain conjectural.

Whilst it is known that idiopathic, acquired, laryngeal paralysis involves disease of the recurrent laryngeal nerves, it is not known for certain if this disease is restricted to those nerves, or if it is part of a more generalised peripheral nerve disease, or of central nervous origin. Recently, it has been suggested (Braund *et al*, 1989) that both the congenital and acquired forms are just one clinical sign of a more generalised polyneuropathy which affects mainly the distal motor axons and has a particular predilection for the recurrent laryngeal or vagus nerves. The distribution of the electrophysiologic abnormalities detected by these authors suggests a dying-back neuropathy characterised by axonal degeneration that selectively involves the distal part of long and large diameter nerve fibres, with a slow proximal spread of nerve fibre break down with time (Cavanagh, 1964).

Other causes of laryngeal paralysis, which should be excluded before a diagnosis of idiopathic, acquired, laryngeal paralysis can be made, are: known causes of peripheral nerve damage such as neck trauma or previous surgery, intrathoracic or extrathoracic masses involving or compressing the recurrent laryngeal nerves, neoplasia involving the vagus or recurrent laryngeal nerves, posterior brain stem masses or trauma, Addisons disease and organophosphate poisoning (Reinke and Suter, 1978; Schaer, Zaki, Harvey and O'Reilly, 1979; Hardie, Kolata, Stone and Steiss, 1981).

## I<sub>2</sub>. ANATOMY

Descriptions of the anatomy of the dog's larynx are contained in standard text books (Bradley, 1935; Miller, Christensen and Evans 1964) and scientific papers (Wykes, 1983; Greenfield, 1987). A precis of the anatomy is included here since a sound knowledge of the arrangement and inter-relationship of parts of the larynx is essential to accomplish the surgical techniques required to relieve the effects of laryngeal paralysis.

The larynx is attached cranially to the hyoid apparatus by the articulation of the thyroid cartilage to the thyroid cornu of the hyoid bone. Caudally, the cricoid cartilage is attached to the first tracheal ring by fibroelastic tissue. Extrinsic muscles attached to the cartilages and hyoid bones serve to stabilise the larynx and alter its position.

### i) Cartilages

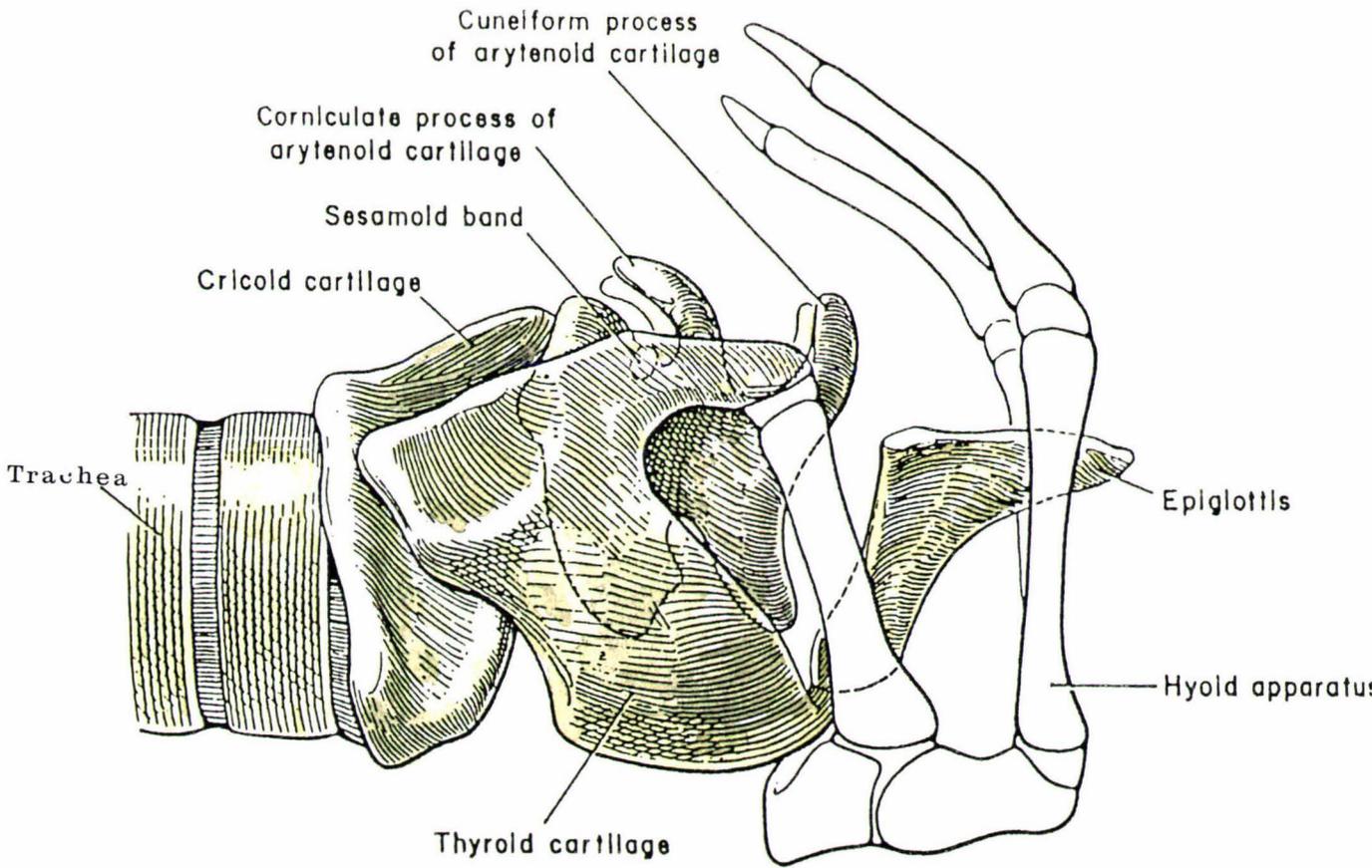
The larynx is composed of five main cartilages (see Figure 1.) - the epiglottis, the thyroid, the cricoid and the paired arytenoids.

The epiglottic cartilage forms the most rostral portion of the larynx. The shape of the cartilage resembles a sharp pointed spade with the apex pointing rostrally. It has two surfaces, a concave, dorsocaudal or aboral surface and a ventral, oral surface. The oral surface is attached to the middle of the body of the hyoid by the hyoepiglottic muscle. The mid caudoventral portion of the epiglottis forms a stalk, and is attached by the thyroepiglottic ligament to the ventro-rostral surface of the thyroid cartilage. This, in conjunction with the elasticity of the epiglottis itself, allows the cartilage to move freely.

The thyroid cartilage is the largest laryngeal cartilage. It consists of left and right laminae fused ventrally to form a U shape. Each lamina is expanded dorsally to form the cranial and caudal cornua. The cranial border is joined to the thyroid cornu of the hyoid bone by the hyo-thyroid membrane and the caudal border is united to the ventral arch of the cricoid cartilage by the cricothyroid ligament.

The cricoid cartilage (the most caudal cartilage) is a complete ring, the dorsal portion (dorsal lamina) being about five times larger than the ventral portion (arch). It bears two pairs of articular facets which are true synovial joints (i) the cricothyroid articulation situated caudolaterally and (ii) the cricoarytenoid articulation situated rostrolaterally. Movement of the latter articulation consists of a sliding and rotational mechanism that brings the two arytenoid cartilages closer together or carries them further apart. The thyroid cartilage partially overlaps the cricoid cartilage laterally.

**Figure 1.** Lateral view of the laryngeal cartilages.



The paired arytenoid cartilages form the rostradorsal limit of the larynx and lie medial to the thyroid laminae. They are frequently joined together dorsally by a connective tissue band which may contain a sesamoid cartilage. In the dog, the arytenoid cartilage has four processes:

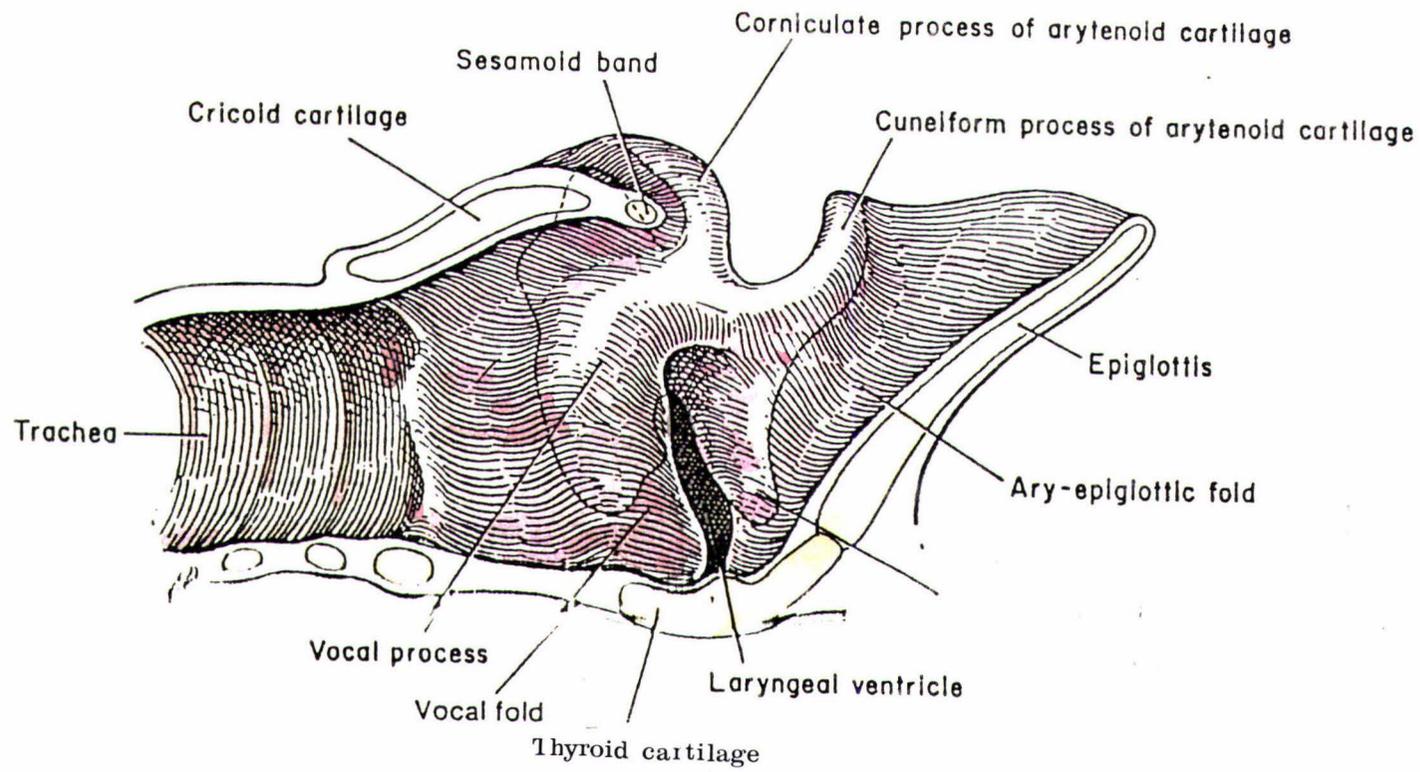
- i) Cuneiform process, the most rostral process; the ventral part of which lies in the aryepiglottic fold and the dorsal portion forms part of the laryngeal inlet.
- ii) Corniculate process, the most dorsal process forming the dorsal margin to the laryngeal inlet.
- iii) Muscular process, the most caudal process, providing insertion for the dorsal cricoarytenoid, lateral cricoarytenoid, thyroarytenoid and transverse arytenoid muscles. Under this process lies the articular surface for the cricoarytenoid articulation.
- iv) Vocal process, a caudoventral projection to which the vocal ligament and vocal muscle attach.

Two other small cartilages may be found in the larynx. The first, a sesamoid cartilage is found dorsally in a fibrous band between the arytenoid cartilages just rostral to the cricoid cartilage. The second, is the interarytenoid cartilage or nodule which lies rostral to the cricoid lamina and caudodorsal to the sesamoid cartilage. It is embedded in connective tissue which attaches to the arytenoid cartilages and provides insertion for the transverse arytenoid and *ventricularis* muscles.

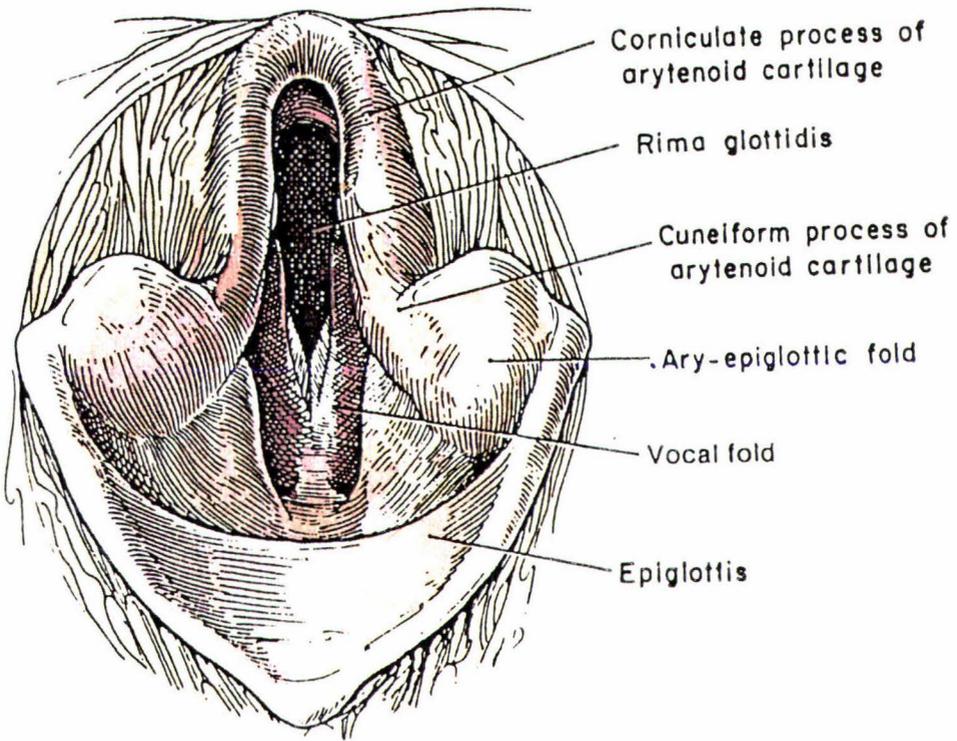
## ii) Laryngeal mucosa and associated structures

The mucous membrane of the larynx is continuous with the pharyngeal and tracheal mucosae and is composed of pseudocolumnar, ciliated epithelium caudal to the vocal cords and stratified squamous epithelium rostrally (Tucker, Vidic, Tucker and Stead, 1976). The dog has well developed aryepiglottic folds supported by, and connected between, the caudolateral border of the epiglottis and the cuneiform processes of the arytenoid cartilages. The ventricular bands ("false vocal cords") are folds of mucosa that extend from the ventral margin of the cuneiform cartilage to the rostradorsal surface of the thyroid cartilage and are parallel and rostralateral to the true vocal cords. The vocal cords are fleshy structures that pass from the ventral midline of the thyroid cartilage to the vocal process of the arytenoid cartilages. Between the vocal cord and the ventricular band is a reflection of mucosa which forms a pocket - the laryngeal saccule. These structures are shown in Figure 2.

**Figure 2.** Sagittal section through the larynx showing cartilages and their associated structures.



**Figure 3.** Cranial view of the normal larynx.



The cranial opening of the larynx, or *aditus laryngis* (see Figure 3.) is thus formed by the paired corniculate processes dorsally, cuneiform processes and aryepiglottic folds laterally and epiglottis ventrally. The narrowest part of the laryngeal airway (*rima glottidis*) is bounded by the paired arytenoid cartilages dorsally and the paired ventricular bands and vocal cords ventrally. The functional size of the *rima glottidis* is determined primarily by the respiratory needs of the dog and it is the movement of the arytenoid cartilages and vocal cords that regulate the size of this opening.

iii) Laryngeal muscles

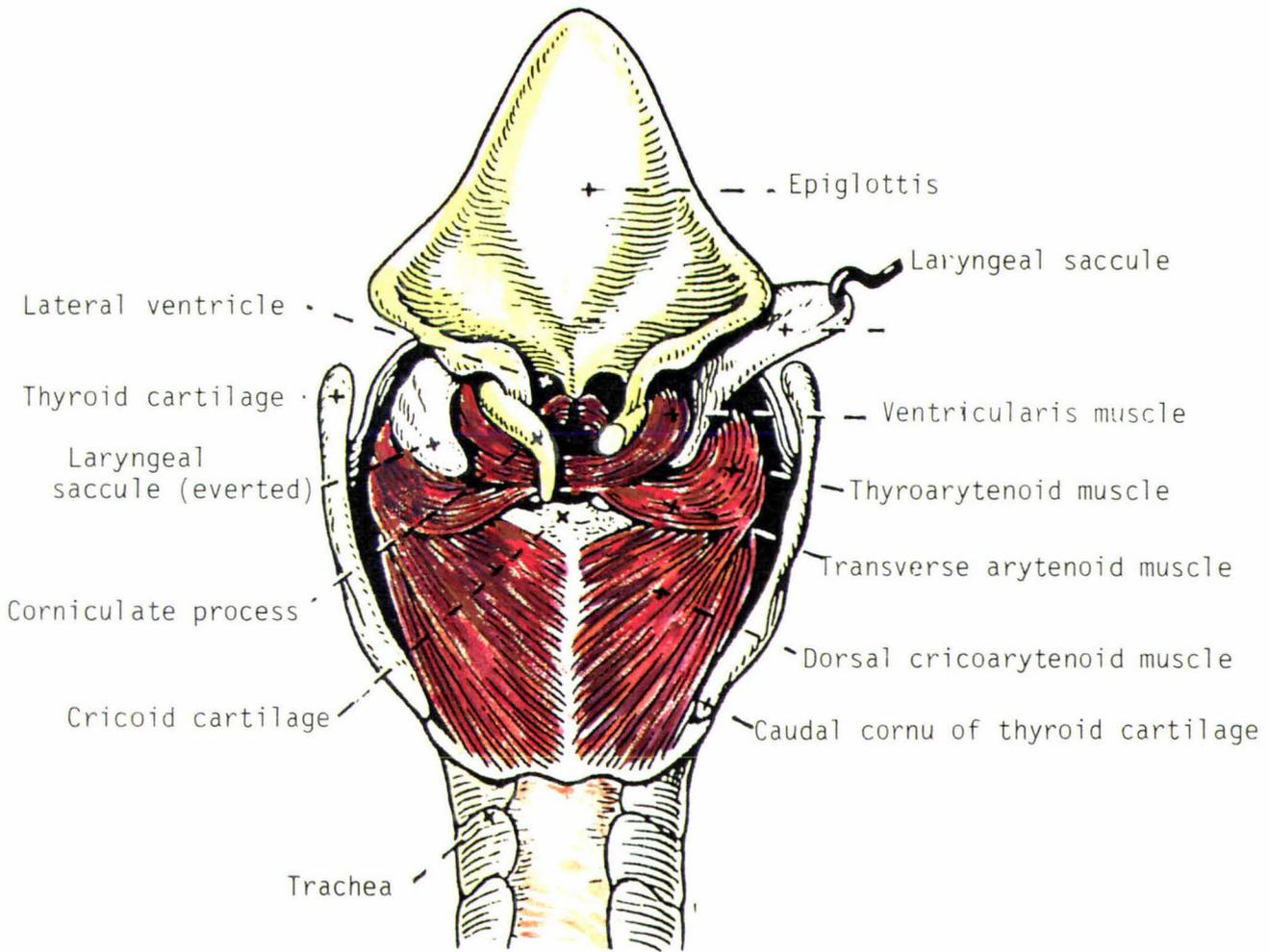
The extrinsic muscles of the larynx can be divided into three groups:

- a) those that have a pharyngeal function. These muscles consist of the *thyropharyngeus*, which arises from the thyroid cartilage lamina, and the *cricopharyngeus*, which arises from the lateral surface of the cricoid cartilage. Both muscles insert on the median dorsal raphe of the pharynx.
- (b) those that have a respiratory function. These muscles consist of the *thyrohyoideus* muscle, which runs from the lamina of the thyroid cartilage to the caudal aspect of the thyrohyoid bone, and the *sternothyroideus* muscle, which arises from the first costal cartilages and inserts on the lateral surface of the thyroid cartilage. This latter muscle has been shown to contract during inspiration (Armstrong and Smith, 1955) and acts to draw the larynx caudally.
- (c) the hyoepiglottic muscle. This originates on the medial surface of the keratohyoid bone and inserts on the ventral midline of the epiglottis. Its action serves to hold the epiglottis ventrally during respiration and to return it to its resting position following swallowing.

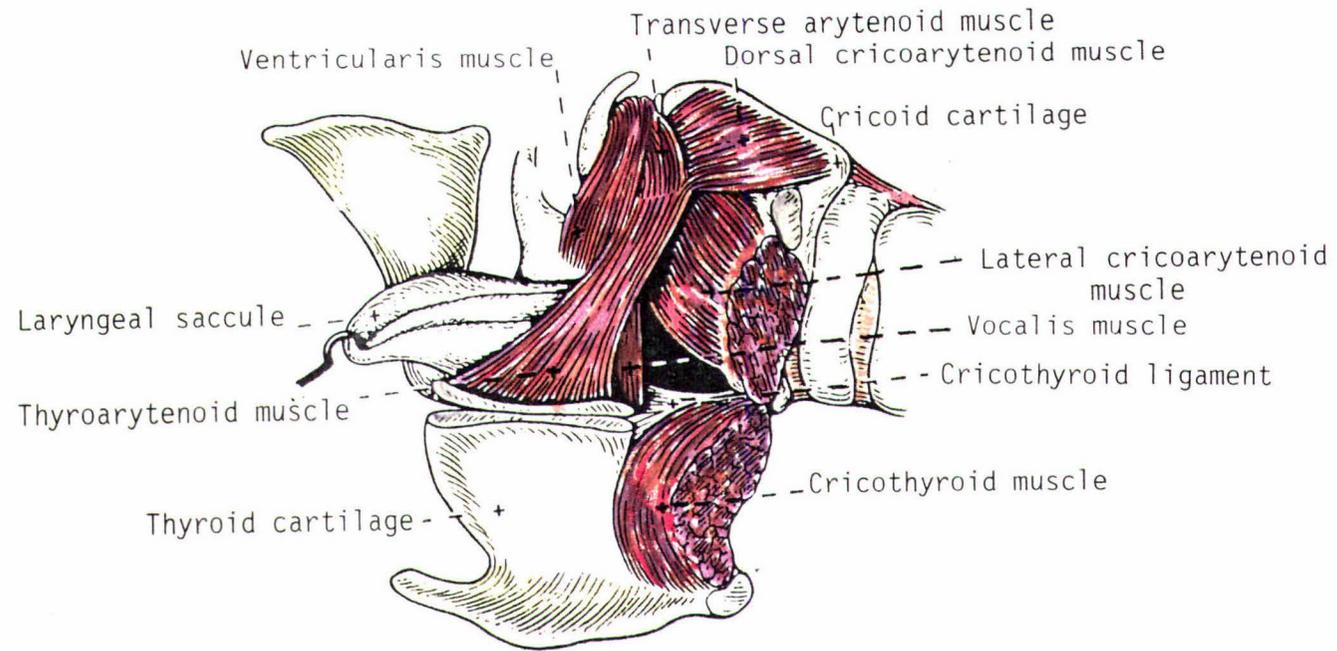
The intrinsic laryngeal muscles (demonstrated in Figure 4. and 5.) can be divided into three categories according to their action: the dorsal cricoarytenoid muscle, the cricothyroid muscle and the adductor muscles.

The dorsal cricoarytenoid muscle arises from the dorsolateral surface of the cricoid cartilage, passes craniolaterally, and inserts on the muscular process of the arytenoid cartilage. It has been shown to act synchronously with the inspiratory phase of respiration and to abduct the arytenoid cartilages and vocal cords (Nakamura, Uyeda and Sonoda, 1958. Hast, 1967). Nakamura et al (1958) and Siribodhi, Sundmaker, Atkins and Bonnes (1963) also recorded some activity, albeit reduced, during the quiet phase of respiration indicating their role in

**Figure 4.** Dorsal aspect of the larynx showing the intrinsic laryngeal muscles.



**Figure 5.** Lateral aspect of the larynx with the left lamina of the thyroid cartilage, cut and reflected, to show the intrinsic laryngeal muscles.



maintaining the arytenoid cartilages in their normal resting position. Hast (1967), in his work on dogs, described this muscle as a "slow contracting muscle" capable of prolonged and continuous activity. This contrasts to a more recent study on the muscle fibre type of the dorsal cricoarytenoid muscle (Braund, Steiss, Marshall, Mehta, Toivio-Kinnucan and Amling; 1988) in clinically normal dogs which showed a type 1 / type 2 ratio of 40.60. This muscle fibre composition is compatible with serving both sustained and prolonged activity (type 1 fibres) during the whole of respiration and fast activity (type 2 fibres) required for abducting the vocal cords. Gunn (1972) and Anderson (1984) demonstrated a similar fibre type ratio of this muscle in the horse.

The cricothyroid muscle runs along the ventrolateral surface of the larynx between the thyroid lamina and cricoid cartilage. It passes ventral to the cricothyroid articulation and extends between the caudal and medial surface of the thyroid cartilage. One of this muscle's actions is to tense the vocal cord by pivoting the cricoid cartilage at the cricothyroid articulation. This has been demonstrated by several research workers (Armstrong and Smith, 1955; Freedman, 1955; Murakami and Kirchner, 1971). Whilst most authors have reported activity of the cricothyroid muscle in different species during expiration (Murtagh and Campbell, 1954; Nakamura *et al*, 1958; Siribodhi *et al*, 1963; Shin, Brewer, Graeber and Syracuse, 1969) some have noted activity during the inspiratory phase (Armstrong and Smith, 1955; Faaborg-Anderson, 1957; Hiroto, Hirano, Toyozumi and Shin, 1964; Goulden, Barnes and Quinlan, 1976). This has left some debate as to whether the cricothyroid muscle is an adductor muscle of the larynx or plays a role in assisting the dorsal cricoarytenoid muscle in opening the glottic airway during inspiration.

Konrad and Rattenborg (1969), recorded that electrical stimulation of the cricothyroid muscle alone resulted in an increase in laryngeal resistance and a narrowing of the glottic cross sectional area. Conversely, stimulation of the dorsal cricoarytenoid muscle caused a reduction in laryngeal resistance with a widening of the glottis. When both muscles were stimulated simultaneously it resulted in a greater reduction in laryngeal resistance and an increase in glottic cross sectional area. As a consequence of these results, Konrad and Rattenborg concluded that the two muscles worked synergistically to widen the glottis.

The cricothyroid muscle has a type 1:2 fibre ratio similar to that of the dorsal cricoarytenoid muscle (Braund *et al*, 1988).

Of the adductor muscles, the thyroarytenoid is the most important, since it is the parent muscle mass for the *vocalis* and *ventricularis* muscles. It runs from the medial midline surface of the thyroid cartilage and passes caudodorsally to the arytenoid cartilages. The muscle subdivides into a medial division, which gives rise to the *vocalis* muscle, and a cranial division, which gives rise to the *ventricularis* muscle. A few fibres blend with the

dorsal cricoarytenoid muscle and some also blend with the transverse arytenoid muscle at the muscular process of the arytenoid cartilage. It is most active during expiration (Nakamura *et al*, 1958; Siribodhi *et al*, 1963) and has a type 1.2 fibre ratio of 5:95 which allows the muscle to contract rapidly and repeatedly (Braund *et al*, 1988).

The other two adductors of the larynx (i) the lateral cricoarytenoid muscle and (ii) the transverse arytenoid muscle are small muscles. The former runs from the craniolateral aspect of the cricoid cartilage to the muscular process of the arytenoid and the latter runs from the muscular process of the arytenoid to the interarytenoid cartilage.

The adductor muscles have been observed to be most active during expiration (Green and Neil, 1955; Siribodhi *et al*, 1963; Goulden *et al*, 1976). Some authors have, however, recorded no activity in the thyroarytenoid in dogs (Nakamura *et al*, 1958) and lateral cricoarytenoid and transverse arytenoid muscles in horses (Goulden *et al*, 1976) throughout the breathing cycle. In the latter case, it was suggested that this may have been due to large numbers of motor units remaining inactive during quiet respiration or alternatively due to the depth of anaesthesia. Suzuki and Kirchner (1969) had earlier observed that, in cats, deepening the anaesthetic plane diminished the activity of the adductor muscles before that of the abductor muscles.

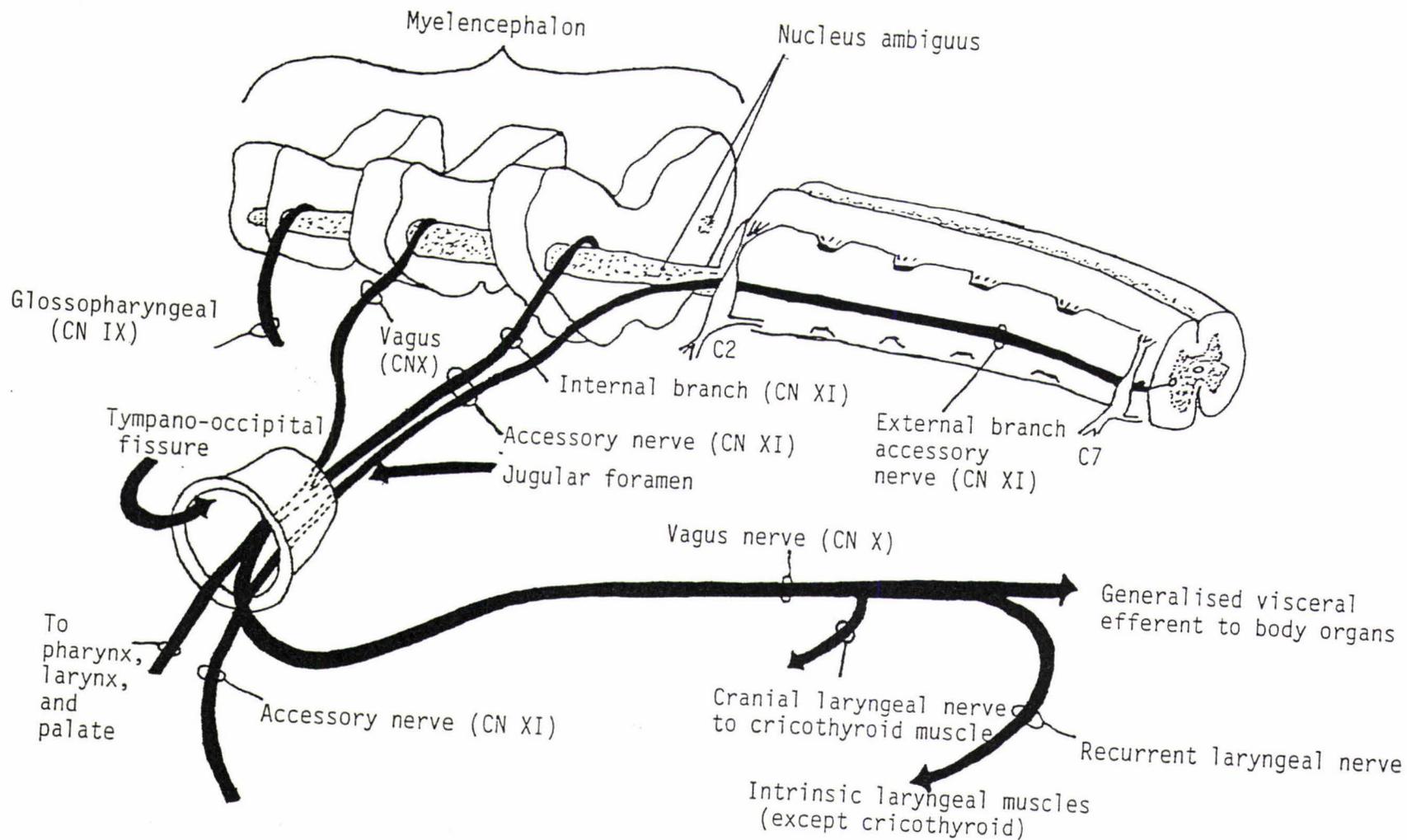
#### iv) Laryngeal nerves

Studies in dogs of the special visceral efferent cell bodies which give rise to the laryngeal nerves have shown that their location is in the *nucleus ambiguus* (Kosaka, 1909; Szentagothai, 1943). The motor neurons leave the central nervous system via the vagus and internal accessory nerves (Lemere, 1932). The axons in these latter nerves are thought to innervate the muscles of the larynx by way of the recurrent laryngeal nerves (Watson, 1974). As the vagus and accessory nerve pass through the jugular foramen and tympano-occipital foramen, the internal branch of the accessory nerve joins the vagus distal to the jugular ganglion (Figure 6.).

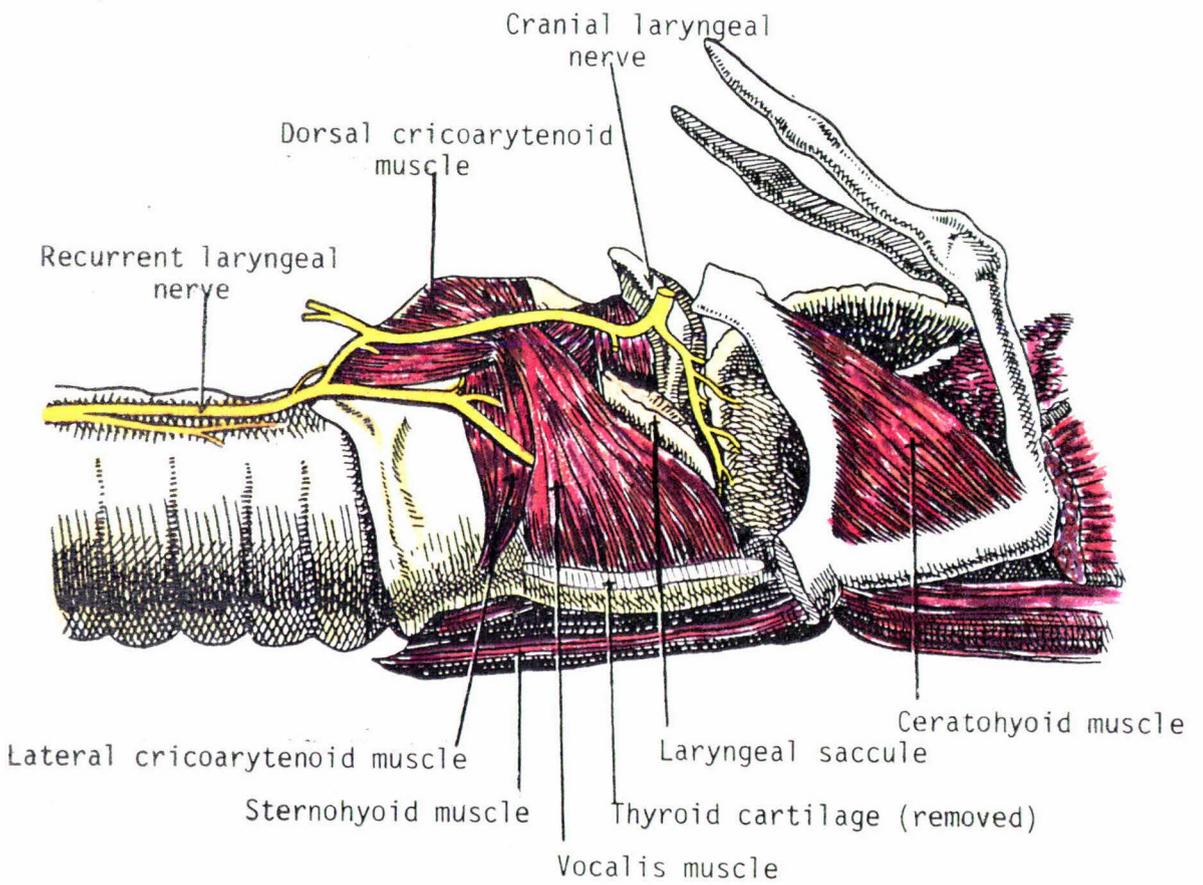
The glossopharyngeal and vagus nerves supply the *thyropharyngeus* and *cricopharyngeus* muscles. The other extrinsic laryngeal muscles are innervated by the accessory and cervical nerves, except for the hypoepiglottic muscle which is supplied by the hypoglossus nerve.

Some axons leave the vagus at the level of the nodose ganglion to form the cranial laryngeal nerve. This nerve divides into an internal branch, which provides sensory innervation to the laryngeal mucosa, and an external branch, which provides motor innervation to the cricothyroid muscle.

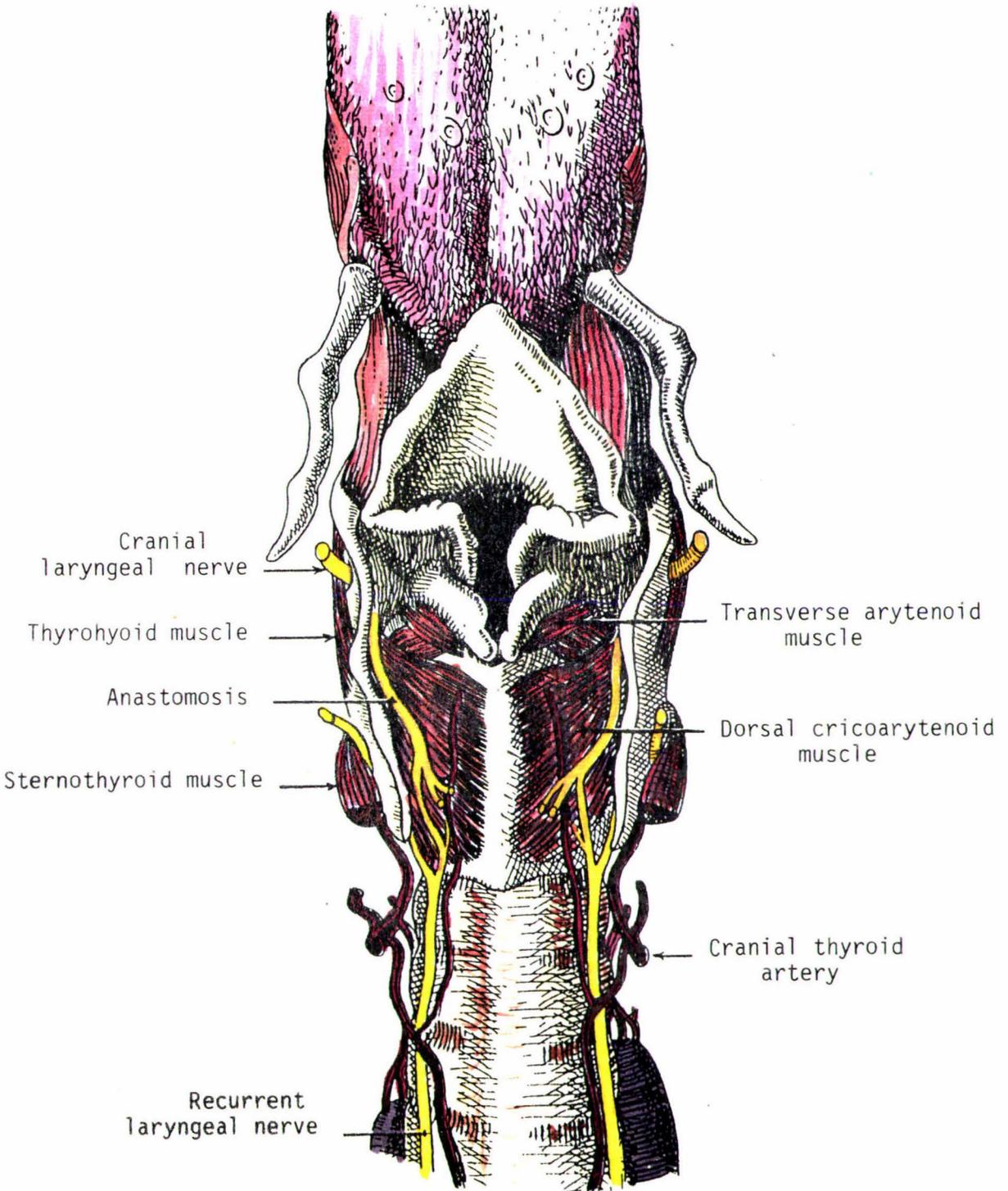
**Figure 6.** Schematic drawing of medulla with *nucleus ambiguus* and its efferent neurons, the cervical spinal cord with external branch of accessory nerve and the vagus.



**Figure 7.** Lateral aspect of larynx showing path of cranial laryngeal and recurrent laryngeal nerves.



**Figure 8.** Dorsal aspect of larynx showing path of cranial laryngeal and recurrent laryngeal nerves.



The recurrent laryngeal nerves accompany the vagus until the thoracic inlet. At this point, on the right side, it separates and loops around the right brachycephalic trunk to proceed along the right dorsal border of the trachea to the larynx. On the left side, the recurrent laryngeal nerve separates from the vagus immediately before looping around the aortic arch to proceed cranially along the left dorsal border of the trachea to the larynx. Thus, the left recurrent laryngeal nerve is longer than the right.

Each recurrent laryngeal nerve divides at the dorso-lateral aspect of the caudal border of the cricoid cartilage into several branches (see Figure 7. and 8.). Most of the branches supply motor innervation to all the intrinsic laryngeal muscles except the cricothyroid muscle. A small dorsal sensory branch on each side anastomoses with the internal branch of the cranial laryngeal nerve (Lemere, 1932). There is believed to be no cross-over motor nerve supply in the canine larynx, apart from the transverse arytenoid muscle which receives its motor supply from both recurrent laryngeal nerves (Dedo, 1970).

Whether the laryngeal muscle motor innervation is purely unilateral as described above, or whether some cross-over motor supply exists is controversial. Venker-van Haagen (1980) undertook experimental electromyographic and histological studies of the intrinsic laryngeal muscles in dogs and concluded that basically the classical description of the innervation to the larynx was correct, but that some supplementary innervation did exist; the pattern varying from dog to dog. He also found that although this supplementary innervation was important in electromyographic studies it did not indicate functional capability.

The larynx also receives an autonomic nerve supply. Parasympathetic fibres accompany the cranial and recurrent laryngeal nerves. The sympathetic supply to the larynx arises from the cranial cervical sympathetic ganglion.

### I<sub>3</sub>.      **PHYSIOLOGY**

The function of the larynx will be considered in three parts

- (i) its action during respiration
- (ii) its action during swallowing and
- (iii) its action during phonation.

#### i)      Action during respiration

The larynx acts as a significant respiratory organ, providing fine regulation of respiratory airflow, particularly in relation to expiratory flow and expiratory duration (Bartlett, Remmers,

Gautier, 1973). During voluntary respiratory manoeuvres, both intrinsic laryngeal and extrinsic respiratory muscles appear to be recruited to effect this ventilatory and respiratory control (Brancatisano, Collett, Engel, 1983) by altering glottic aperture and thus laryngeal resistance.

During normal resting respiration the larynx is held partially open. In conscious humans the glottis has been observed to widen during inspiration and narrow during expiration; the widening generally preceding the onset of inspiration; and the narrowing commencing before the onset of expiration (Brancatisano *et al*, 1983). The partial opening of the glottis is effected by intermittent short duration contraction of muscle bundles of the dorsal cricoarytenoid muscles. As required by this type of work, the dorsal cricoarytenoid muscle in man has the best developed aerobic metabolism and capillary supply of all the intrinsic laryngeal muscles (Ganz, 1971).

Alterations in laryngeal resistance reflect the changes in glottic aperture. In reposed breathing, the increase in laryngeal resistance always starts when inspiratory airflow reaches its maximum value. During expiration, the resistance remains high and only falls when the expiratory airflow decreases towards zero ( Gonzalez-Baron, Bogas, Molina and Garcia-Matilla, 1978). The volume of air inhaled and exhaled during the respiratory cycle at rest is equal, but the time taken for the passage of this air during the two breathing phases differs. The duration of expiration in spontaneous breathing is prolonged relative to inspiration. Thus inspiration and expiration are fundamentally different mechanical processes. During inspiration, the rate of airflow is determined by the creation of a negative pressure caused by muscular contraction of the intercostal and diaphragmatic muscles and a low resistance pathway through the larynx. Expiration is primarily the result of passive recoil of the lungs and chest wall. However, the duration of expiration is approximately twice as long as the time required for passive collapse (Gautier, Remmers, Bartlett, 1973) and since an end-expiratory pause does not exist, some expiratory braking mechanism must act to retard expiratory airflow. It is believed (Gautier, *et al*, 1973) that the larynx, which varies the upper airway resistance by movement of the vocal cords, acts as this expiratory brake. This expiratory slowing of the airflow during expiration by the larynx enables the appropriate respiration rate to be achieved at rest.

During exercise, the rate of airflow must increase greatly above that at rest. This is accomplished by lowering the laryngeal resistance by widening the glottic aperture during both inspiration and expiration. Lowering of the resistance during expiration increases the airflow rate during this phase and reduces the expiratory duration by removing the expiratory brake. This latter effect enables an increase in respiratory rate appropriate for exercise.

The larynx has a rich sensory nerve supply which is the main pathway of several respiratory reflexes. These reflexes, which influence both the patency of the upper airway and the pattern of breathing, are stimulated by transmural pressure and/or airflow in the upper airway. Three types of laryngeal mechanoreceptors have been described in dogs (Sant'Ambrogio, Mathew, Fisher and Sant'Ambrogio, 1983). These are pressure receptors, flow receptors and "drive" receptors (stimulated by the activity of the laryngeal muscles) and although they differ in sensory perception, they all concur in exhibiting a predominant activity during inspiration. All these reflexes are mediated via the cranial laryngeal nerve (Sant'Ambrogio, Mathew, Clark and Sant'Ambrogio, 1985).

Two other types of receptors, both of which are independent of airflow, have also been discovered in the dog. These are:

a) chemoreceptors. These respond to conditions of hypercapnia or hypoxia, bringing about a reduction in laryngeal resistance during both inspiration and expiration (McCaffrey and Kern, 1980). This chemoreceptor-mediated regulation of airway resistance is less sensitive in the young puppy (Blum and McCaffrey, 1984) and may predispose the neonate to airway obstruction during periods of hypoxia or hypercapnia. In neonatal mammals, the introduction of water and some other fluids into the larynx causes prolonged reflex apnoea by stimulation of afferents of the cranial laryngeal nerve. The principle stimulus for this reflex in young puppies is the absence or reduced concentration of chloride ions in the laryngeal fluid (Boggs and Bartlett, 1982).

and

b) thermoreceptors. These are activated by cooling of the air (Sant'Ambrogio, Mathew, Sant'Ambrogio and Fisher, 1985) and are believed in the dog, to be located superficially within the vocal folds (Sant'Ambrogio, Mathew and Sant'Ambrogio, 1988).

Reflex glottic closure is a dominant and stable reflex produced by stimulation of the cranial laryngeal nerve. Its precise execution is basic to successful sphincter protection of the lower airway (Ikari and Sasaki, 1980). Mechanical stimulation of the larynx, soft palate, pharynx, and trachea causes apnea, bradycardia and glottic closure (Angell-James, de Burgh Dayl, 1975; Frisk, Titullaer, Kampine, Zuberku, 1974). When hypoxia occurs secondary to the apnea produced, carotid body chemoreceptor stimulation normally follows, resulting in hyperpnea. However, continued laryngeal stimulation facilitates an inhibiting carotid body reflex on cardiac muscle which, if prolonged, can lead to cardiac and respiratory failure (Angell-James *et al*, 1975).

Mechanical stimulation of the trachea or larynx evokes the cough reflex. At one time it was thought that the glottis closed between the inspiratory and expiratory phase, but it is now known that it remains open through all stages of coughing. The cough cycle commences with a sudden deep inspiratory breath, filling the lungs with air, whilst the larynx is held open.

Sudden contraction of the expiratory muscles and a synergistic reflex dilation of the glottis causes the air to be released in an explosive fashion, carrying with it any foreign material (Jimeriz Vargus, Gonzaliz Baron and Asiron, 1973).

ii) Action during swallowing

Reflex closure of the larynx occurs during swallowing. This results from both external and internal laryngeal muscle activity. As swallowing is initiated, the larynx is pulled rostrally by the cranial hyoid muscles. This movement, together with the caudal movement of the base of the tongue, results in the folding of the epiglottis over the glottis, thus deflecting the food or fluid bolus from the airway (Harvey, 1981). At the same time, the intrinsic laryngeal adductor muscles (thyroarytenoid, interarytenoid, lateral cricoarytenoid, *vocalis* and *ventricularis*) contract, causing adduction of the arytenoid cartilages and vocal cords to complete glottal closure and prevent aspiration. Respiration recommences once the food bolus has been swallowed.

iii) Action during phonation

Barking can be an important function in the dog, not only for communication between dogs but also for hunting, working or guarding, although obviously noise production is not as important as in man. Voice production is related to the movement of air over the vocal and vestibular folds and is influenced by changes of the subglottic pressure and the length, thickness and tension of the vocal cords (O'Brien and Harvey, 1983). The latter characteristic of these cords is dependant primarily upon contraction of the cricothyroid muscle (Armstrong and Smith, 1955).

1<sub>3a</sub>

i) The paralysed larynx

Dogs are able to survive with paralysed larynges, often showing few symptoms until exercised or stressed. There is usually a voice change, which may or may not be noticed by owners.

Numerous studies on the effect of sectioning laryngeal nerves on laryngeal function have been performed on experimental dogs acting as models for man. Before 1970, controversy existed about the nature of the paralysed larynx. This prompted Dedo (1970) to conduct a comprehensive study to determine whether specific patterns of laryngeal immobility and position could be consistently correlated to the paralysis of specific laryngeal nerves. On

**Figure 9.** Diagrammatic representation of  
A) the normal larynx,  
B) the effects of left recurrent laryngeal nerve paralysis  
and  
C) the effects of left cranial laryngeal and recurrent laryngeal nerve paralysis.



A. Normal Larynx

B. Left recurrent laryngeal nerve paralysis (paramedian position)



C. Left cranial laryngeal and recurrent laryngeal nerve paralysis (intermediate position)

the basis of this study, it is now known that sectioning one recurrent laryngeal nerve causes paralysis of the ipsilateral arytenoid cartilage and vocal cord. These take up an adducted or paramedian position (Figure 9B). Since the recurrent laryngeal nerve supplies the only abductor muscle (dorsal cricoarytenoid) of the larynx, this capability is also lost.

When one cranial laryngeal nerve only is sectioned, there is no obvious change in glottic size at rest. However, upon contraction of the contralateral cricothyroid muscle, the ventral commissure of the glottis becomes deviated towards the inactivated side (Tanabe, Isshiki and Kitajima, 1972) resulting in elongation of both vocal cords, though more on the active side. Both vocal cords are seen to vibrate at the same frequency, but a lag phase exists between innervated and denervated sides.

When both ipsilateral cranial and recurrent laryngeal nerves are sectioned, the vocal cords adduct. The adduction observed, however, is less than that seen when only the recurrent laryngeal nerve is sectioned, and is termed the "intermediate" position (Figure 9C). All vocal cord movements cease. This implies that the cranial laryngeal nerve, through its action on the cricothyroid muscle, has a small abducting effect on the vocal cords and accounts for the reduced glottal size (paramedian position) seen when there is paralysis of the recurrent laryngeal nerve only.

The resultant narrowed *rima glottidis*, in the paralysed larynx not only increases resistance to airflow, but also creates turbulence which gives rise to the laryngeal stridor heard in laryngeal paralysis cases (Forgacs, 1978). In addition, the mechanics of the airflow through the narrowed larynx are dramatically altered by the Bernoulli and Venturi effects. The Bernoulli principle denotes that, as the speed of the air passing through the normal larynx is increased, such as during exercise, there is a reduction in intra-luminal pressure. This pressure fall is accentuated in laryngeal paralysis by the Venturi effect. This is because the speed of the air through the narrowed *rima glottidis* has to increase in order to achieve the same flow rate as the remainder of the respiratory tract. In fact, the pressure reduction within the larynx can be so great, that the unstable arytenoid cartilages and vocal cords can be sucked into the lumen, thereby further reducing glottic size and restricting inspiratory flow.

The effect of this dynamic collapse increases as the animal exercises, due to more rapid passage of air. This explains the marked exercise intolerance seen in laryngeal paralysis patients.

#### I<sub>4</sub>. LARYNGEAL PARALYSIS - DIAGNOSTIC TECHNIQUES

A number of diagnostic techniques have been used in the assessment of laryngeal paralysis. These include:

i) Laryngoscopy

Laryngoscopic examination under light general anaesthesia is the most common clinical technique for diagnosing laryngeal paralysis. The plane of anaesthesia during examination is important, since deep anaesthesia can cause cessation of all laryngeal movements, even in the normal dog. The anaesthetised animal should be examined endoscopically when the mouth can be easily opened, but reflex closure of the glottis is present (Greenfield, 1987). The laryngeal movements can then be observed as the dog awakens.

For an accurate diagnosis, it is important to correlate the observed arytenoid and vocal cord movements with the phase of respiration. In the normal dog, the arytenoid processes and vocal folds should abduct symmetrically during inspiration and relax to form a relatively small glottic opening at expiration. In laryngeal paralysis cases, the affected arytenoid cartilages and vocal folds show no active abduction during inspiration. Sometimes, confusing movements may be seen such as the larynx being pulled caudally by the contraction of the accessory muscles of respiration, or the vocal cords can be passively drawn towards the midline during inspiration. In this paradoxical movement, the vocal cords appear to abduct as they return passively to their original position at the end of inspiration. During expiration, the vocal folds can flutter in the flow of expired air (Harvey, 1981; Wykes, 1983; Greenfield, 1987). Asymmetrical abduction is suggestive of hemiplegia. However, asymmetrical or partial movement of the arytenoid cartilages and vocal folds can also be seen where there is the co-existence of normal motor units potentials and denervation potentials within the same dorsal cricoarytenoid muscle (Venker-van Haagen, 1980).

ii) Blood Gas Analysis

The values of arterial blood oxygen ( $\text{PaO}_2$ ) and carbon dioxide ( $\text{PaCO}_2$ ) tensions represent the final assessment of the effectiveness of breathing and analysis of these tensions has proved useful in the diagnosis of respiratory disease in dogs (Clark, Jones and Clark, 1977). Moreover they are useful in evaluating the success of laryngeal paralysis treatment in these animals (Love, *et al*, 1987). The mean  $\text{PaO}_2$  tension in normal dogs has been documented as  $101.3 \pm 5.6$  mmHg (Clark *et al*, 1977). However, this is reduced in sedated animals to approximately 90 mmHg (Love *et al*, 1987; Cornelius and Rawlings, 1981).

Dogs with laryngeal paralysis show a moderate degree of arterial hypoxemia at rest and improvement following surgery for the relief of laryngeal paralysis has been recorded (Love *et al*, 1987).

Blood samples can be easily obtained from the femoral artery and the analysis is quickly and accurately performed by using a blood gas analysis machine.

### iii) Tidal Breathing Flow Volume Loops

The standard flow-volume loop is a graphic expression of the air flow plotted against volume produced by a maximum forced expiration and inspiration during a single breath (Figure 10). The measurements of the volume of air and the rate of it's flow are often obtained by using respiratory flowmeters (pneumotachographs). To obtain a flow-volume loop, the patient is required to take a deep breath to total lung capacity, then exhale as quickly as possible, and then inhale again as quickly as possible to total lung capacity. The movements of the thorax during breathing transmit forces through the pleural space to the lungs, which passively follow all volume changes of the chest. The force required to distend the lungs during inspiration is determined by the viscoelastic behaviour of the lung tissue. Maximum expiratory flow rates are determined mainly by the flow resistance of peripheral airways and by lung elastic recoil. Lung size and age influence the shape and size of the maximum expiratory flow-volume curves and obstructive and restrictive lung diseases produce abnormal curve patterns. As such they have been utilised for evaluating disease of the major airways in adult humans (Dayman, 1951; Fry and Hyatt, 1960; Miller and Hyatt, 1973).

Three characteristic flow-volume loop patterns were originally described by Miller and Hyatt (1973) using forced expiratory and forced inspiratory vital capacity measurements (Figure 11).

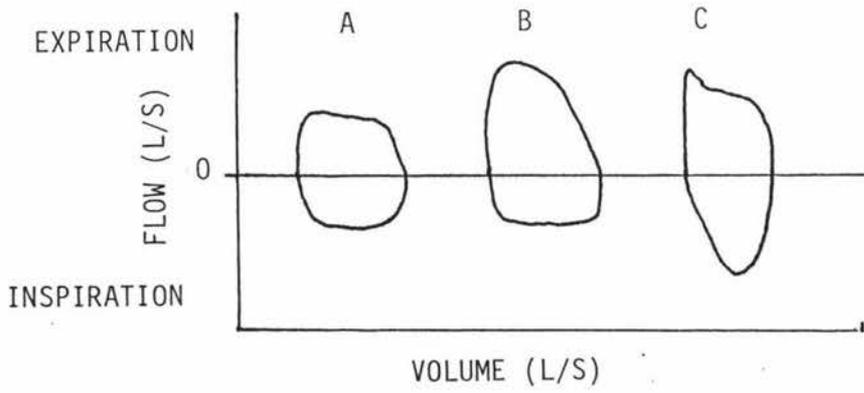
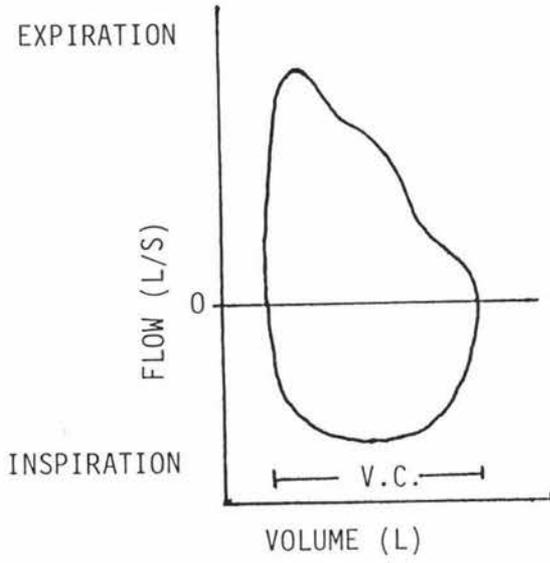
These were:

- a) where the peak flows of both inspiration and expiration were reduced indicating a fixed lesion.
- b) where there was a predominant distortion of the inspiratory arc indicating a variable extra thoracic lesion.
- c) where there was a predominant distortion of the expiratory arc indicating a variable intrathoracic lesion.

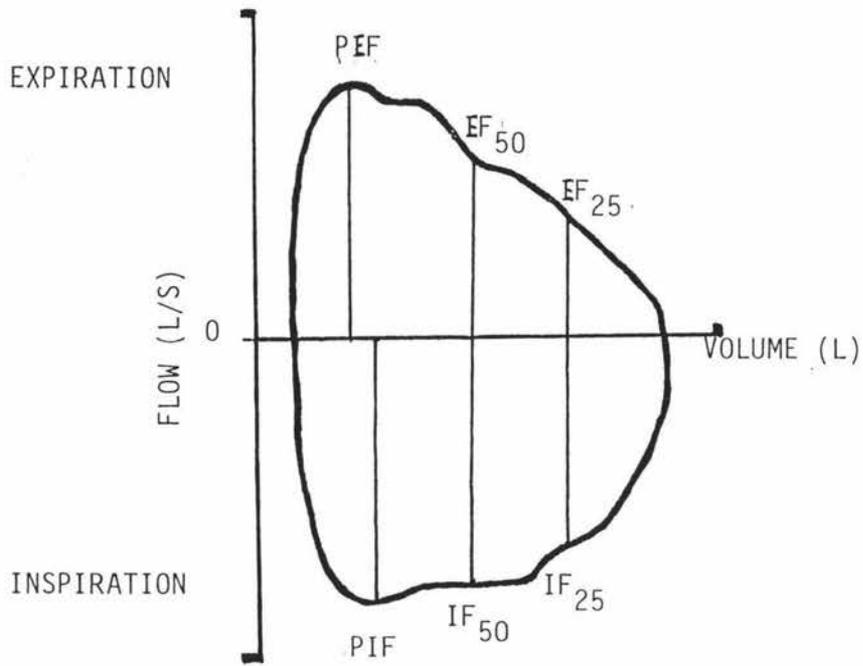
A standard flow volume loop is impossible to obtain in neonates and infants, due to lack of patient co-operation in achieving maximum expiratory and inspiratory effort. Abramson and Hyatt (1982), therefore, developed the technique of using tidal breathing flow-volume loops

**Figure 10.** Maximum flow-volume loop of a healthy adult human showing typical expiratory and inspiratory tracings.  
V.C. Vital Capacity

**Figure 11.** Characteristic flow-volume loops produced by major airway lesions.  
A) a fixed lesion showing reduced peak flow of both inspiration and expiration.  
B) a variable extrathoracic lesion showing distortion of the inspiratory arc.  
C) a variable intrathoracic lesion showing distortion of the expiratory arc.



**Figure 12.** Characteristic tidal breathing flow-volume loops of a healthy dog showing peak expiratory flow (PEF), peak inspiratory flow (PIF), midtidal expiratory flow ( $EF_{50}$ ), midtidal inspiratory flow ( $IF_{50}$ ), and expiratory and inspiratory flow at 25% of the tidal volume remaining to be exhaled ( $EF_{25}$  and  $IF_{25}$  respectively).



from sleeping patients for evaluating upper airway function. Interpretation of the tidal breathing flow-volume loops follow similar principles to that of standard loop analysis - the latter are more sensitive to early detection of airway disease since they represent maximum effort and are therefore not subject to effort dependence and breathing patterns. Nevertheless, Abramson *et al* concluded that tidal breathing flow-volume loops were a non-invasive, rapid and reproducible, pulmonary function test. The pattern of loop changes seen in disease mimicked those of the standard flow-volume loop.

Amis and Kurpershoeck (1986) evaluated the use of tidal breathing flow-volume loops in normal conscious dogs and those suffering from airway obstruction, and found they were easily obtained and assisted in diagnosis. They analysed the loop shapes by measuring the flow at various standard volumes during the breathing cycle. These were peak expiratory and inspiratory flow, 50% and 25% of the tidal volume remaining to be exhaled (Figure 12). Since the loop shapes are affected by body size and breathing effort, they evaluated the loops using ratios of absolute flow measurements in an attempt to standardise the indices. Later Amis, Smith, Gaber and Kurpershoeck (1986) also used tidal breathing flow-volume loops to assess the type and severity of airway obstruction in 30 dogs with laryngeal paralysis. They concluded that the loop provided both a qualitative and quantitative evaluation of the airway obstruction, with 17 of the dogs having a non-fixed inspiratory upper airway obstruction (Type 2), 10 a fixed inspiratory/expiratory obstruction (Type 3), and three had normal loops (Type 1).

#### iv) Upper Airway Flow Mechanics

Measurements of upper airway flow mechanics during exercise have been used as a functional assessment of the effect of laryngeal hemiplegia in the horse and to determine the efficacy of surgical intervention (Derksen, Stick, Scott, Robinson and Slocombe, 1986; Shappell, Derksen, Stick and Robinson, 1988). For these purposes, upper airway pressure differences across the larynx were obtained by using two pneumotachographs, one measuring the barometric pressure at the nostrils and one the pressure in the cranial trachea. It was found that laryngeal hemiplegia resulted in inspiratory flow limitations at peak inspiratory flow and increased inspiratory resistance at fast exercise. Measurements taken after surgical treatment by prosthetic laryngoplasty demonstrated a reduction of the previous flow limitations and inspiratory resistance.

This technique has not been recorded in the dog.

v) Electromyography

Electromyography of the intrinsic laryngeal muscles can help confirm a diagnosis of laryngeal paralysis by detecting the presence of denervation potentials during insertion of bipolar electrodes into the affected muscle. Several authors (Venker-van Haagen, 1978, 1981; Harvey and O'Brien, 1982) have recorded its use in suspect laryngeal paralysis cases. Venker-van Haagen (1978) advocated placement of electrodes via the mouth to evaluate the dorsal cricoarytenoid, *ventricularis* and thyroarytenoid muscles. In one experimental study (Siribodhi *et al*, 1963), denervation potentials were found to be fully developed approximately five days after trauma to the recurrent laryngeal nerve.

Electromyographic investigations, whilst useful for the detection of laryngeal paralysis, have little merit in the evaluation of the effect of surgery.

vi) Palpation

Palpation of the larynx is performed as a screening process primarily to identify non-neurogenic causes of upper respiratory distress such as, an extralaryngeal mass displacing or compressing the larynx and trachea, fractured hyoid bone, avulsion of the larynx, or laryngeal trauma resulting in the formation of subcutaneous emphysema.

Wykes (1983) considered that the laryngeal cartilages may feel more prominent and defined in dogs suffering from laryngeal paralysis, because of the intrinsic muscle atrophy.

vii) Radiography

Radiography is of limited value in the diagnosis of laryngeal paralysis (Wykes, 1983; Gaber *et al*, 1985) but can be used to differentiate extrinsic laryngeal disorders from intrinsic laryngeal disorders, prior to laryngoscopic examination. It can also contribute to case management by the detection of conditions which occur concurrently in some laryngeal paralysis patients such as bronchopneumonia and megaesophagus.

viii) Histopathology

Biopsies of the intrinsic laryngeal muscles (mainly dorsal cricoarytenoid and vocal) taken at the the time of surgery have been used by several authors (O'Brien *et al*, 1973; Reinke and Suter, 1978; Venker-van Haagen, 1978) to confirm neurogenic atrophy in suspect laryngeal paralysis cases.

ix) Histochemistry on Laryngeal Muscle Fibres

Histochemical "profiles" of laryngeal muscles can be used to demonstrate the metabolism of the muscle fibres, the distribution of muscle fibre types, and some aspects of muscle fibre architecture. The histochemical stains usually used (Buchthal and Schmalbruch, 1980) are:

- i) Myosin adenosine triphosphatase to define the fibre's contraction speed
- ii) Succinate dehydrogenase to indicate the level of the fibre's aerobic metabolism and
- iii) Glycogen phosphorylase to indicate its anaerobic metabolism.

The effects of denervation on muscle fibres can be assessed by the above histochemical techniques. The affected muscle fibres shrink, whilst adjacent muscle fibres appear normal in size or hypertrophied. Characteristically collateral sprouting occurs and, as a result of reinnervation, fibre type grouping (Cahill and Goulden, 1986).

Histochemical studies have been performed on the intrinsic laryngeal muscles in clinically normal dogs (Braund *et al*, 1988) and in six cases of laryngeal paralysis (Braund *et al*, 1989). In the latter study, the research workers found scattered small or large fibre group atrophy of type 1 and type 2 fibres and fibre type grouping indicating a neurogenic atrophy. Whilst this diagnostic technique is useful in the diagnosis and understanding of the disease, idiopathic laryngeal paralysis, it is not helpful for evaluating the effects of surgery.

x) Nerve Transmission Studies

Quantitative evaluation of recurrent laryngeal nerve function by determination of nerve conduction velocities, latencies, and characteristics of compound muscle action potentials have been studied in clinically normal dogs (Steiss and Marshall, 1988). The technique involved electrically stimulating recurrent laryngeal nerve fibres at two points along their extralaryngeal course and evoking compound muscle action potentials in the ipsilateral intrinsic laryngeal muscles using a percutaneous needle electrode.

Although, there are no reports utilising this electromyographic technique for the diagnosis of idiopathic laryngeal paralysis in dogs, it is suited for distinguishing a neuropathy from a primary myopathy in cases of laryngeal dysfunction.

## I<sub>5</sub>. LARYNGEAL PARALYSIS - A REVIEW OF SURGICAL TECHNIQUES

Laryngeal paralysis is characterised by inadequate abduction and instability of the arytenoid cartilages, aryepiglottic folds and vocal cords. Surgery performed to alleviate these effects should ideally aim to:

- a) reduce the constriction caused by the paralysed arytenoid cartilages, and
- b) prevent dynamic collapse.

Five surgical techniques to alleviate the effect of laryngeal paralysis in dogs have been described. These are:

### i) Partial Laryngectomy

This technique, which consists of either unilateral or bilateral vocal cord excision with unilateral arytenoidectomy, has been described by several authors (Baker, 1972; O'Brien *et al*, 1973; Lane, 1978; Harvey and Venker-van Haagen, 1975; and Harvey and O'Brien, 1982).

Two surgical approaches to the larynx have been reported. The oral route is the simplest and allows sections of the vocal cord, arytenoid cartilage and aryepiglottic fold to be removed, but occasionally access by this approach can be difficult. The ventral route involves a midline incision through the thyroid cartilage and crico-thyroid membrane. This allows for improved access to the larynx. During post-operative healing of both techniques, a web of excessive granulation tissue may occasionally form across the ventral floor of the larynx causing a stenosis of the glottis (Harvey and Venker-van Haagen, 1975; Wykes, 1983). Most authors agree that a tracheostomy tube should be used during this operation.

There have been few post operative evaluations of the effects of this technique. In a study of the larynges from cadavers, Harvey (1983) demonstrated that the technique increased the cross sectional area of the glottis by 84.8%. Early reports on single case studies by Reinke and Suter (1978) using bilateral vocal cord resection and Cook (1964), Roullard and Feher (1978) using partial laryngectomy indicated that the techniques were promising. However, this was not supported in later studies by Harvey and O'Brien (1982) and Gaber *et al* (1985). The former authors cited a 30% mortality rate in the immediate post operative period, an additional 11% required further surgery and over 80% of cases died subsequently of an airway disease. Inhalation pneumonia was noted as a frequent problem, a feature which according to Harvey (1983) may be related more to a generalised neuromuscular disease associated with idiopathic laryngeal paralysis, than to the surgical technique of partial laryngectomy.

For these reasons, this operation is no longer favoured by most surgeons.

## ii) Arytenoid Cartilage Lateralisation

The aim of this technique is to achieve permanent abduction of the vocal cord by lateral fixation of the corresponding arytenoid cartilage to the caudolateral border of the thyroid or cricoid cartilage.

There are still opposing arguments regarding the use of a unilateral versus a bilateral lateralisation procedure. Venker-van Haagen (1980) and LaHue (1989) both considered that unilateral lateralisation allowed resumption of normal activities in pet dogs suffering from idiopathic laryngeal paralysis. The former author, also considered that a bilateral technique caused extensive surgical trauma which healed by scar tissue formation resulting in laryngeal narrowing. Contrary to these opinions, Rosin and Greenwood (1982), concluded that a unilateral procedure failed to increase the size of the airway sufficiently for normal breathing.. These authors performed bilateral lateralisation on three cases of laryngeal paralysis. Two of these dogs survived for three years or more and had no recurrence of respiratory symptoms. The remaining animal was euthanased five months after surgery because of a hind limb weakness.

The need for a tracheostomy tube during the operation is also disputed by surgeons (Rosin and Greenwood, 1982; LaHue 1989).

The effect of the lateralisation procedures on the laryngeal width has been demonstrated by Harvey (1983) who showed that a unilateral operation increased the glottal cross sectional area by approximately 70%, while the bilateral technique increased it by about 180%. Some functional improvement, as evidenced by increased post operative arterial oxygen tension, has been noted in seven cases undergoing a unilateral surgical technique (Love *et al*, 1987). No respiratory function tests for the assessment of the bilateral procedure have, prior to this present study, been reported.

The aim of this operation is to abduct the arytenoid cartilages, thus reducing the resistance to inspiratory flow. The resultant abduction of the vocal folds should also reduce turbulence at maximal inspiratory effort and, hence improve exercise tolerance. However, with the arytenoids and vocal folds permanently abducted, some of the laryngeal reflexes, such as glottal closure during swallowing, must be impaired and this may lead to secondary lower respiratory tract disease.

iii) Castellated Laryngofissure and Vocal Fold Resection

This technique involves widening of the glottis and subglottis by making a stepped (castellated) incision in the ventral aspect of the thyroid cartilage and then stabilising the created central flap by suturing it to the incised edge of the opposite cranial thyroid cartilage segment. The vocal folds are, also, removed.

The technique is based on the principles of tracheoplasty in humans (Evans and Todd, 1974; Cotton and Richardson, 1981) and has been used for the treatment of laryngeal paralysis in four dogs by Gourley, Paul and Gregory (1983). Satisfactory results were obtained in all four dogs as, post operatively, they either showed no clinical evidence of respiratory disease or the presence of only slight inspiratory stridor. Tracheostomy tubes were used during each operation and removed between three to six days after surgery. No objective follow-up study of the effectiveness of this technique has been reported.

The aim of this technique is to widen the *rima glottidis* ventrally. Whilst it may well accomplish this, it fails to abduct the arytenoids which presumably still remain in a paramedian position. The net effect of reduced inspiratory resistance is therefore a fine balance between increase in width achieved ventrally and degree of obstruction by the arytenoids that remains dorsally. It also relies on the strength of a segment of thyroid cartilage to maintain the increase in glottic width.

iv) Modified Castellated Laryngofissure

The modified castellated laryngofissure involves performing a castellated laryngofissure with vocal fold resection and stabilising the arytenoid cartilage by suturing it to the thyroid cartilage. A tracheostomy tube is used during and after this surgery.

The purpose of stabilising the arytenoids in this modified technique is to prevent any upper airway obstruction that may occur due to the dynamic collapse of these cartilages into the glottis during inspiration.

The technique has been clinically assessed and objectively evaluated using tidal breathing flow-volume loops (Smith, Gourley, Kurpershoeck and Amis, 1986) in 12 dogs with idiopathic laryngeal paralysis. Clinical improvement occurred post operatively in 11 out of 12 dogs and the tidal breathing flow-volume loops returned to normal in seven of the 10 dogs monitored. It therefore seems to be of some benefit in alleviating the effects of laryngeal paralysis.

v) Neuromuscular Pedicle Graft

Greenfield, Walshaw, Kumber, Lowrie and Derksen (1988) using eight dogs with experimentally induced left laryngeal hemiplegia, transplanted neuromuscular grafts from a terminal branch of the first cervical nerve and adjacent sternothyroid muscle to the denervated cricoarytenoid muscle. The sternothyroid was chosen as the donor, since it is the only accessory respiratory muscle in dogs, known to contract synchronously with inspiration. Arytenoid movement was evaluated several times during the first 44 weeks following surgery. Biopsy samples of the dorsal cricoarytenoid muscle were examined histologically and histochemically in four of the dogs, 19 weeks after transplant. Results indicated that there was gradual improvement in arytenoid movement in the transplanted left side until 33 weeks, when the abduction observed was equal to that of the normal right side. The outcome of the biopsies samples taken from the four dogs, however, lacked consistency. Normal muscle was present in two dogs, evidence of re-innervation in one, and neurogenic atrophy present in the remaining animal.

The authors encountered four operative difficulties or complications. These were (i) haemorrhage at the graft site, (ii) finding a suitably long branch of the first cervical nerve, (iii) obtaining the muscular portion of the pedicle without transecting the supplying cervical nerve branch, and (iv) aligning the muscle pedicle fibres parallel to those of the recipient dorsal cricoarytenoid muscle.

Some controversy exists over the occurrence of laryngeal re-innervation following neuromuscular grafting. Advocates of the technique are Takenouchi and Sato (1968) and Tucker, Harvey and Ogura (1970). However Crimley (1982) demonstrated that re-innervation of the dorsal cricoarytenoid muscle does not occur via the nerve in the transplanted pedicle and that any glottic airway improvement appeared to be mediated by the accessory muscles of respiration. He also demonstrated that if abductor function was to be restored to the paralysed dorsal cricoarytenoid muscle, free nerve grafts were required.

In all these studies, transplant of the neuromuscular graft was performed in recent denervated dorsal cricoarytenoid muscle. In dogs with laryngeal paralysis, the muscle has already undergone denervation atrophy with the resultant histological changes of fibrosis and motor end plate alteration. A study by Morledge, Lauvstad and Calcaterra (1973) showed that re-innervation of the dorsal cricoarytenoid muscle after nine to 12 months of denervation was unlikely to occur. It is therefore equally unlikely, that idiopathic laryngeal paralysis in dogs, would respond significantly to neuromuscular pedicle grafting.

## CHAPTER II. MATERIALS AND METHODS

Ten adult mongrel dogs, seven male and three females, with an average body weight of 20kg (range 13kg to 25kg) were used in this investigation.

Each dog, once clinically examined and found to be healthy, was wormed with pyrantel and oxantel pamoate<sup>1</sup> and vaccinated against distemper, hepatitis, and parvovirus<sup>2</sup>. The dogs were then kept in isolation facilities for three weeks before commencement of the study.

### II<sub>1</sub>. INVESTIGATIVE PROCEDURES

#### i) Radiography

Radiographs of the thorax (both dorsoventral and right lateral recumbent views) were taken using an Explorer x-ray machine<sup>3</sup> with a fast screen-film combination<sup>4</sup> and a stationary 40 lines/cm 100cm focused grid<sup>5</sup>.

#### ii) Laryngoscopy

With each dog in sternal recumbency, laryngoscopy was performed as they returned to consciousness from a light plane of anaesthesia. The laryngeal movements were observed through an endoscope<sup>6</sup> with the distal end of the scope positioned to just overlie the tip of the epiglottis. Photographs of the laryngeal airway and movements were taken. The laryngeal movements were observed until the gag reflex had returned, thus ensuring that the maximum range of movements were seen.

- 
- |   |                                                                                      |
|---|--------------------------------------------------------------------------------------|
| 1 | Cancare Plus : Ancare Distributors Ltd, 44 Raymond Terrace, Birkenhead, Auckland 10. |
| 2 | Nobivac P : Intervet International BV Boxmeer, Holland.                              |
| 3 | Picker International Ltd, P.O. Box 2, East Lane, Wembley, Middlesex, England.        |
| 4 | Standard film, Kodak Fast Screen.                                                    |
| 5 | Lysholm Focused Grid, Elema Schonander, Solna, Sweden.                               |
| 6 | Panendoscope F8, American Cytoscope Makers Inc, Watson Victo Ltd, Australia.         |

iii) Blood Gas Analysis

All arterial samples were collected from the femoral artery using an aseptic puncture technique with a 25 gauge needle. The blood was collected into an attached two ml syringe containing 0.3ml of heparinised saline (10IU/l). Each sample collected was analysed immediately using a blood gas machine<sup>7</sup>. The rectal temperature of each dog was recorded at the time of each arterial collection.

iv) Tidal Breathing Flow-volume Loop Analysis

To obtain tidal breathing flow-volume loops, each dog had a tightly fitting rubber mask held over its whole muzzle (Figure 10). The mask was equipped with a pneumotachograph<sup>8</sup> which was connected to a differential pressure transducer<sup>9</sup>. The signal from the pressure transducer was amplified to obtain a recording of inhaled and exhaled gas flow. Electrical integration by an integrator<sup>10</sup> of this flow signal was used to obtain a recording of inhaled and exhaled gas volume. Flow and volume signals were displayed on a multi channel recorder<sup>11</sup> and documented on a X-Y plotter<sup>12</sup> thereby obtaining individual tidal breathing flow volume loops (Figures 11, 12, and 13).

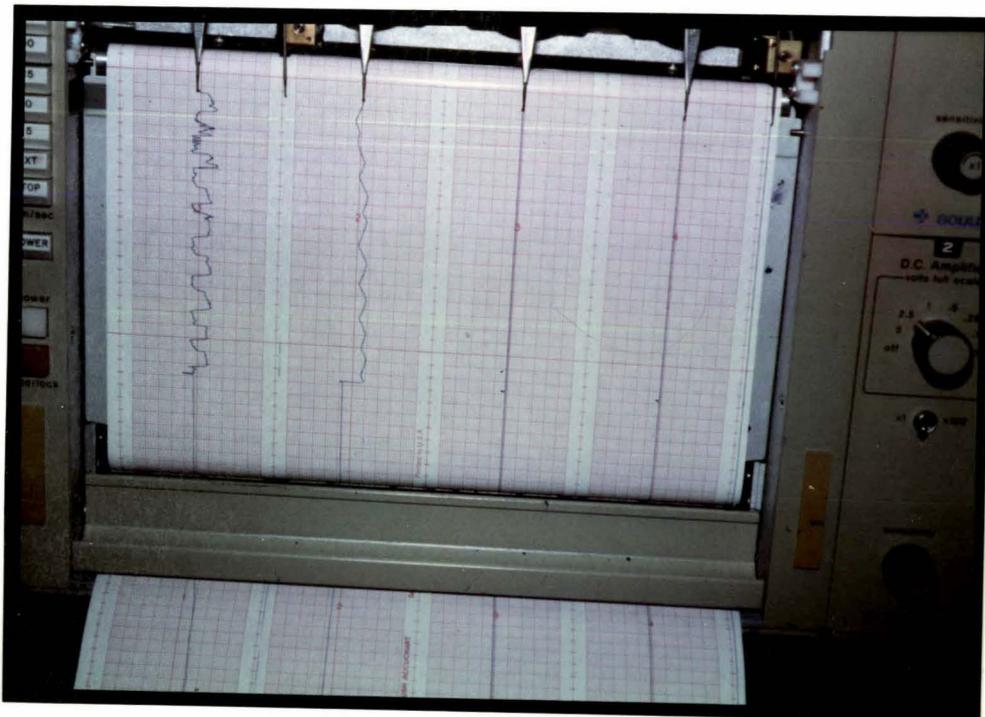
Both flow and volume signals were stored on a cassette data recorder<sup>13</sup>.

The apparatus was calibrated with room air using a rotameter for gas flow calibration and a two litre syringe for volume calibration.

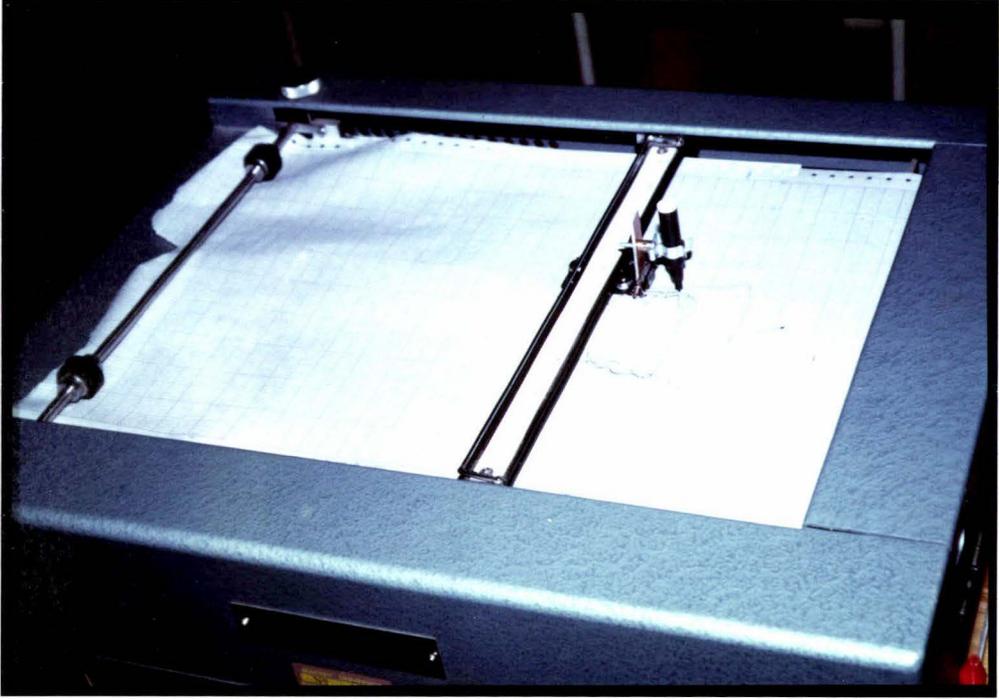
- 
- |    |                                                                                                                                          |
|----|------------------------------------------------------------------------------------------------------------------------------------------|
| 7  | ABL-300 Acid-Base Laboratory, Radiometer Ais, EM Druprej 72 DK 2400, Copenhagen N.V., Denmark.                                           |
| 8  | Fleish Pneumotachograph, OEM Medical, Whittaker Medical Manufacturing, 8741 Landmark Road, P.O. Box 27604, Richmond, Virginia 2360, USA. |
| 9  | Validyne Engineering Corp, 8626 Wilbur Ave, Northridge, CA91324-4498, USA.                                                               |
| 10 | Neomedux Systems, 17 Villiers Place, Dee Why West Sydney, NSW 2099, Australia.                                                           |
| 11 | 2400S Gould Inc, Recording System Division, 3631 Perkins Ave, Cleveland, Ohio 44114, USA.                                                |
| 12 | Model D-5N, Riken Dewhi Co. Ltd.                                                                                                         |
| 13 | Teac XR 310, Teac Corp, 3-7-3 Nak-cho, Musashino, Tokyo, Japan.                                                                          |

**Figure 13.** Photograph of one of the experimental dogs with rubber mask and pneumotachograph placed over the muzzle.

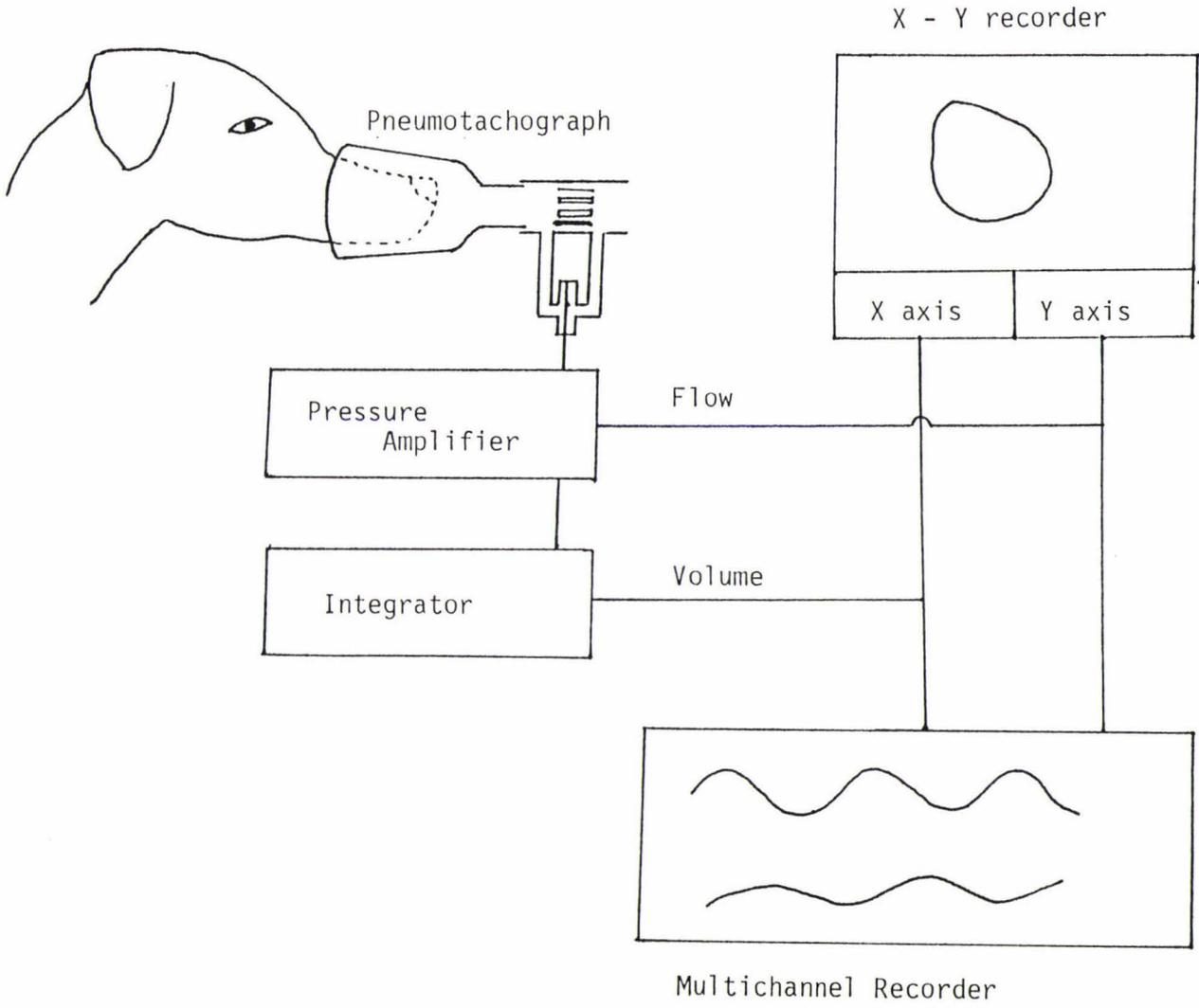
**Figure 14.** Photograph of multichannel recorder displaying the flow and volume signals.



**Figure 15.** Photograph of X-Y plotter displaying tidal breathing flow-volume loops.



**Figure 16.** Experimental arrangement used to obtain tidal breathing flow-volume loops in conscious dogs.



The dogs were evaluated in either standing or sitting positions. With the masked and attached pneumotachograph placed over the dog's muzzle, a series of four to five consecutive breaths were recorded until representative tidal flow-volume loops were obtained. Those loops acquired when the dogs were either panting or sighing, were excluded. The loops were evaluated for shape, tidal volume ( $V_T$ ), peak expiratory flow (PEF), mid tidal expiratory flow ( $EF_{50}$ ), peak inspiratory flow (PIF) and midtidal inspiratory flow ( $IF_{50}$ ) and inspiratory and expiratory gas flow rates at 25% of the tidal volume remaining to be exhaled ( $IF_{25}$  and  $EF_{25}$  respectively). Ratios of loop shape were then calculated by division of these flow measurements (ie. PEF/PIF,  $EF_{50}/IF_{50}$ , etc). Respiration rate (RR), expiratory time ( $T_E$ ) and inspiratory time ( $T_I$ ) were also measured and recorded.

## II<sub>2</sub>. SURGICAL PROCEDURES

### i) Bilateral Recurrent Laryngeal Nerve Section

Each dog was fasted for 12 hours and given preanaesthetic sedation with acepromazine<sup>14</sup> (dose rate 0.1mg/kg body weight). Anaesthesia was induced using intravenous thiopentone sodium<sup>15</sup> (dose range 15-25mg/kg body weight) and, after endotracheal intubation, maintained with a mixture of halothane and oxygen delivered through a circle system.

Each dog was then placed in dorsal recumbency and the area of skin from the thoracic inlet to the ramus of the mandible clipped, cleaned, and prepared aseptically for surgery. The head and neck were extended in order to allow the trachea and larynx to be palpated easily and the midline identified clearly.

The area was draped, and a midline skin incision made from the caudal edge of the cricoid cartilage to the mid-cervical region. The subcutaneous tissues were incised and the paired *sternohyoideus* muscles separated along the midline, and retracted laterally. A vein (*Thyroidea ima*) runs along the ventral midline between the *sternohyoideus* muscles, and divides into left and right branches at the level of the third or fourth tracheal ring. One of these branches was transected and ligated in order to allow access to the fascia overlying the trachea.

Using blunt dissection, the peritracheal fascia was parted to facilitate identification of the left and right recurrent laryngeal nerves. These nerves were situated on the dorsolateral aspect of the trachea as they passed dorsal to the thyroid glands. On the left side, the nerve

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14

ACP, C-Vet International, Bury St Edmunds, UK.

15

Intraval Sodium, May and Baker, Dagenham, UK.

is accompanied by the left caudal thyroid artery and vein, and on the right side, by the right internal jugular vein and branching right caudal thyroid vein and right thyroid artery. These vessels form a small plexus just cranial to the thyroid gland and careful dissection is required if haemorrhage is to be avoided.

In each case, a length of left and right recurrent laryngeal nerve was isolated by blunt and sharp dissection from the caudal edge of the cricoid cartilage to approximately the eighth tracheal ring. To ensure that the correct nerve was isolated, each nerve was then stimulated intermittently five or six times via electrodes from a Student stimulator and the resultant contraction of the intrinsic laryngeal muscles confirmed by palpation with the surgeon's index finger. A one cm section of the nerve was then excised, two cms from the caudal edge of the cricoid cartilage.

Closure of the cervical surgical incision was effected by placing 3/0 polyglactin 910<sup>16</sup> simple interrupted sutures to oppose the *sternohyoideus* muscles and 3/0 polyglactin 910 simple continuous sutures in the overlying subcutaneous fascia. The skin was closed with 3/0 polypropylene<sup>17</sup> simple interrupted sutures.

On completion of the surgery, the dogs were extubated and the larynx observed with a laryngoscope. The position of the vocal folds and movements of the arytenoid cartilages were viewed until the gag reflex returned.

#### ii) Castellated Laryngofissure and Vocal Fold Resection

Dogs undergoing this surgical technique were anaesthetised and prepared for surgery as described for recurrent laryngeal nerve resection, except that the area of skin prepared was extended rostrally to the mid mandibular region.

With the dog in dorsal recumbency, the midline neck incision from the previous surgery was reopened at the level of the fifth to sixth tracheal ring. Using a No. 11 scalpel blade<sup>18</sup>, a "T" shaped opening was made through two to three tracheal cartilages into the tracheal lumen. The endotracheal tube was removed and a 10mm cuffed tracheostomy tube<sup>19</sup> inserted through the tracheal opening. Once the tube was correctly positioned, the cuff was inflated

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16	Coated Vicryl, Ethicon Inc. Sommerville, New Jersey, 08876-0151, USA.
17	Prolene, Ethicon Inc., Sommerville, New Jersey, 08876-0151, USA.
18	Surgical blades, Swann-Morten Ltd, Sheffield, UK.
19	Blue line cuffed tracheostomy tube, Portex Ltd, Hythe, Kent, UK.

and the tube secured in place by skin sutures. Anaesthesia was maintained throughout the remainder of the surgery by administering a halothane and oxygen mixture through this tube.

With the dog remaining in dorsal recumbency, the neck and head were extended and the skin over the ventral region of the larynx prepared for surgery. A midline skin incision was made from the basioid bone to the second tracheal ring (Figure 17) and continued through the subcutaneous fascia. The paired *sternohyoideus* muscles were parted proximally along the midline and retracted to expose the thyroid and cricoid cartilages (Figure 18). The overlying fascia was removed from the ventral aspect of the thyroid cartilage and the rostral and caudal borders of this cartilage identified. Two points, equidistant from each other and the rostral and caudal borders of the thyroid cartilages, were placed on the mid-ventral line of the thyroid prominence. Two further marks were then made on the right thyroid lamina, approximately 1cm away from the midline and at right angles from the former marks on the thyroid prominence. A castellated incision joining the marks described above was then made through the thyroid cartilage using a No. II scapel blade<sup>18</sup>. This incision was then extended caudally along the midline of the cricothyroid membrane (see Figures 19 and 20).

Using Mayo scissors, the laryngeal mucosa was opened along the same castellated line, with care taken to avoid excessive surgical injury to the ventral attachments of the vocal folds. The two thyroid laminae and associated structures were reflected laterally by retractors<sup>20</sup> to allow inspection of the ventricular and vocal folds (Figure 21). Excision of the dorsal two-thirds of each vocal fold was accomplished by grasping them with rat toothed forceps and cutting with a curved Knapp Strabismus<sup>21</sup> (10cm) scissors. Haemorrhage was controlled by applying pressure. Blood clots, which formed within the laryngeal lumen were removed with saline dampened swabs.

The previously created central flap of the thyroid cartilage was aligned with the incised rostral edge of the right thyroid lamina (Figure 22), and stabilised in this position by four single interrupted sutures of 3/0 polypropylene. Two of the sutures were placed through the central flap and the opposing rostral thyroid cartilage segment (Figure 23) and two through the rostral edge of the central flap and around the basioid bone (Figure 24 and 24a). All sutures were preplaced before tightening. Care was taken to avoid puncturing or occluding the hyoid venous arch just caudal to the basioid bone. Swaged-on needles were used to minimise the risk of causing laceration or fracture of the cartilage whilst the sutures were being placed.

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20

Kilner skin retractors, Thackray Instruments, P.O. Box HP177, Shine Oak Street, Auckland.

21

Knapp Strabismus Scissors (10cm). Medical Instruments, 8200, Tuttlingen, Germany.

**SEQUENTIAL VIEWS**

**OF SURGICAL PROCEDURE**

**FOR CASTELLATED LARYNGOFISSURE**

**AND VOCAL FOLD RESECTION**

**IN THE DOG**

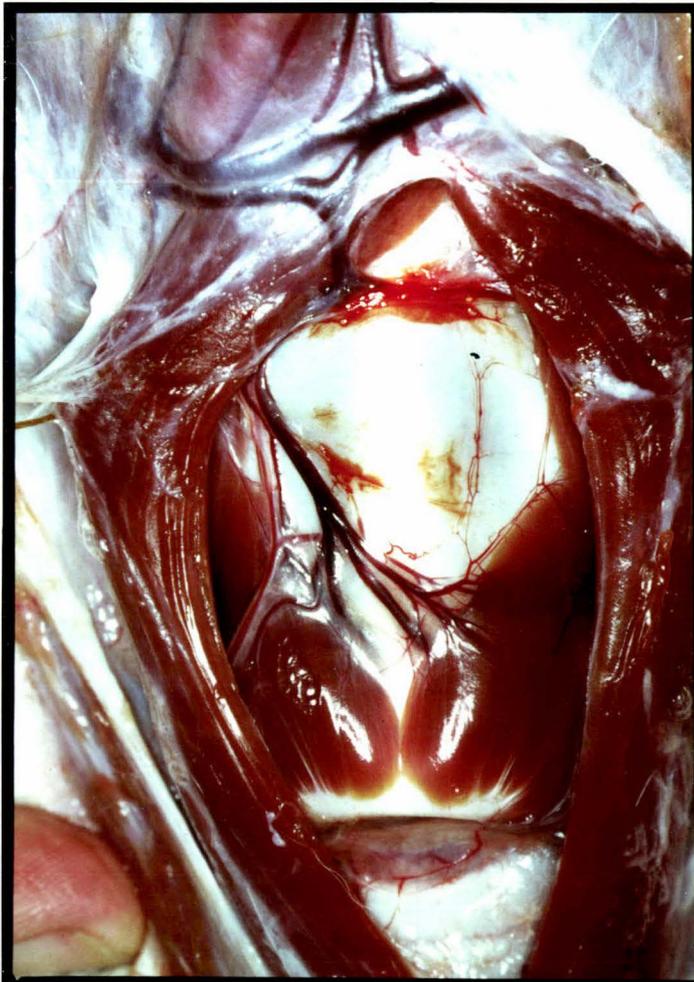
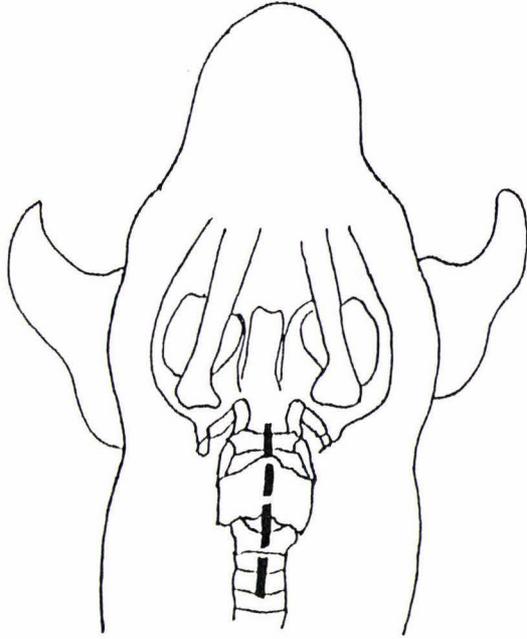
**Figure 17**

Dotted lines show the site of the ventral midline skin incision over the larynx.

The views of the larynx in the following pictures have been taken with the dog maintained in the same position, as shown in this diagram.

**Figure 18**

Photograph of the ventral aspect of the larynx. The paired *sternohyoideus* muscles have been separated along the ventral midline and retracted laterally.

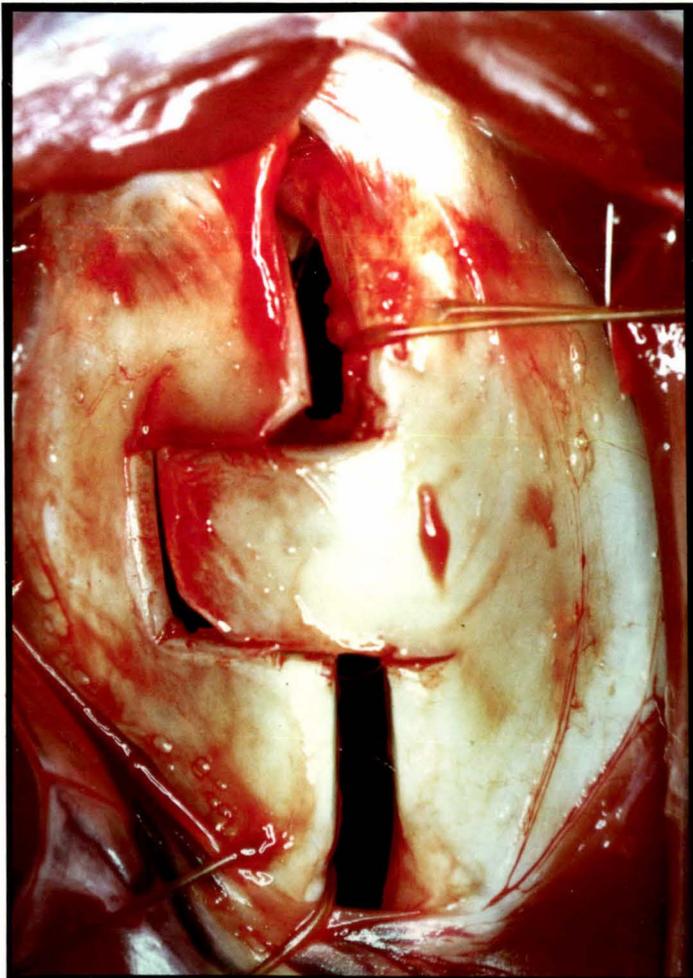
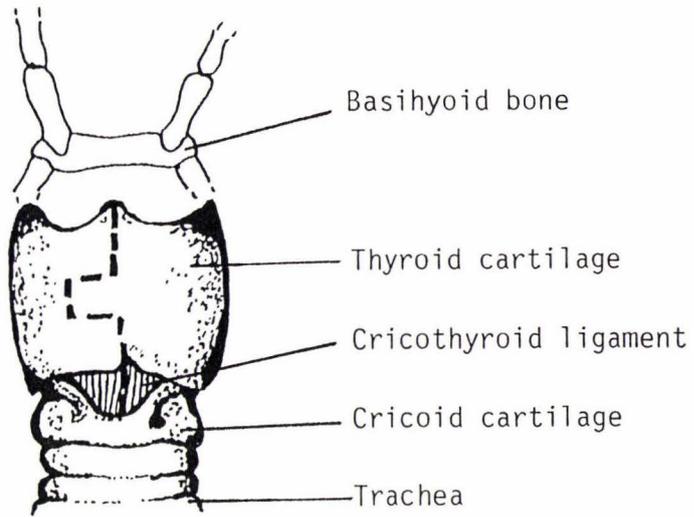


**Figure 19**

Diagrammatic representation of the ventral aspect of the larynx. The dotted line represents the castellated incision made through the thyroid cartilage and cricothyroid ligament.

**Figure 20**

Photograph of castellated incision through the thyroid cartilage and cricothyroid ligament.

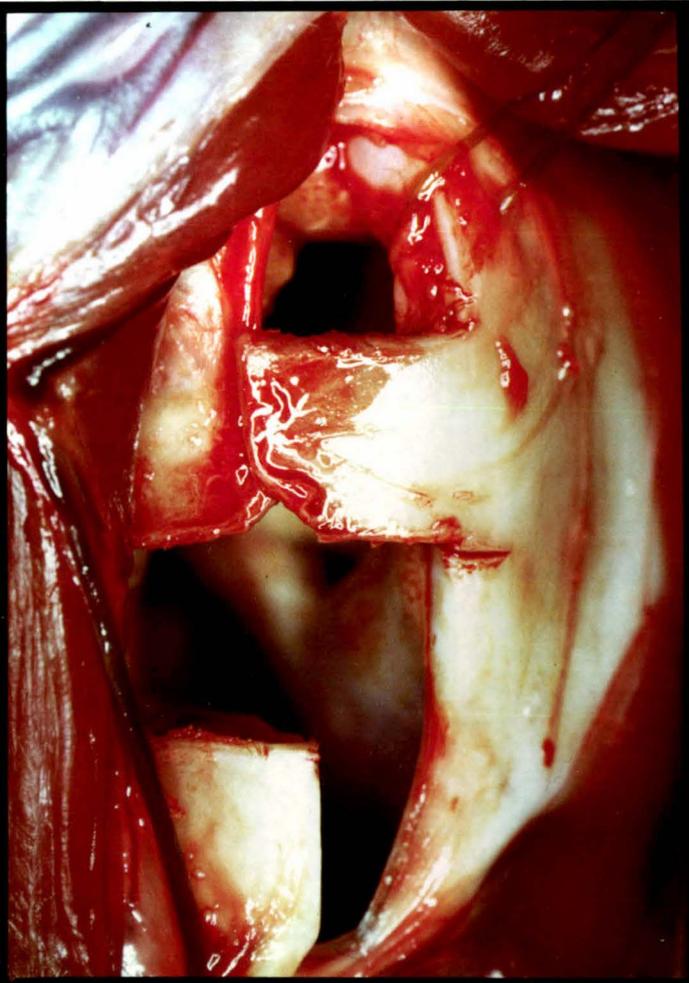
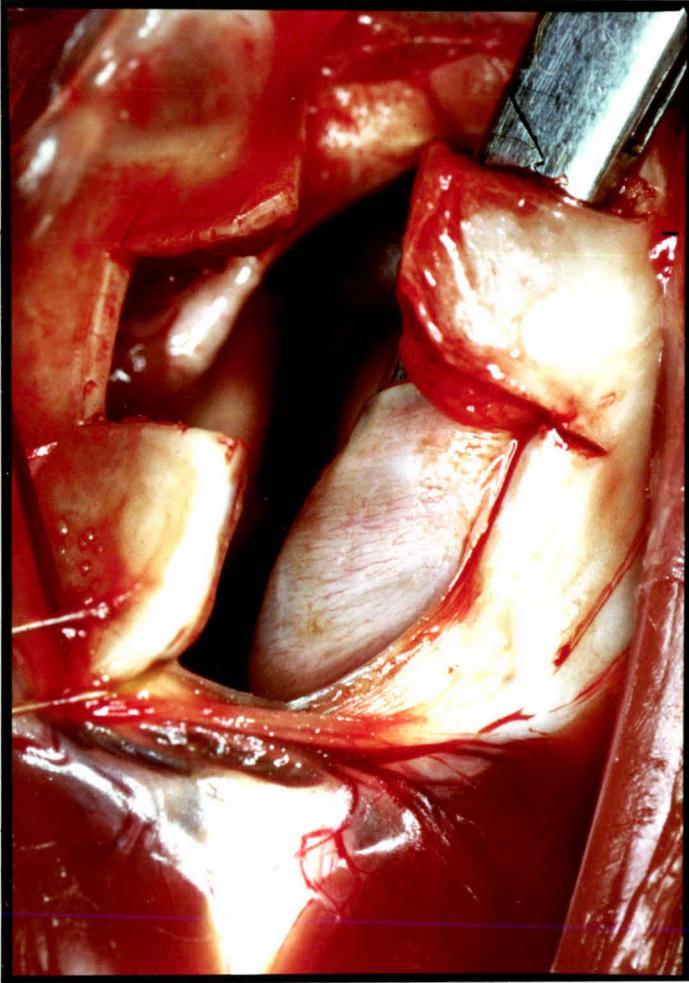


**Figure 21**

The two thyroid segments have been separated along the castellated incision. Mosquito forceps have been placed into the laryngeal saccule and are displacing the vocal fold into the lumen to allow for easy inspection. The vocal folds are excised at this stage.

**Figure 22**

The caudal flap of the left lamina of the thyroid cartilage has been aligned to the opposing cranial flap of the right lamina.

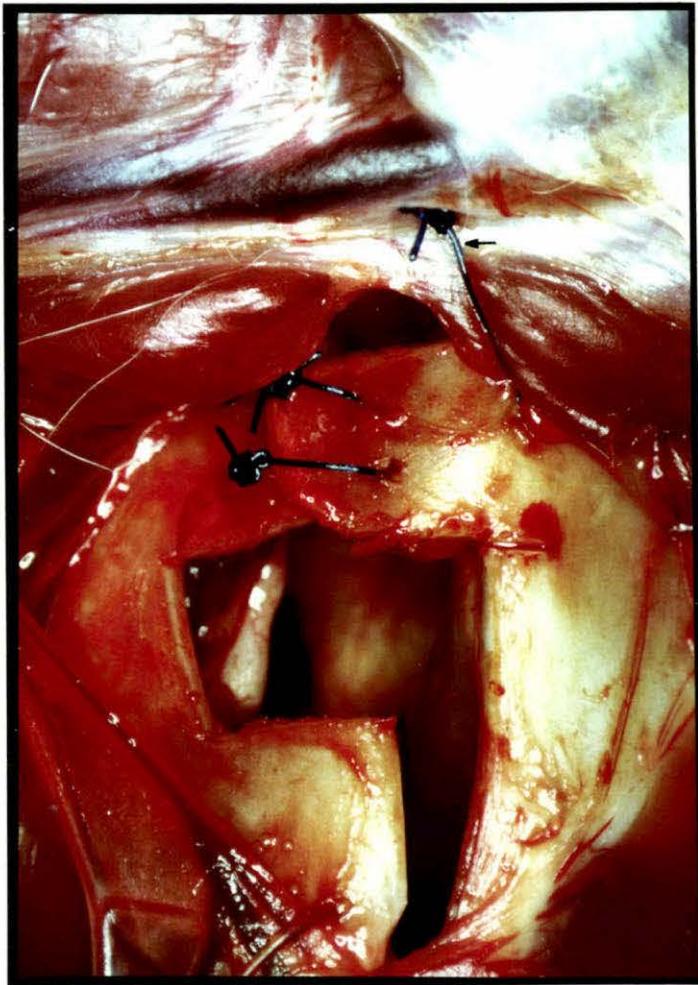
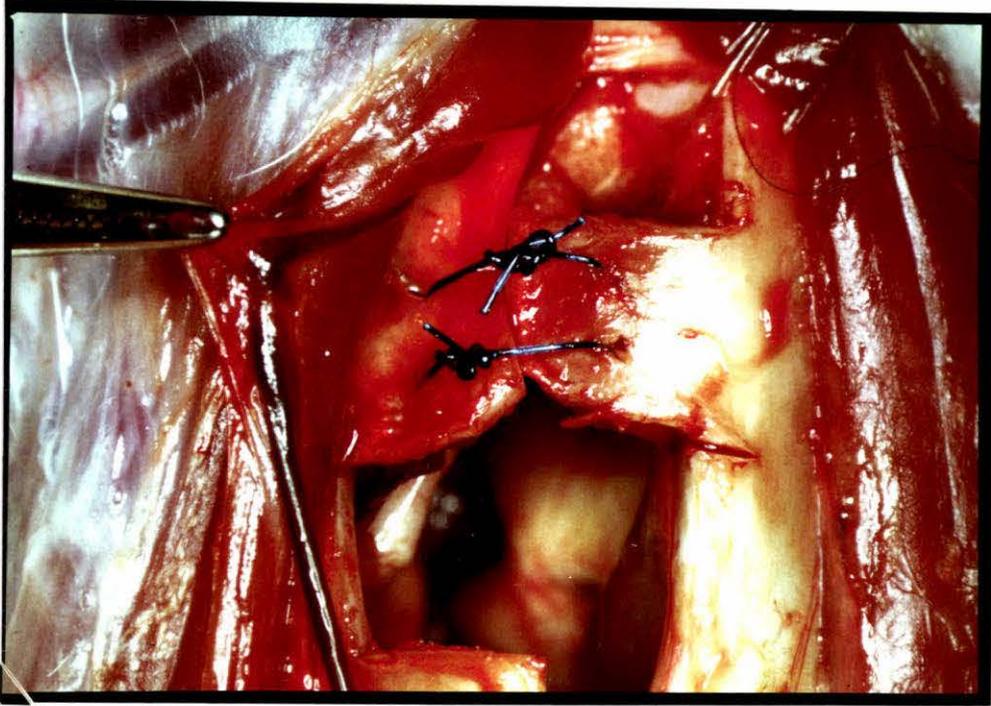


**Figure 23**

Two polypropylene simple interrupted sutures have been placed across the flaps made in the thyroid cartilage.

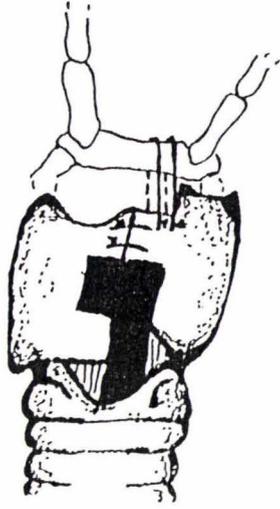
**Figure 24**

An additional polypropylene suture (arrow) has been placed from the repositioned caudal flap of the left thyroid lamina around the basihyoid bone. This bone lies beneath this *sternohyoideus* muscles.



**Figure 24a**

Diagrammatic representation of the completed castellated laryngofissure.



The cricothyroid membrane and overlying *sternohyoideus* muscles were then opposed with 3/0 polydioxanone<sup>22</sup> simple interrupted sutures to close the remaining laryngeal defect. The subcutaneous fascia was closed using a simple continuous suture of 3/0 polyglactin 910 and the skin with 3/0 polypropylene simple interrupted sutures.

The anaesthesia was discontinued and the animal allowed to recover, with the tracheostomy tube in place. Once a swallowing reflex had returned, buprenorphine<sup>23</sup> (a synthetic opiate) was given intravenously at a dose rate of 0.01mg/kg body weight to produce analgesia.

Once the dog had achieved sternal recumbency, the tracheostomy tube was removed (approximately two to four hours after surgery). The tracheostomy wound was left open but cleaned twice daily with swabs dampened with aqueous hibitane (0.5%) until healing occurred.

### iii) Bilateral Arytenoid Lateralisation

The five dogs undergoing this surgical technique were premedicated with acepromazine (dose rate 0.1mg/kg body weight) and anaesthesia induced with thiopentone sodium intravenously. After endotracheal intubation, anaesthesia was maintained with the patient breathing a mixture of halothane and oxygen through a circle system.

An area of skin on both sides of the neck, extending from the base of the ears over the jugular furrows to the proximal trachea was clipped and prepared for surgery. The dog was positioned in right lateral recumbency with the nose and ventral neck tilted downwards at an angle of 30 degrees from the horizontal.

A longitudinal skin incision, five centimetres in length was made one centimetre below the junction of the external jugular and external maxillary veins (Figure 25). The incision was then extended through the subcutaneous fascia, platymus muscle and fat.

Sharp dissection was continued over the dorsolateral aspect of the larynx until the *thyrohyoideus* and *sternothyroideus* muscles could be seen and the thyroid cartilage palpated. Care was taken not to sever the small artery which crosses the lateral aspect of the cricothyroid muscle.

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22

PDS, Ethicon Inc., Sommerville, New Jersey, 08876-0151, USA.

23

Temgesic, Rickett and Colman, Avondale, Auckland.

**DIAGRAMS SHOWING**

**SURGICAL PROCEDURE**

**OF BILATERAL**

**ARYTENOID LATERALISATION**

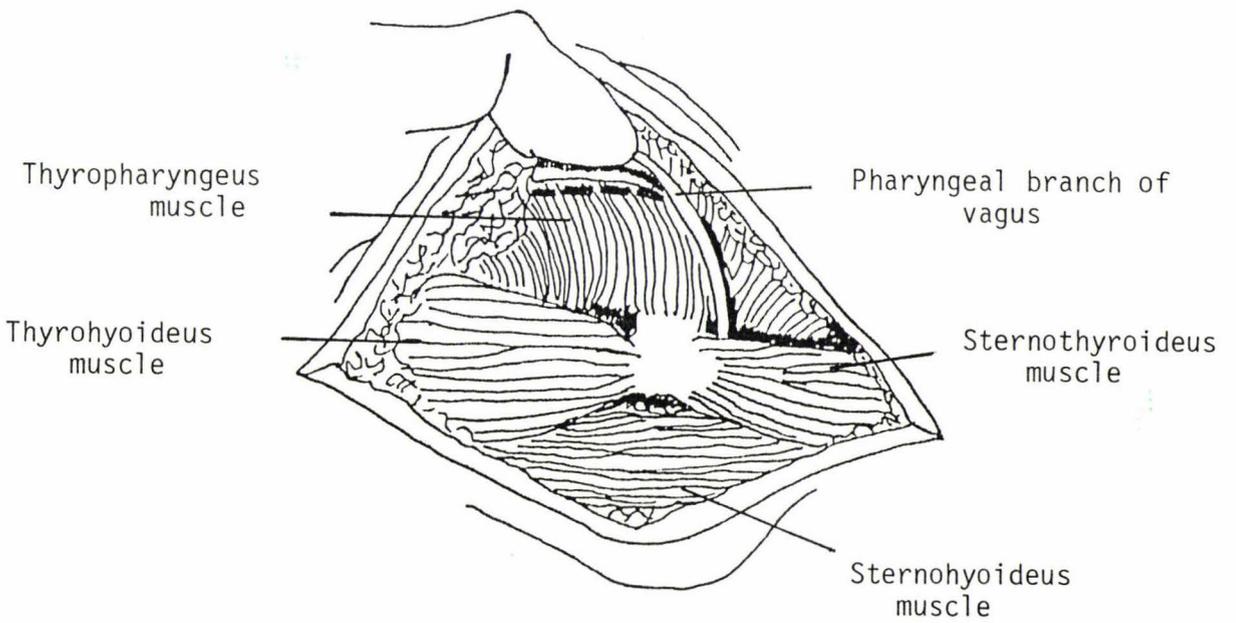
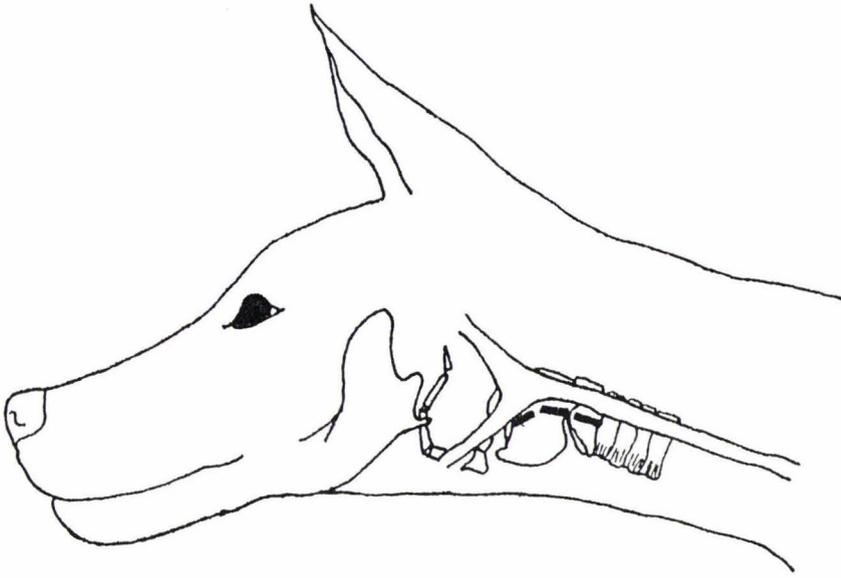
**OF THE DOG**

**Figure 25**

Dotted lines shows site of skin incision ventral to the external jugular and maxillary veins.

**Figure 26**

The surgeon's finger has been inserted medial to the thyroid lamina. The dotted line shows the site of transection of the *thyropharyngeus* muscle.

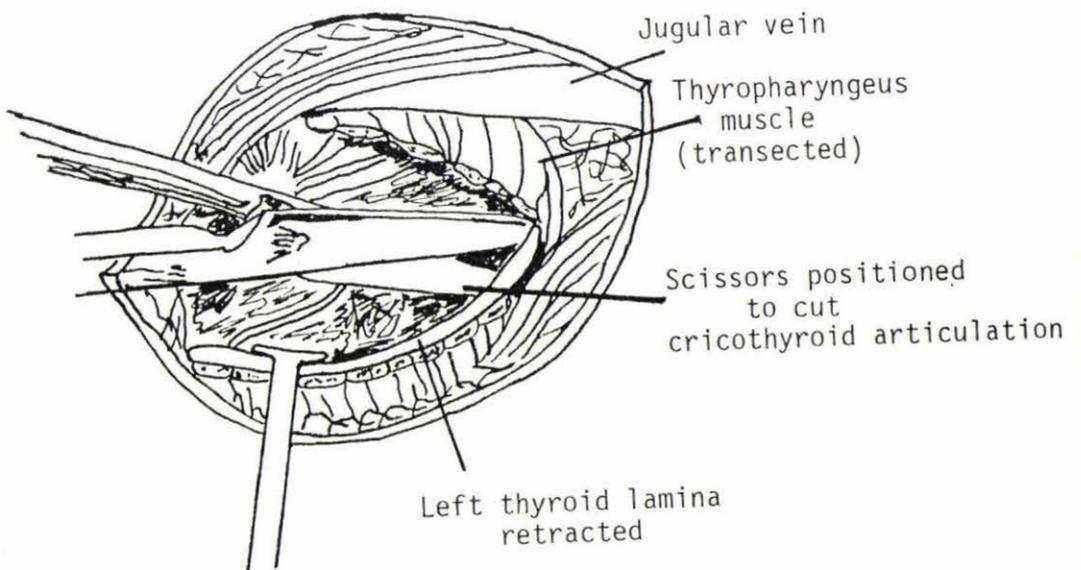
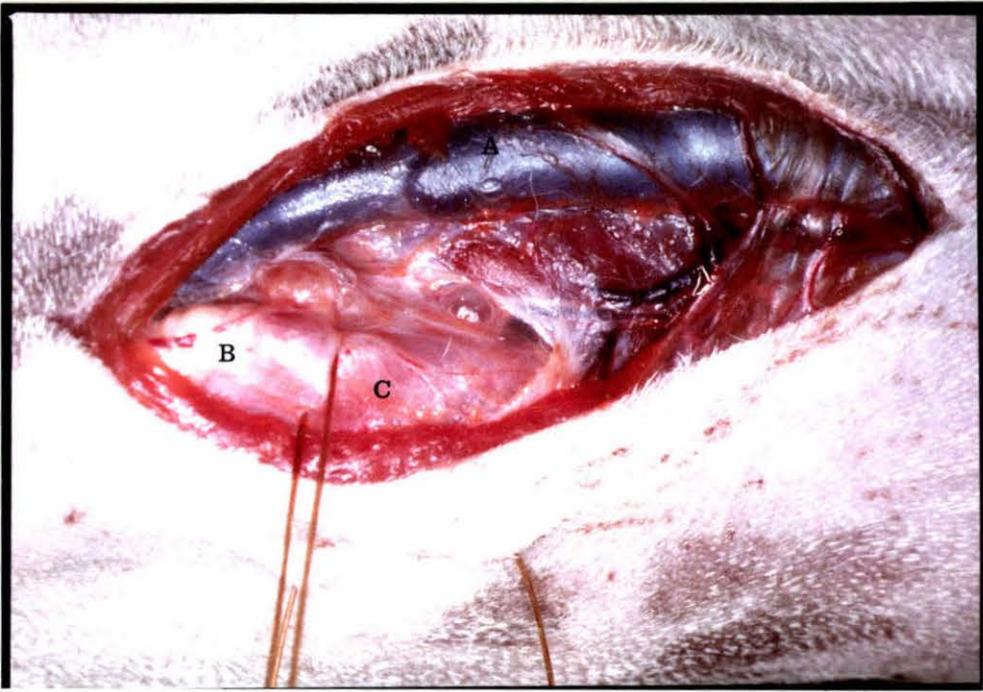


**Figure 27**

Photographs of the dissection of the lateral approach to the larynx showing the external jugular vein (A) and the retracted (with suture material) dorsal border of the left lamina of the thyroid cartilage (B). The *thyropharyngeus* muscle (C) can be seen overlying the thyroid lamina.

**Figure 28**

The *thyropharyngeus* muscle has been transected. The scissors are positioned to cut the cricothyroid articulation.

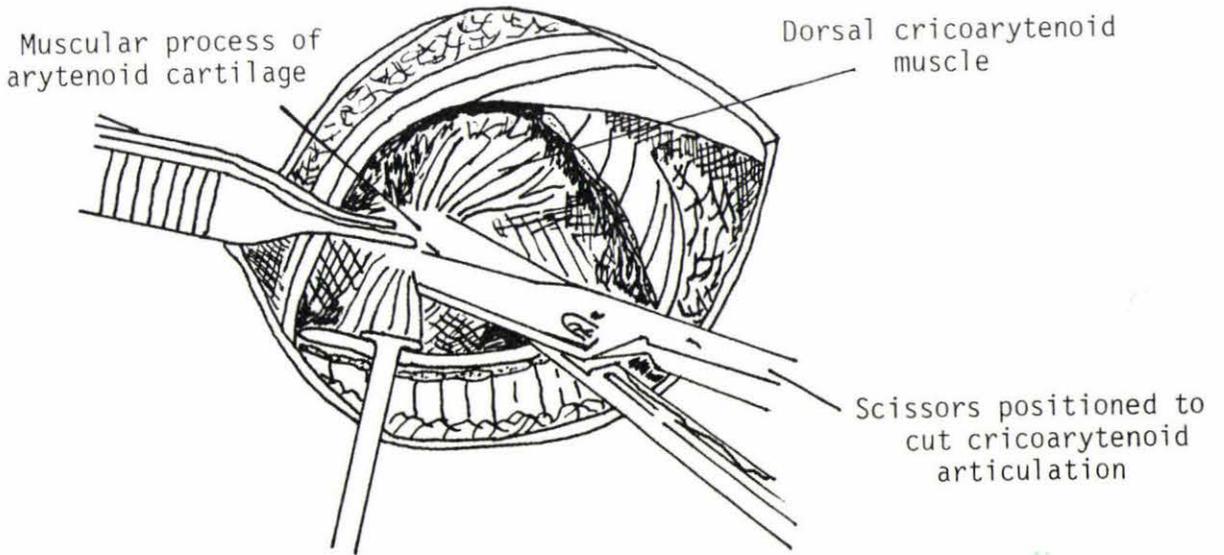


**Figure 29**

Photograph of the larynx after cricothyroid disarticulation showing intrinsic laryngeal muscles (A). The muscular process of the arytenoid cartilage lies beneath the thyroid lamina.

**Figure 30**

With the left thyroid lamina retracted, the scissors are placed to divide the cricoarytenoid articulation.

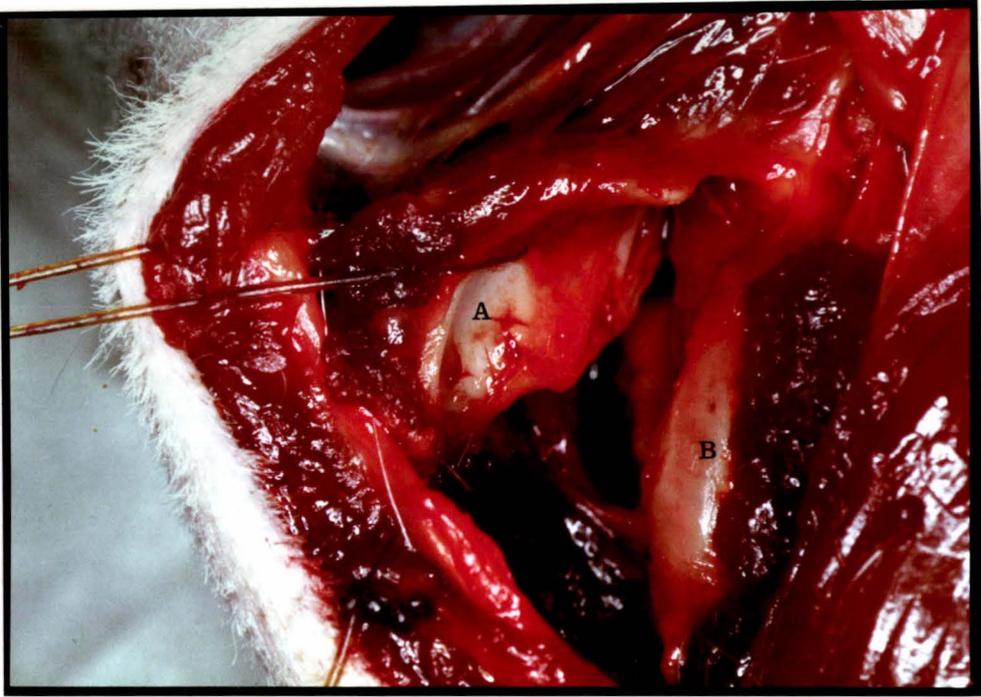


**Figure 31**

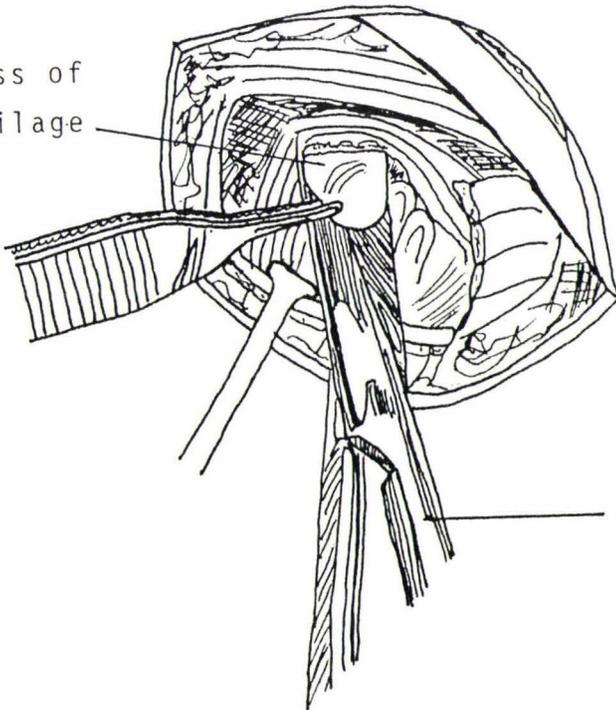
Photograph showing articular surfaces of cricoarytenoid joint (A and B) after rostradorsal retraction of the muscular process of the left arytenoid cartilage by suture material.

**Figure 32**

With the muscular process of the arytenoid cartilage retracted the scissors are inserted medially to divide the sesamoid band connecting the apices of the paired arytenoid cartilages dorsally.



Muscular process of  
arytenoid cartilage



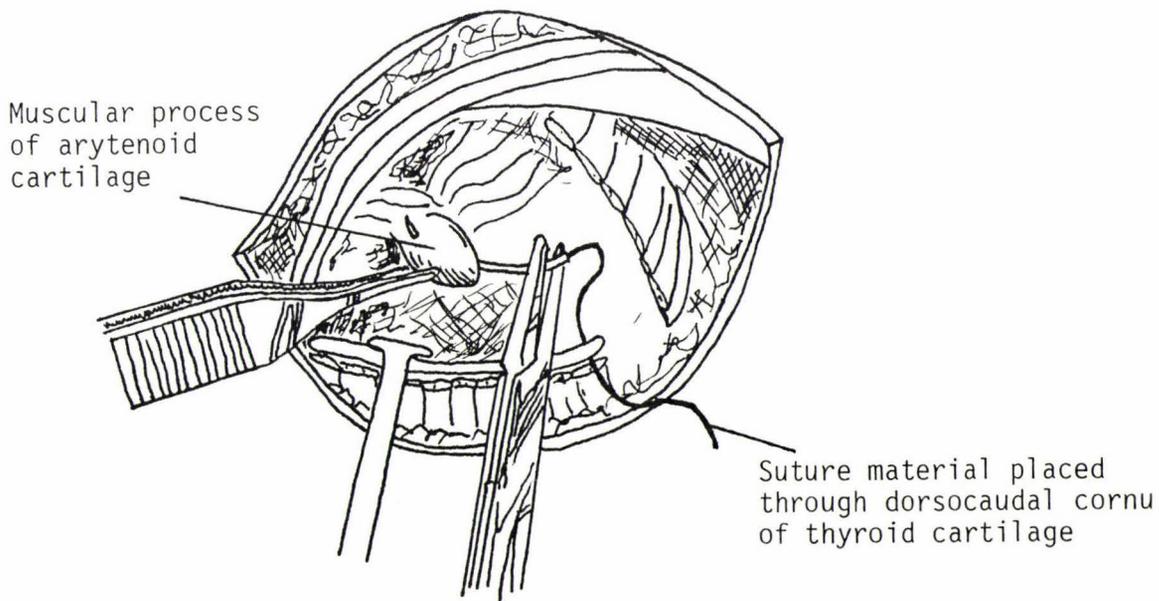
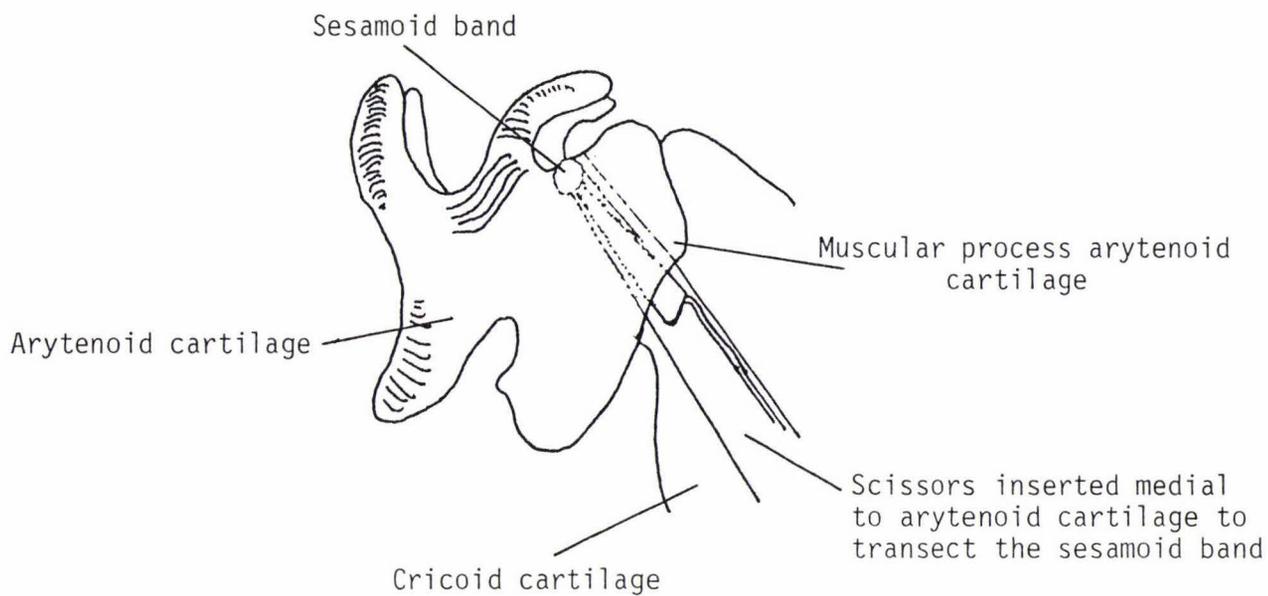
Scissors positioned  
medial to muscular  
process to cut  
sesamoid band

**Figure 32a**

Diagram showing positioning of the scissors medial to the muscular process of the arytenoid cartilage to achieve the division of the sesamoid band.

**Figure 33**

A suture is placed through the caudal cornu of the thyroid cartilage and muscular process of the arytenoid cartilage.

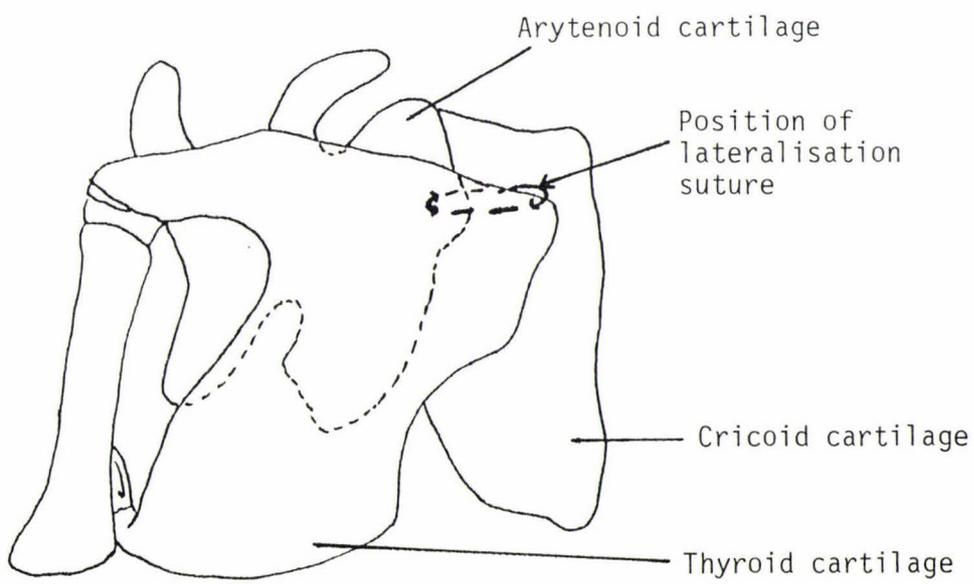
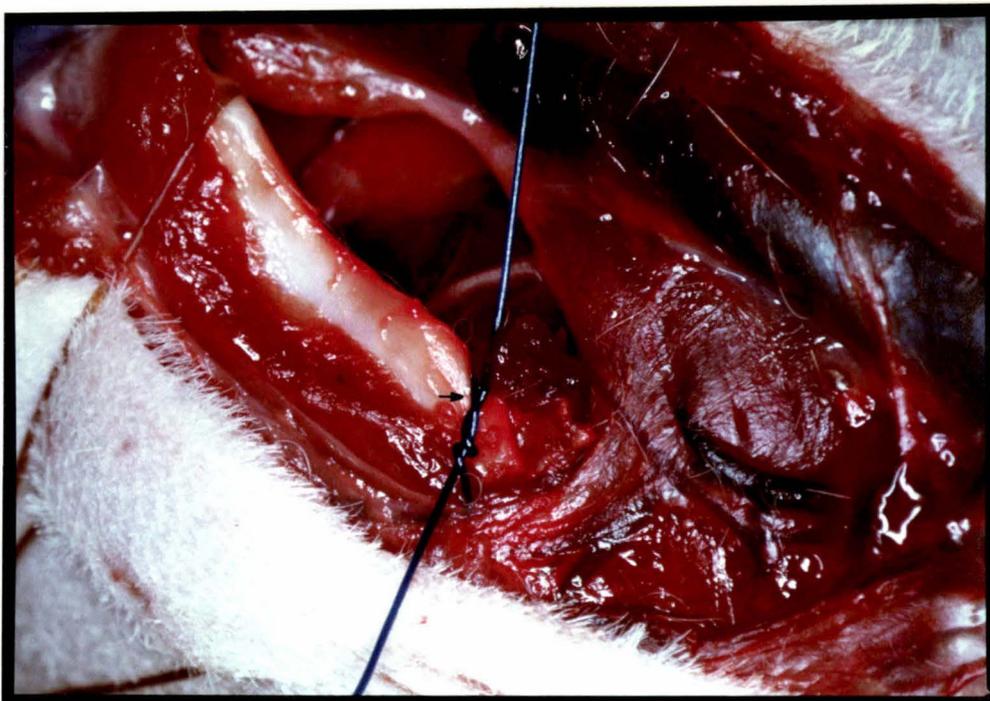


**Figure 34**

Photograph showing the position and tying of suture between the caudal cornu of the thyroid cartilage (arrow) and the muscular process of the arytenoid.

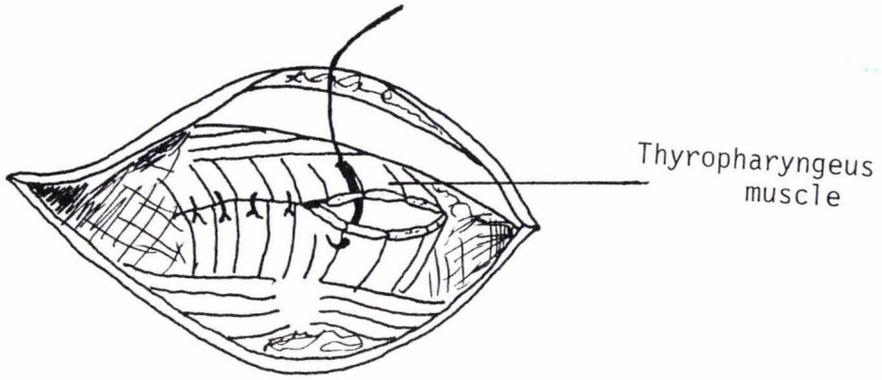
**Figure 34a**

Diagrammatic representation of the position of the lateralisation suture through caudal cornu of the thyroid cartilage and muscular process of arytenoid cartilage.



**Figure 35**

The incision in the *thyropharyngeus* muscle is closed by simple interrupted absorbable sutures.



The dorsocaudal rim of the left thyroid lamina was then palpated. The surgeon's finger was inserted medial to the thyroid lamina and the larynx rotated into the wound (Figure 26 and 27) thus exposing the *thyropharyngeus* and *cricopharyngeus* muscles. The *thyropharyngeus* muscle and underlying fascia were sharply transected along the dorsal rim of the thyroid lamina (Figure 26) and the exposed edge of the thyroid cartilage was then retracted laterally. Keeping the blade of the scissors close to the dorsomedial surface of the thyroid cartilage (Figure 28), and cutting blindly in a caudal direction, the cricothyroid articulation was separated, thus exposing the intrinsic laryngeal muscles (Figure 29).

The muscular process of the left arytenoid cartilage was then identified by palpation as a solid nodular structure and by viewing the attached insertions of the dorsal and lateral cricoarytenoid muscles. It was grasped with rat-toothed forceps and, with the scissor blade inserted between it and the cricoid cartilage (Figure 30), cuts were made in a craniomedial direction to disarticulate the cricoarytenoid joint. Once free, the muscular process was further retracted rostrally and laterally away from the cricoid cartilage (Figure 31). The blade of the scissors was then placed medial to the muscular process and, following in a rostro-dorsal direction (Figure 32 and 32a), the sesamoid band between the arytenoids was transected blindly in the dorsal midline. Once separation was achieved, the left arytenoid cartilage could be freely moved with finger pressure. During the blind midline section, care was taken to minimise the chances of penetration of the laryngeal mucosa.

A single suture of 2/0 polypropylene was placed through the caudodorsal border of the thyroid cartilage and the muscular process of the arytenoid cartilage (Figure 33). It was then tied tightly using a four knot throw (Figure 34). The effect was to pull the left arytenoid cartilage caudally and thus produce abduction.

The thyroid lamina was returned to its normal position and the severed *thyropharyngeus* muscle re-united with simple interrupted sutures of 3/0 polydioxanone (Figure 35). The subcutaneous fascia was closed with simple continuous sutures of the same material and the skin by 3/0 polypropylene using a simple interrupted technique.

The patient was then repositioned on its left side and an identical procedure (apart from separation of the arytenoid cartilages) performed on the right side of the larynx.

After completion of surgery, the patient was allowed to recover from anaesthesia and, following extubation, analgesia was provided by administering buprenorphine intravenously (dose rate 0.01mg/kg body weight).

II<sub>3</sub>-

## EXPERIMENTAL PROCEDURE

At commencement of the experiment, each dog was clinically examined and radiographs of the chest taken to detect the presence of any co-existent lower respiratory tract disease. An arterial blood sample for blood gas measurement was taken from each dog at rest and then within 20 seconds after exercise. The exercise consisted of a 100m run performed within 20 seconds. Each sample was analysed immediately after collection and the haemoglobin concentration, blood oxygen and carbon dioxide tensions recorded. Tidal breathing flow-volume loop analysis was then carried out, and a predominant loop pattern was determined for each dog. Flow, volume and loop tracings were recorded.

Each dog was then anaesthetised for resection of both recurrent laryngeal nerves. Prior to this procedure being performed, laryngeal function was assessed. This was achieved by endoscope examination *per os* after the dog had been extubated. The larynx and laryngeal movements were viewed through the endoscope as the animal recovered from the anaesthesia. Several endoscopic photographs were taken both during the inspiratory and expiratory phases of the respiratory cycle so that the size of the *rima glottidis* and the nature and degree of arytenoid movements could be determined. Immediately following these assessments, the animal was re-intubated and anaesthetised as before until a surgical plane of anaesthesia was produced.

Bilateral resection of the recurrent laryngeal nerves was then performed and all the dogs allowed to recover.

The dogs were examined clinically every 12 hours for signs of respiratory changes. Forty eight hours after recurrent laryngeal nerve resection, blood gas analysis, at rest and after exercise, was recorded again. A further tidal breathing flow-volume loop pattern was also established. The dogs were then re-anaesthetised and photographs of the larynx taken. Once photographs via an endoscope had been obtained, the patient was reintubated and a surgical plane of anaesthesia produced. One of the two laryngeal surgical procedures described was then performed. Five dogs received a castellated laryngofissure with vocal fold resection, and a further five, bilateral arytenoid lateralisation.

Once recovered from anaesthesia, the dogs were examined clinically every 12 hours for the following seven days. Skin sutures were removed nine days post operatively. Five weeks later, each animal was given a thorough clinical examination. Radiographs of their chest and blood gas analysis at rest and after exercise were repeated. A tidal breathing flow-volume loop was also recorded.

The animals were re-anaesthetised six weeks after surgery. Laryngoscopic examination and photographs of the larynx were obtained, as before. The animals were then humanely killed with intravenous barbiturates and an autopsy examination performed immediately. During the latter procedure careful dissection of the larynx, site of recurrent larynx nerve resection, tracheostomy site, trachea, and lungs was performed and assessment of any changes noted. Photographs of the larynx were taken to record the size and shape of the *rima glottidis*, and the appearance of the dorsal cricoarytenoid muscles, and tracheostomy sites.

II<sub>4</sub>.

#### STATISTICAL ANALYSIS OF RESULTS

The students t test was used to statistically analyse the blood gas and tidal breathing flow-volume loop results.

When considering matters of statistical significance, the convention of Brookes, Bettely and Loxstan (1966) was used. In accepting or rejecting a null hypothesis that no relationship existed, the following criteria was used.

Probability	Conclusion
Greater than 0.1	Not significant Hypothesis accepted
<0.1 to 0.05	Possibly significant Some doubt cast on Hypothesis but further evidence need be sought before rejection
<0.05 to 0.01	Significant Hypothesis rejected
<0.01 to 0.001	Highly significant Hypothesis confidently rejected
<0.001	Very highly significant Hypothesis confidently rejected

**CHAPTER III.****RESULTS****III<sub>1</sub>. CLINICAL ASSESSMENT****a) Examination Prior to Neurectomy**

No significant abnormalities were found in any of the dogs at the initial clinical examination.

**b) Examination After Neurectomy**

After bilateral recurrent laryngeal nerve resection, all dogs had a hoarse bark and an inspiratory stridor. The latter sign was most marked during, and immediately after, exercise. Reduced exercise tolerance was noted in all neurectomised dogs during vigorous physical activity, but was not discernible when the dogs were walking. Awkward gulping and swallowing movements were observed in all dogs immediately after recovery from the neurectomy and during their first post-operative feed. However, this sign diminished rapidly and was no longer apparent by the second post-operative day. One other clinical sign that occurred during the first 48 hours after neurectomy in three of the dogs (Nos. 2, 5 and 8) was a soft intermittent cough.

**c) Examination After Castellated Laryngofissure and Vocal Fold Resection**

All five dogs recovered from the second surgical procedure of castellated laryngofissure and reached sternal recumbency between two and four hours after surgery. The surgical wounds healed satisfactorily. The tracheostomy sites healed by secondary intention within two weeks of surgery. All of the dogs in this group had a soft intermittent cough for the first seven to ten days following surgery. This cough was exacerbated by exercise. When examined six weeks post-operatively, all dogs showed improved exercise tolerance compared to the neurectomised stage. Two dogs (No. 1 and 9) had an inspiratory stridor on exercise. One dog (No. 2) frequently coughed and also had occasional bouts of cyanosis which appeared to be related to periods of excitement or stress, rather than to exercise. This respiratory embarrassment was exacerbated by cold (<5° Celcius) climatic conditions. No abnormalities were found on clinical examination in the remaining two dogs (No. 6 and 10.).

**d) Examination After Bilateral Arytenoid Lateralisation**

All of the dogs in this group recovered from the surgical procedure of bilateral arytenoid lateralisation and reached sternal recumbency between two and four hours after surgery. All surgical wounds healed satisfactorily.

Of the five dogs in this group, blood was found on the endotracheal tube in two (Nos 5. and 7.). This was taken to indicate some penetration of the laryngeal mucosa at the time of surgery. Both of these dogs coughed intermittently for 24 hours after the operation and one (No 5.) also experienced some difficulty in swallowing food during the first 36 hours after surgery. When examined six weeks post-operatively, all of the dogs showed improved exercise tolerance from the post neurectomy stage. No abnormalities were found on clinical examination of the five dogs in this group.

### III<sub>2</sub>.            RADIOGRAPHY

Thoracic radiographs of all the dogs taken on the two occasions during the experiment revealed normal lung patterns.

### III<sub>3</sub>.            LARYNGOSCOPY

Photographs of the larynxes are shown in Figures 36 to 45.

#### a)                Examination Prior to Neurectomy

The initial laryngoscopic observations were similar in all dogs. Under surgical plane of anaesthesia the *rima glottidis* was partially open and appeared symmetrical. There was minimal movement of the vocal folds and arytenoid cartilages during the respiratory cycle. As the animal began to recover from anaesthesia, the vocal folds were progressively abducted, but not always in a symmetrical manner. The degree of arytenoid movement varied, but as the larynx was observed two to three times from surgical plane anaesthesia to that which allowed a return of the swallowing reflex, each dog did exhibit abduction of both vocal folds and arytenoid cartilage movement. The stage of anaesthesia at which arytenoid movement was first observed varied in each dog. However, arytenoid abduction was always seen, and was usually symmetrical once the swallow reflex had returned. Arytenoid abduction was associated with the inspiratory phase of the breathing cycle. In all instances, the arytenoid cartilages and vocal folds would return to their resting position after expiration. Occasionally the vocal cords would flutter in the expiratory air flow.

#### b)                Examination After Neurectomy

Observation of the larynx after recurrent laryngeal nerve resection revealed that, in all dogs, the arytenoid cartilages and vocal folds were positioned medially, thus reducing the width of the *rima glottidis*. No abduction of the cartilages or vocal folds was seen as the dogs

**Photographs of larynges**  
**taken at**  
**endoscopic examination**  
**of the**  
**ten experimental dogs.**

**Figure 36. DOG 1**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

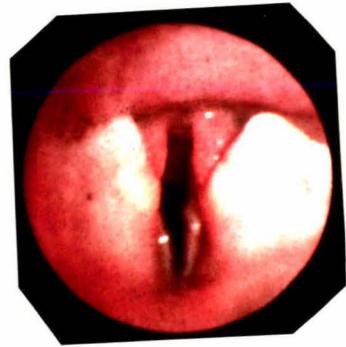
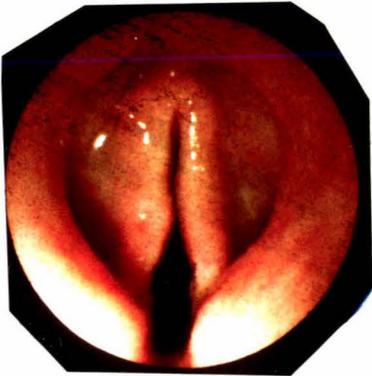
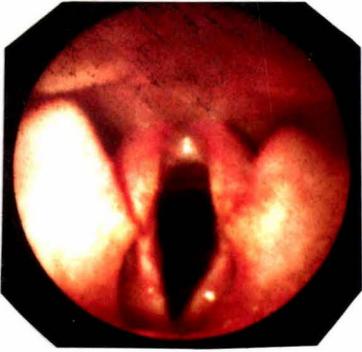
C) Appearance six weeks after castellated laryngofissure and vocal fold resection.

**Figure 37. DOG 2**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after castellated laryngofissure and vocal fold resection.



**Figure 38. DOG 6**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

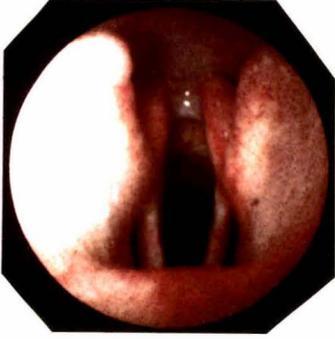
C) Appearance six weeks after castellated laryngofissure and vocal fold resection.

**Figure 39. DOG 9**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after castellated laryngofissure and vocal fold resection.

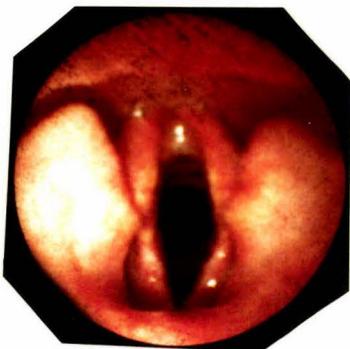
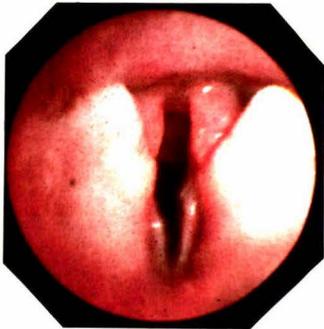
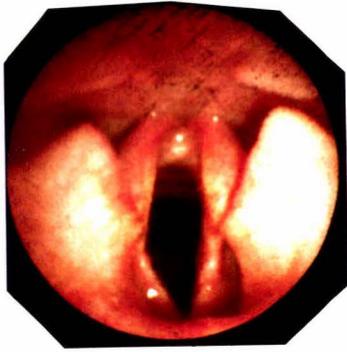


**Figure 40. DOG 10**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after castellated laryngofissure and vocal fold resection.



**Figure 41. DOG 3**

**Figure 42. DOG 4**

A) Appearance prior to neurectomy.

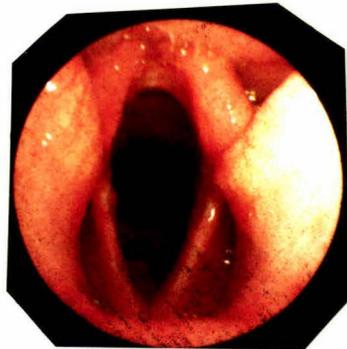
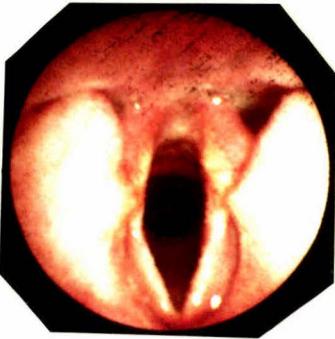
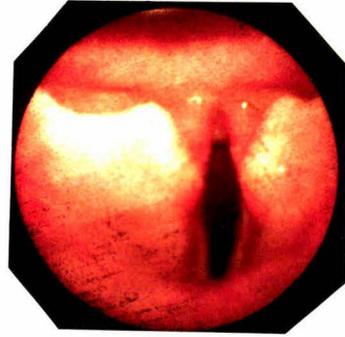
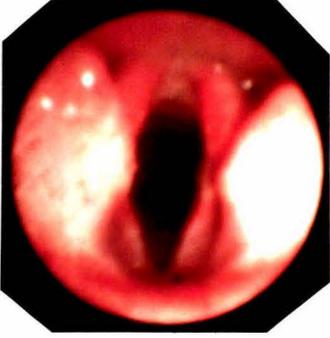
A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after bilateral arytenoid lateralisation.

C) Appearance six weeks after bilateral arytenoid lateralisation.



**Figure 43. DOG 5**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

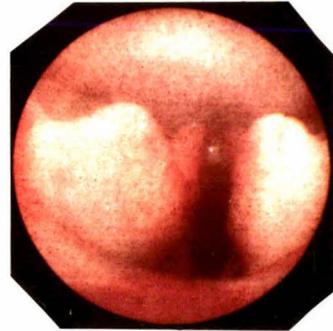
C) Appearance six weeks after bilateral arytenoid lateralisation.

**Figure 44. DOG 7**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after bilateral arytenoid lateralisation.

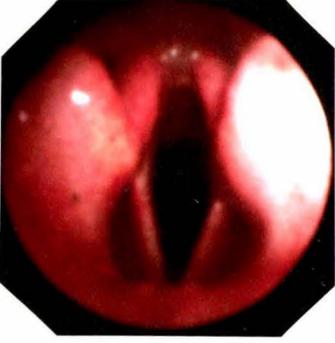


**Figure 45. DOG 8**

A) Appearance prior to neurectomy.

B) Appearance after bilateral recurrent laryngeal nerve resection.

C) Appearance six weeks after bilateral arytenoid lateralisation.



recovered from anaesthesia to consciousness. The degree of passive vocal cord flattening during expiration, which had been observed in the dogs prior to neurectomy, was increased after surgery.

c) Examination after castellated laryngofissure and vocal fold resection

Laryngoscopic examination of all dogs six weeks after castellated laryngofissure and vocal fold resection revealed that the glottis was wider than that seen in the paralysed condition, the arytenoid cartilages, nevertheless, remaining immobile. In three dogs (Nos. 1, 6 and 10), the glottal width appeared similar to that of the original, non-abducted larynx but, in two others (Nos. 2 and 9), it appeared smaller. Some irregularity of the ventral vocal fold margin was noted in three of the five dogs (Nos. 2, 6 and 9).

d) Examination After Bilateral Arytenoid Lateralisation

Laryngoscopic examination of the dogs which underwent bilateral arytenoid lateralisation revealed that in two of them (Nos. 3 and 5), the glottic opening which appeared to be of similar size to the original, normal non-abducted larynx. In the remaining three dogs (Nos. 4, 7 and 8), the glottis was considerably wider. In these animals, the corniculate process of the arytenoid cartilages were held in an abducted position.

### III<sub>4</sub> BLOOD GAS ANALYSIS

The results of blood gas analyses for each dog are recorded in Table 1. and Table 2. The difference between PaO<sub>2</sub> tension at rest and after exercise was inconsistent, with some of the dogs showing an increase in value, whilst in others it remained the same or fell. The rectal temperatures of each dog remained unchanged between rest and exercise.

a) After Neurectomy

A comparison of PaO<sub>2</sub> values between the dogs prior to and after neurectomy demonstrated a statistically significant ( $p < 0.05$ ) fall both at rest and after exercise.

b) After Castellated Laryngofissure with Focal Vold Resection

There was no statistically significant improvement in the PaO<sub>2</sub> values between neurectomised dogs and dogs after surgical treatment by castellated laryngofissure with vocal fold resection. The general trend, however, was towards increased PaO<sub>2</sub> values in the post operative samples.

TABLE 1.

RESULT OF BLOOD GAS ANALYSES FROM DOGS UNDERGOING CASTELLATED  
LARYNGOFISSURE AND VOCAL FOLD RESECTION

DOG	BEFORE SURGERY				AFTER RECURRENT LARYNGEAL NERVE SECTION (48 HOURS)				AFTER CASTELLATED LARYNGOFISSURE (SIX WEEKS)				
	Hb (g/dl)	Rest		After exercise		Rest		After exercise		Rest		After exercise	
		PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>
1	15.3	115.3	32.0	109.3	36.3	97.0	33.7	88.6	32.2	118.2	38.4	131.3	35.4
2	14.8	105.7	33.4	115.1	34.4	99.5	36.6	107.5	30.1	107.4	38.4	105.3	39.9
6	16.5	119.1	39.3	114.7	36.2	104.7	36.0	106.2	37.2	104.9	36.1	90.6	41.4
9	12.2	115.6	28.4	115.8	35.8	99.2	30.6	99.3	30.4	93.0	30.7	103.7	24.9
10	13.8	114.4	30.5	107.4	33.7	103.3	31.5	96.3	33.6	112.4	32.1	102.1	31.6

Legends

Hb - Haemoglobin  
 PaO<sub>2</sub> - Arterial oxygen tensions  
 PaCO<sub>2</sub> - Arterial carbon dioxide tensions

TABLE 2.

RESULT OF BLOOD GAS ANALYSES FROM DOGS UNDERGOING BILATERAL  
ARYTENOID LATERALISATION

DOG	BEFORE SURGERY				AFTER RECURRENT LARYNGEAL NERVE SECTION (48 HOURS)				AFTER BILATERAL ARYTENOID LATERALISATION (SIX WEEKS)				
	Hb (g/dl)	Rest		After exercise		Rest		After exercise		Rest		After exercise	
		PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>	PaO <sub>2</sub>	PaCO <sub>2</sub>
3	15.0	106.3	37.8	104.1	40.8	99.9	33.9	102.6	28.9	102.6	38.6	101.9	40.2
4	12.7	112.7	41.8	108.3	38.5	104.0	31.6	101.7	33.0	106.4	37.3	108.0	40.6
5	13.1	91.7	38.7	86.1	35.7	83.9	36.7	79.9	39.4	102.0	37.4	98.9	37.6
7	15.4	113.0	37.1	113.0	34.7	105.2	34.1	104.2	35.4	113.3	34.9	112.0	34.4
8	14.0	115.8	35.8	113.2	33.3	102.6	30.6	99.3	30.4	109.3	35.0	104.9	34.4

Legends

Hb - Haemoglobin  
 PaO<sub>2</sub> - Arterial oxygen tensions  
 PaCO<sub>2</sub> - Arterial carbon dioxide tensions

c) After Bilateral Arytenoid Lateralisation

In the group of dogs that underwent bilateral arytenoid lateralisation, there was a significant improvement ( $p < 0.05$ ) in the  $\text{PaO}_2$  values when compared to those values obtained after neurectomy. This improvement was seen both at rest and after exercise.

There was no significant differences between any of the  $\text{PaCO}_2$  values.

### III<sub>5</sub>. TIDAL BREATHING FLOW LOOP ANALYSIS

Representative tidal breathing flow-loops were obtained from all ten dogs before and after resection of the recurrent laryngeal nerves and after the surgical procedures of either castellated laryngofissure or bilateral arytenoid lateralisation. Mean values and loop ratio figures are shown in Tables three to six. The loop shapes are demonstrated in Figures 46-55. Flow turbulence produced electrical disturbances which resulted in "noise" or irregular undulations in the loop tracing. In the figures, these irregularities have been removed and replaced by smooth lines for the simplicity of display.

a) Differences in Tidal Breathing Flow-volume Loops Before and After Neurectomy

The main difference between the loop shape obtained before and after recurrent laryngeal nerve resection was the reduced amplitude of the inspiratory trace seen in the latter state. This resulted in two statistically significant ( $p < 0.05$ ) differences between the two loop shapes. Firstly, the peak expiratory flow/peak inspiratory flow ratio (PEF/PIF) was significantly increased in the neurectomised dogs as a consequence of the reduced peak inspiratory flow value. Secondly the midtidal inspiratory flow/respiratory flow at 25% of the tidal volume remaining to be exhaled ( $\text{IF}_{50}/\text{IF}_{25}$ ) was significantly reduced indicating some flattening of the inspiratory arc. The neurectomised dogs also exhibited a statistically significant ( $p < 0.05$ ) reduced expiratory/inspiratory time ratio (TE/TI) as a result of a prolonged inspiratory duration.

Surprisingly, although the peak inspiratory flow value was reduced in the neurectomised dogs, the ratios of (a) peak inspiratory flow/inspiratory flow at 25% of the tidal volume remaining to be exhaled ( $\text{PIF}/\text{IF}_{25}$ ) and (b) peak inspiratory flow/midtidal inspiratory flow ( $\text{PIF}/\text{IF}_{50}$ ) were not significantly different from the ratios obtained in the control dogs. This was due to a relatively high peak inspiratory flow value, in most loops obtained from the neurectomised dogs when compared to the remainder of the inspiratory traces.

After recurrent laryngeal nerve resection, most of the loops obtained from the dogs were a Type 2 shape indicating a variable extrathoracic airway obstruction. Loops from dogs Nos. 3 and 6, however, had a reduction in both expiratory and inspiratory tracings (Type 3 loop) implying the presence of a fixed obstructive lesion.

b) Tidal Breathing Flow-volume Loops After Castellated Laryngofissure

A comparison of tidal breathing flow-volume loop ratios in dogs before and after neurectomy and castellated laryngofissure revealed no statistically significant differences. The loop shape, however, suggested marginally increased inspiratory air flow in dogs Nos. 1, 6 and 9.

c) Tidal Breathing Flow-volume Loops After Bilateral Arytenoid Lateralisation

A statistically significant difference ( $p < 0.05$ ) was revealed in a comparison of loop ratios between those obtained from the neurectomised dogs and dogs treated by bilateral arytenoid lateralisation. This difference was a significant reduction of the expiratory/inspiratory flow ratios at the peak, midtidal and 25% of the tidal volume remaining to be exhaled, volume values (ie PEF/PIF,  $EF_{50}$  and  $EF_{25}/IF_{25}$ ) in the loops of the bilateral arytenoid lateralisation treated dogs. The cause of this ratio reduction was the increased amplitude of the inspiratory flow tracing from all the dogs in this group. The loop shape, however, demonstrated a loss of the characteristic expiratory curve indicating that the expiratory braking capability of the larynx had been lost.

TABLE 3.

MEAN VALUES FOR TIDAL BREATHING FLOW-VOLUME LOOPS FROM DOGS  
AFTER CASTELLATED LARYNGOFISSURE AND VOCAL FOLD RESECTION

Dog	State	VT (L)	PEF (L/S)	PIF (L/S)	EF <sub>50</sub> (L/S)	IF <sub>50</sub> (L/S)	EF <sub>25</sub> (L/S)	IF <sub>25</sub> (L/S)	RR /min	TE (S)	T1 (S)
1	N	0.8	1.7	1.2	1.0	1.1	1.2	0.9	35	0.9	0.8
	RLN	0.6	1.1	0.6	0.8	0.4	0.6	0.4	33	0.9	0.9
	CLF	0.8	1.9	1.3	1.3	0.9	0.8	0.8	35	0.9	0.8
2	N	1.4	2.8	3.1	2.1	3.0	1.7	2.4	30	1.3	1.3
	RLN	0.9	1.9	1.0	1.1	1.1	0.5	0.9	26	1.3	1.2
	CLF	1.0	2.1	1.2	1.8	1.0	1.6	0.8	28	1.2	1.0
6	N	1.0	1.5	1.3	1.3	1.1	0.9	1.1	28	1.2	1.0
	RLN	1.0	0.8	0.7	0.7	0.5	0.6	0.4	26	1.5	1.0
	CLF	0.7	1.1	0.9	0.8	0.8	0.6	0.6	25	1.6	1.0
9	N	1.1	1.9	2.1	1.7	2.0	1.2	1.7	27	1.0	0.8
	RLN	0.6	0.9	0.7	0.8	0.5	0.4	0.4	30	1.2	1.0
	CLF	1.0	1.3	1.3	0.9	1.0	0.4	0.6	32	1.6	1.0
10	N	1.1	1.3	0.9	1.0	0.8	1.0	0.7	24	1.0	0.8
	RLN	0.8	1.6	1.1	1.0	1.1	0.7	0.9	25	1.3	1.0
	CLF	0.8	1.0	0.9	0.7	0.8	0.7	0.6	26	1.3	1.1

Legends

N = dogs prior to neurectomy  
 RLN = dogs after recurrent laryngeal neurectomy  
 CLF = dogs after castellated laryngofissure  
 VT = tidal volume  
 PEF = peak expiratory air flow rate  
 PIF = peak inspiratory air flow rate  
 EF50 = midtidal expiratory air flow rate  
 IF50 = midtidal inspiratory air flow rate

L = litres  
 S = seconds  
 min = minutes  
 EF25 = expiratory air flow rate at end of expiratory volume plus 25% VT  
 IF25 = inspiratory air flow rate at end of inspiratory volume plus 25% VT  
 RR = respiratory rate  
 TE = expiratory time  
 T1 = inspiratory rate

TABLE 4:

INDICES OF TIDAL BREATHING FLOW-VOLUME LOOPS FROM DOGS  
AFTER CASTELLATED LARYNGOFISSURE AND VOCAL FOLD RESECTION

Dog	State	VT (L)	PEF PIF	EF <sub>50</sub> IF <sub>50</sub>	EF <sub>25</sub> IF <sub>25</sub>	PEF EF <sub>50</sub>	PEF EF <sub>25</sub>	PEF EF <sub>25</sub>	PIF IF <sub>50</sub>	PIF IF <sub>25</sub>	IF <sub>50</sub> IF <sub>25</sub>	RR /MIN	TE T1
1	N	0.8	1.4	0.9	1.3	1.7	1.4	0.8	1.0	1.3	1.2	30	1.1
	RLN	0.6	1.8	1.3	2.0	1.4	1.8	1.8	1.5	1.5	1.0	33	1.0
	CLF	0.8	1.5	1.4	1.0	1.5	2.4	1.6	1.4	1.6	1.1	30	1.1
2	N	1.4	0.9	0.7	0.7	1.3	1.6	1.2	1.0	1.3	1.3	24	1.4
	RLN	0.9	1.9	1.0	0.5	1.7	3.8	2.2	0.9	1.1	1.2	30	1.0
	CLF	0.9	1.7	1.2	0.8	1.2	1.3	1.1	0.8	0.6	0.8	26	1.0
6	N	1.0	1.1	1.2	0.8	1.2	1.6	1.4	1.2	1.2	1.0	28	1.2
	RLN	1.0	1.0	1.4	1.9	1.1	1.1	1.1	1.4	2.1	1.5	26	1.5
	CLF	0.7	1.2	1.0	1.0	1.3	1.8	1.3	1.1	1.5	1.3	25	1.6
9	N	1.1	0.9	0.8	0.6	1.1	1.6	1.4	1.0	1.2	1.2	27	1.3
	RLN	0.6	1.1	1.1	1.0	1.1	2.2	2.0	1.1	2.0	1.7	30	1.2
	CLF	1.0	0.9	0.9	0.6	1.2	2.7	2.2	1.0	2.0	1.6	32	1.6
10	N	1.1	1.3	1.3	1.4	1.3	1.3	1.0	1.1	1.3	1.1	24	1.3
	RLN	0.8	0.9	0.9	0.8	1.6	2.2	1.4	1.2	1.2	1.2	25	1.3
	CLF	0.8	0.9	0.9	1.1	1.4	1.1	1.0	1.3	1.5	1.3	26	1.2

Legends

N = dogs prior to neurectomy  
 RLN = dogs after recurrent laryngeal neurectomy  
 CLF = dogs after castellated laryngofissure  
 VT = tidal volume  
 PEF = peak expiratory air flow rate  
 PIF = peak inspiratory air flow rate  
 EF50 = midtidal expiratory air flow rate  
 IF50 = midtidal inspiratory air flow rate

L = litres  
 S = seconds  
 min = minutes  
 EF25 = expiratory air flow rate at end of expiratory volume plus 25% VT  
 IF25 = inspiratory air flow rate at end of inspiratory volume plus 25% VT  
 RR = respiratory rate  
 TE = expiratory time  
 T1 = inspiratory rate

TABLE 5.

MEAN VALUES FOR TIDAL BREATHING FLOW-VOLUME LOOPS FROM DOGS  
AFTER BILATERAL ARYTENOID LATERALISATION

Dog	State	VT (L)	PEF (L/S)	PIF (L/S)	EF <sub>50</sub> (L/S)	IF <sub>50</sub> (L/S)	EF <sub>25</sub> (L/S)	IF <sub>25</sub> (L/S)	RR /min	TE (S)	T1 (S)
3	N	1.2	2.0	1.7	0.8	1.0	0.7	1.0	20	2.0	1.0
	RLN	1.0	1.3	0.7	0.9	0.7	0.7	0.6	25	1.5	1.3
	BAL	0.7	1.2	1.6	1.0	1.2	0.8	1.2	24	1.8	1.0
4	N	1.3	1.8	1.4	1.3	1.4	0.7	0.7	28	1.4	1.2
	RLN	0.9	1.4	0.6	1.0	0.5	0.4	0.4	25	1.6	1.8
	BAL	0.7	1.6	1.4	1.0	1.2	0.6	1.0	27	1.5	1.4
5	N	1.0	0.9	1.2	0.7	1.0	0.3	0.7	16	2.1	1.7
	RLN	0.7	0.8	0.6	0.7	0.5	0.6	0.5	22	1.5	1.4
	BAL	1.0	1.4	1.6	0.8	1.2	0.6	1.0	26	1.5	1.1
7	N	0.9	1.8	1.4	1.0	1.3	0.5	1.0	24	1.4	0.9
	RLN	0.5	1.0	0.6	0.8	0.5	0.5	0.4	29	1.2	1.1
	BAL	0.9	1.4	1.2	0.8	1.0	0.7	0.9	28	1.2	1.1
8	N	0.8	1.0	1.2	0.7	1.0	0.5	0.6	24	1.5	1.0
	RLN	0.5	1.2	0.7	0.5	0.5	0.6	0.4	26	1.5	1.1
	BAL	1.1	1.4	1.4	1.0	1.3	0.9	1.0	28	1.3	1.2

Legends

N = dogs prior to neurectomy  
 RLN = dogs after recurrent laryngeal neurectomy  
 BAL = dogs after bilateral arytenoid lateralisation  
 VT = tidal volume  
 PEF = peak expiratory air flow rate  
 PIF = peak inspiratory air flow rate  
 EF50 = midthal expiratory air flow rate  
 IF50 = midthal inspiratory air flow rate

L = litres  
 S = seconds  
 min = minutes  
 EF25 = expiratory air flow rate at end of expiratory volume plus 25% VT  
 IF25 = inspiratory air flow rate at end of inspiratory volume plus 25% VT  
 RR = respiratory rate  
 TE = expiratory time  
 T1 = inspiratory rate

TABLE 6:

INDICES OF TIDAL BREATHING FLOW-VOLUME LOOP FROM DOGS  
AFTER BILATERAL ARYTENOID LATERALISATION

Dog	State	VT (L)	PEF PIF	EF <sub>50</sub> IF <sub>50</sub>	EF <sub>25</sub> IF <sub>25</sub>	PEF EF <sub>50</sub>	PEF EF <sub>25</sub>	EF <sub>50</sub> EF <sub>25</sub>	PIF IF <sub>50</sub>	PIF IF <sub>25</sub>	IF <sub>50</sub> IF <sub>25</sub>	RR /MIN	TE TI
3	N	1.2	1.2	0.8	0.7	2.5	2.8	1.1	1.7	1.7	1.0	20	2.0
	RLN	1.0	2.1	1.3	1.1	1.4	1.9	1.3	1.0	1.2	1.1	25	1.2
	BAL	0.7	0.8	0.8	0.6	1.2	1.5	1.3	1.3	1.3	1.0	24	1.8
4	N	1.3	1.3	0.9	1.1	1.4	2.6	1.0	1.0	2.0	1.3	28	1.2
	RLN	0.9	2.3	2.0	1.0	1.3	3.4	2.5	1.2	1.5	1.3	25	0.8
	BAL	0.7	1.1	0.8	0.6	1.6	2.6	1.7	1.2	1.4	1.2	27	1.0
5	N	1.0	0.8	0.7	0.4	1.3	3.0	2.3	1.2	1.7	1.3	16	1.2
	RLN	0.7	1.3	1.4	1.2	1.1	1.3	1.2	1.2	1.2	1.0	22	1.0
	BAL	1.0	0.9	0.7	0.6	1.8	2.3	1.3	1.3	1.6	1.2	26	1.3
7	N	0.9	1.3	0.8	0.5	1.8	3.6	2.0	1.0	1.4	1.3	24	1.6
	RLN	0.5	1.7	1.6	1.3	1.2	2.0	1.5	1.2	1.5	1.3	29	1.0
	BAL	0.9	1.2	0.8	0.7	1.8	2.0	1.1	1.2	1.3	1.1	28	1.0
8	N	0.8	0.8	0.7	1.3	1.3	2.0	1.4	1.2	2.0	1.5	24	1.5
	RLN	0.5	1.7	1.0	1.5	2.4	2.0	0.8	1.4	1.7	1.3	26	1.4
	BAL	1.1	1.0	0.8	0.9	1.4	1.6	1.1	1.0	1.4	1.3	28	1.0

Legends

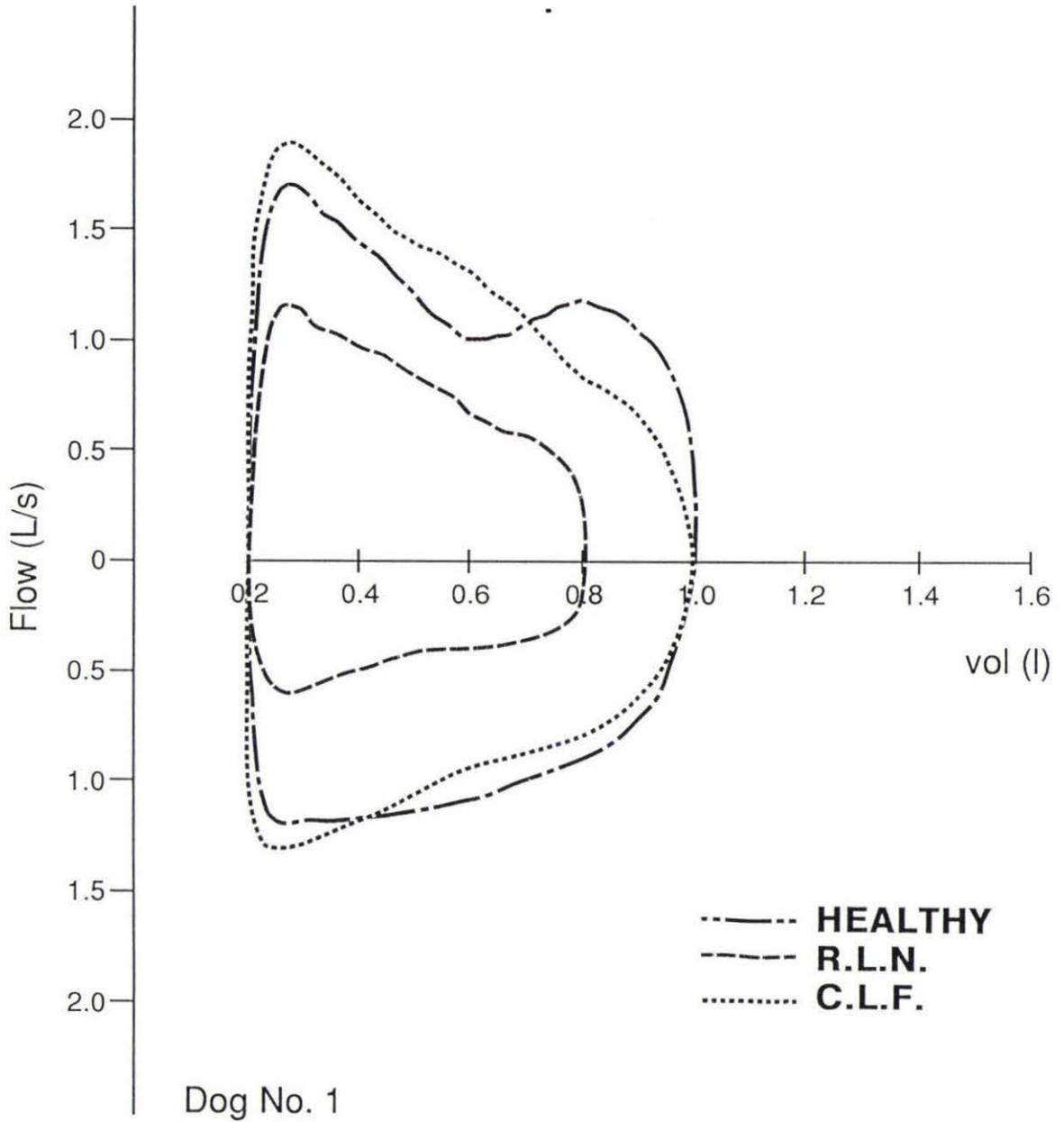
N = dogs prior to neurectomy  
 RLN = dogs after recurrent laryngeal neurectomy  
 BAL = dogs after bilateral arytenuoid lateralisation  
 VT = tidal volume  
 PEF = peak expiratory air flow rate  
 PIF = peak inspiratory air flow rate  
 EF50 = midtidal expiratory air flow rate  
 IF50 = midtidal inspiratory air flow rate

L = litres  
 S = seconds  
 min = minutes  
 EF25 = expiratory air flow rate at end of expiratory volume plus 25% VT  
 IF25 = inspiratory air flow rate at end of inspiratory volume plus 25% VT  
 RR = respiratory rate  
 TE = expiratory time  
 TI = inspiratory time

**Tidal breathing  
flow-volume loops  
of the  
ten experimental dogs.**

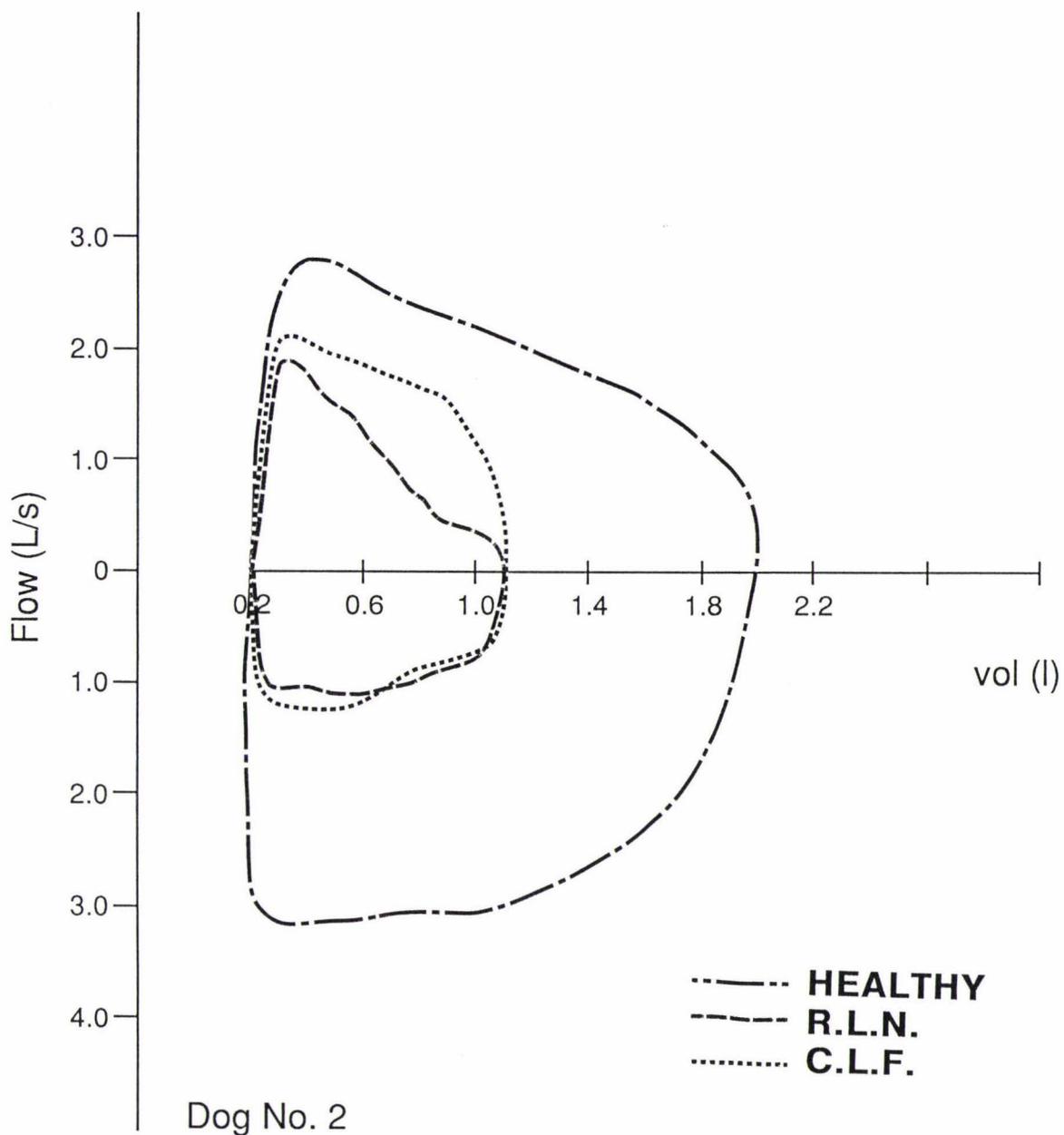
## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
C.L.F.	=	DOG AFTER CASTELLATED LARYNGOFISSURE
L or l	=	LITRES
s	=	SECONDS



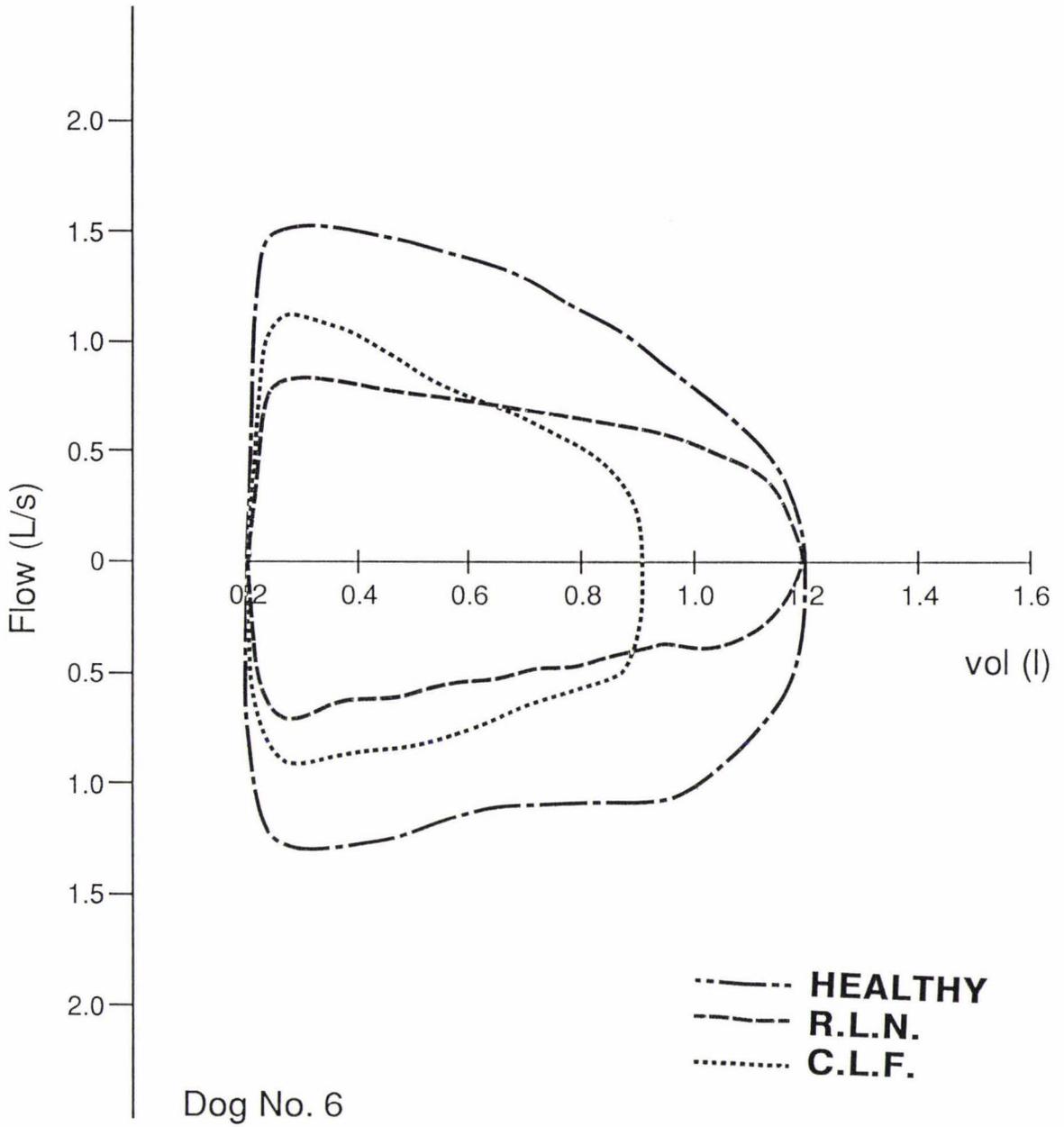
## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
C.L.F.	=	DOG AFTER CASTELLATED LARYNGOFISSURE
L or l	=	LITRES
s	=	SECONDS



LEGEND

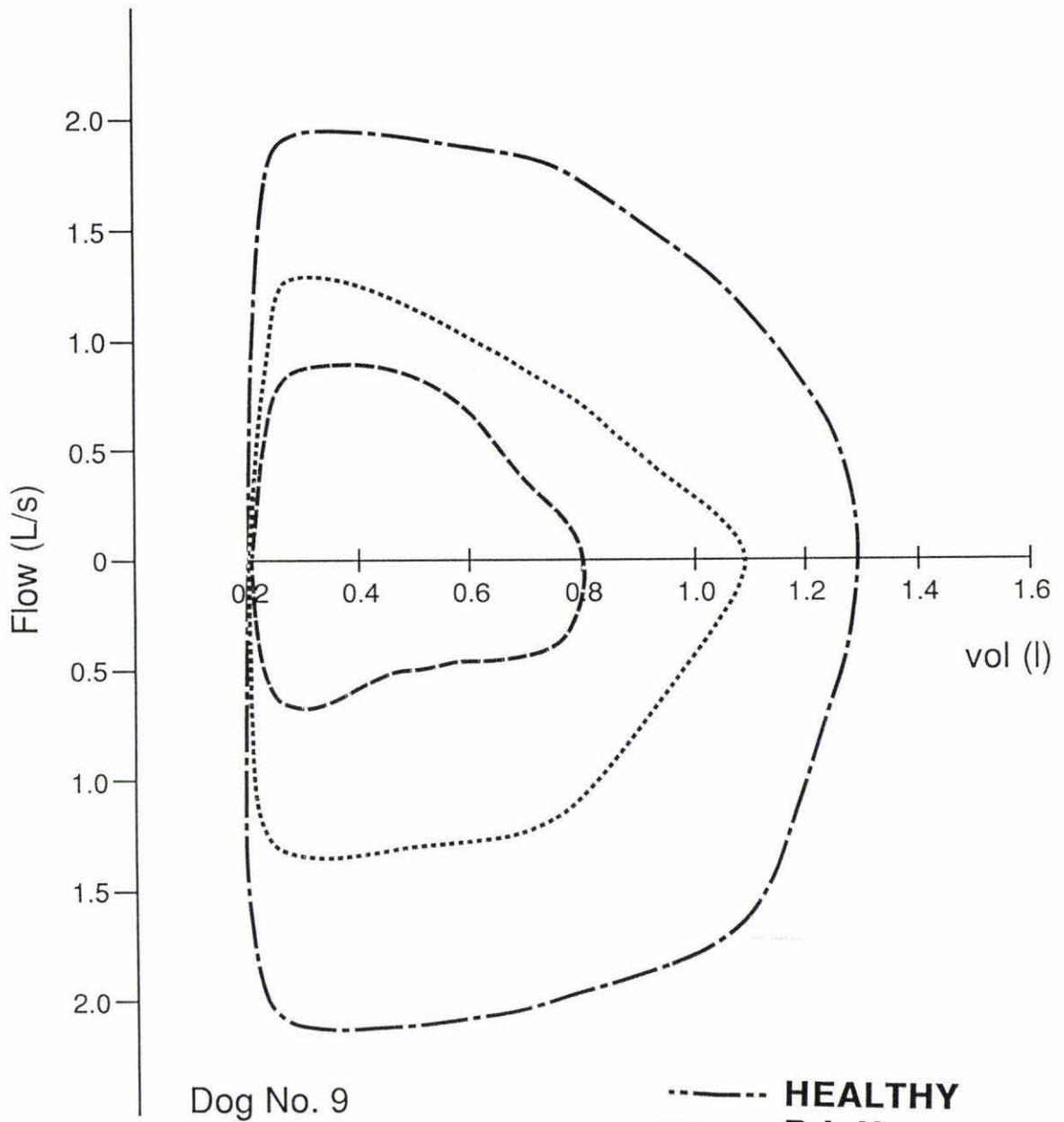
HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
C.L.F.	=	DOG AFTER CASTELLATED LARYNGOFISSURE
L or l	=	LITRES
s	=	SECONDS



Dog No. 6

## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
C.L.F.	=	DOG AFTER CASTELLATED LARYNGOFISSURE
L or l	=	LITRES
s	=	SECONDS



## LEGEND

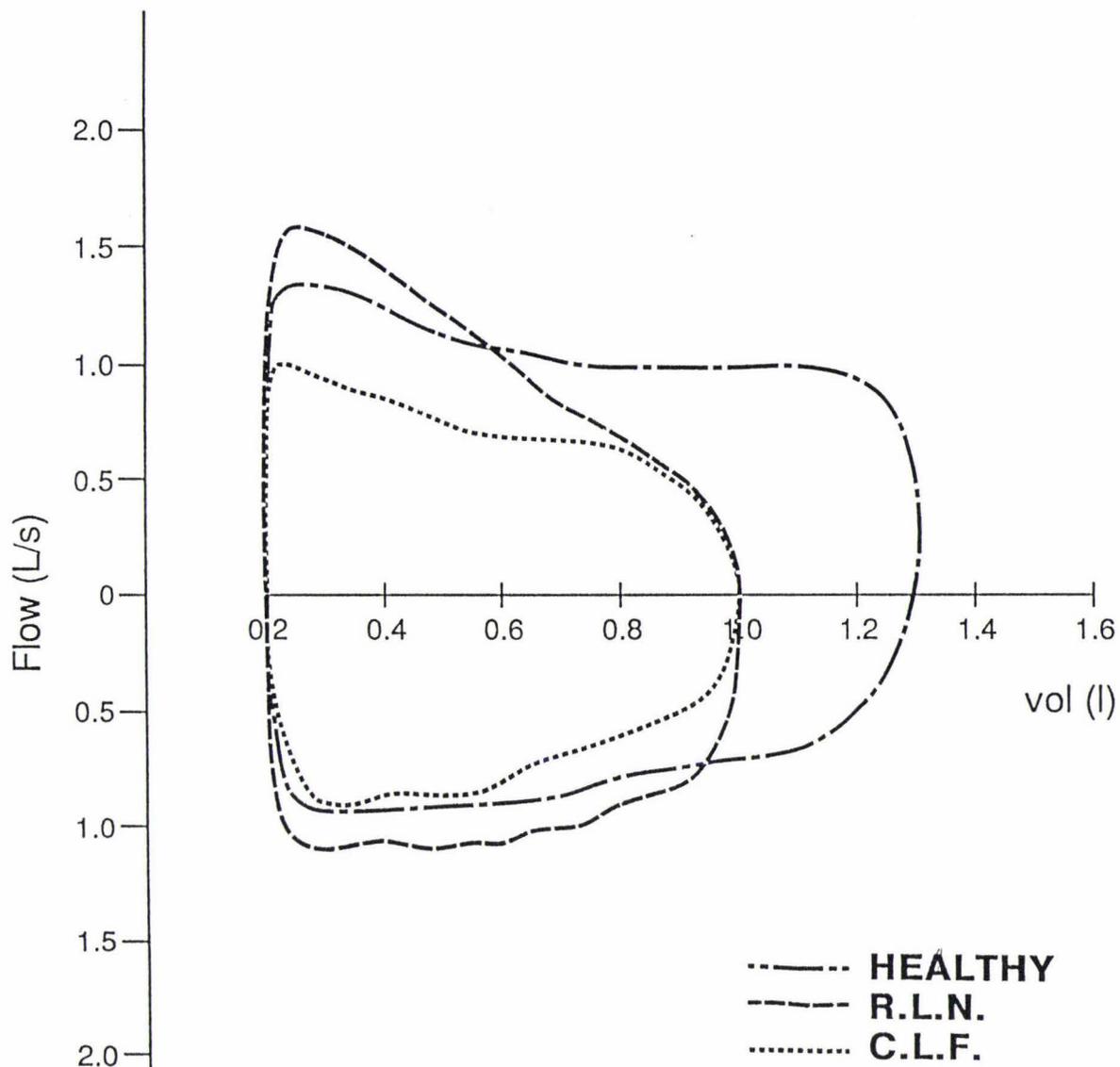
HEALTHY = DOG PRIOR TO NEURECTOMY

R.L.N. = DOG AFTER RECURRENT LARYNGEAL  
NEURECTOMY

C.L.F. = DOG AFTER CASTELLATED  
LARYNGOFISSURE

L or l = LITRES

s = SECONDS



Dog No. 10

## LEGEND

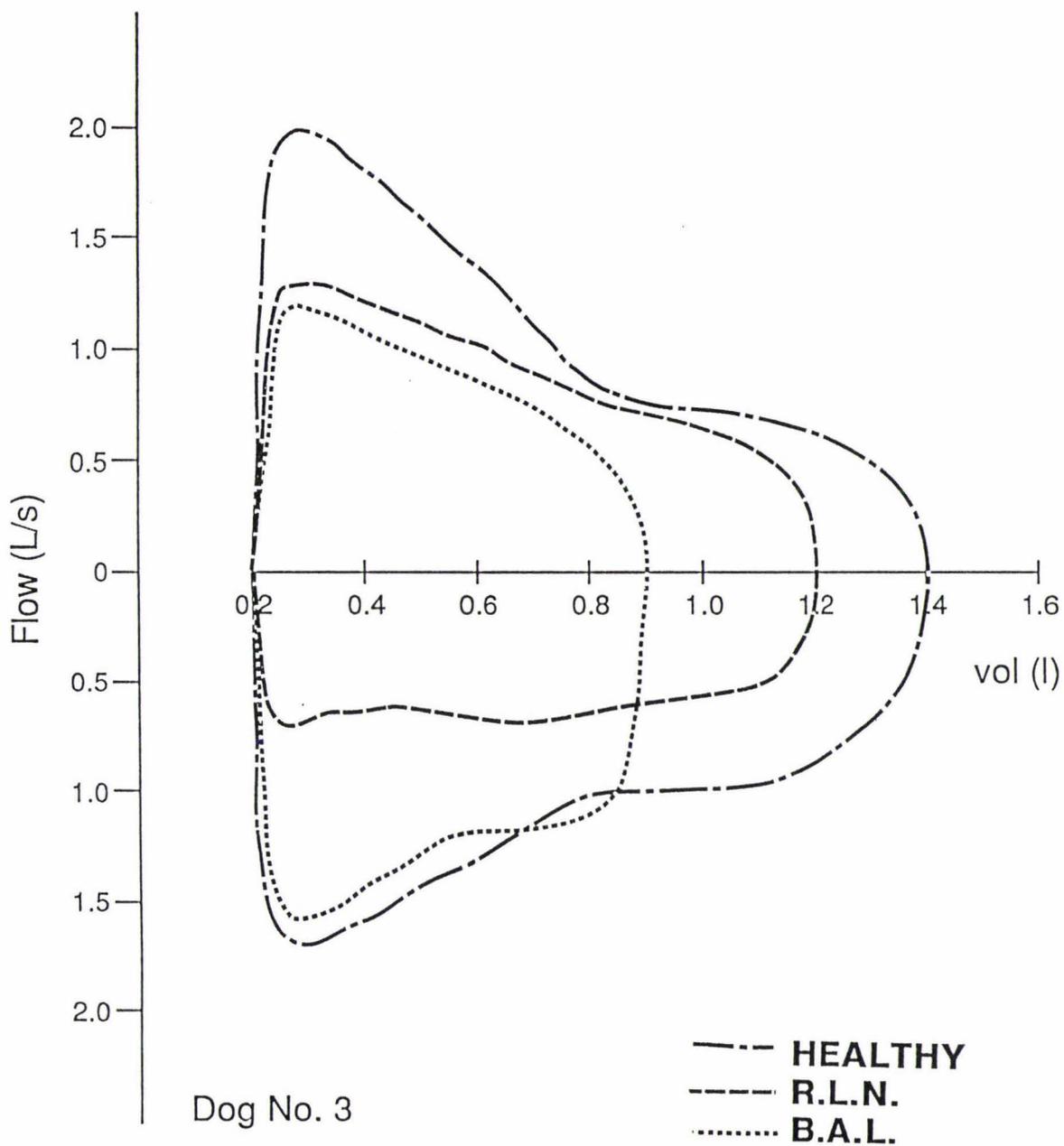
HEALTHY = DOG PRIOR TO NEURECTOMY

R.L.N. = DOG AFTER RECURRENT LARYNGEAL  
NEURECTOMY

B.A.L. = DOG AFTER BILATERAL ARYTENOID  
LATERALISATION

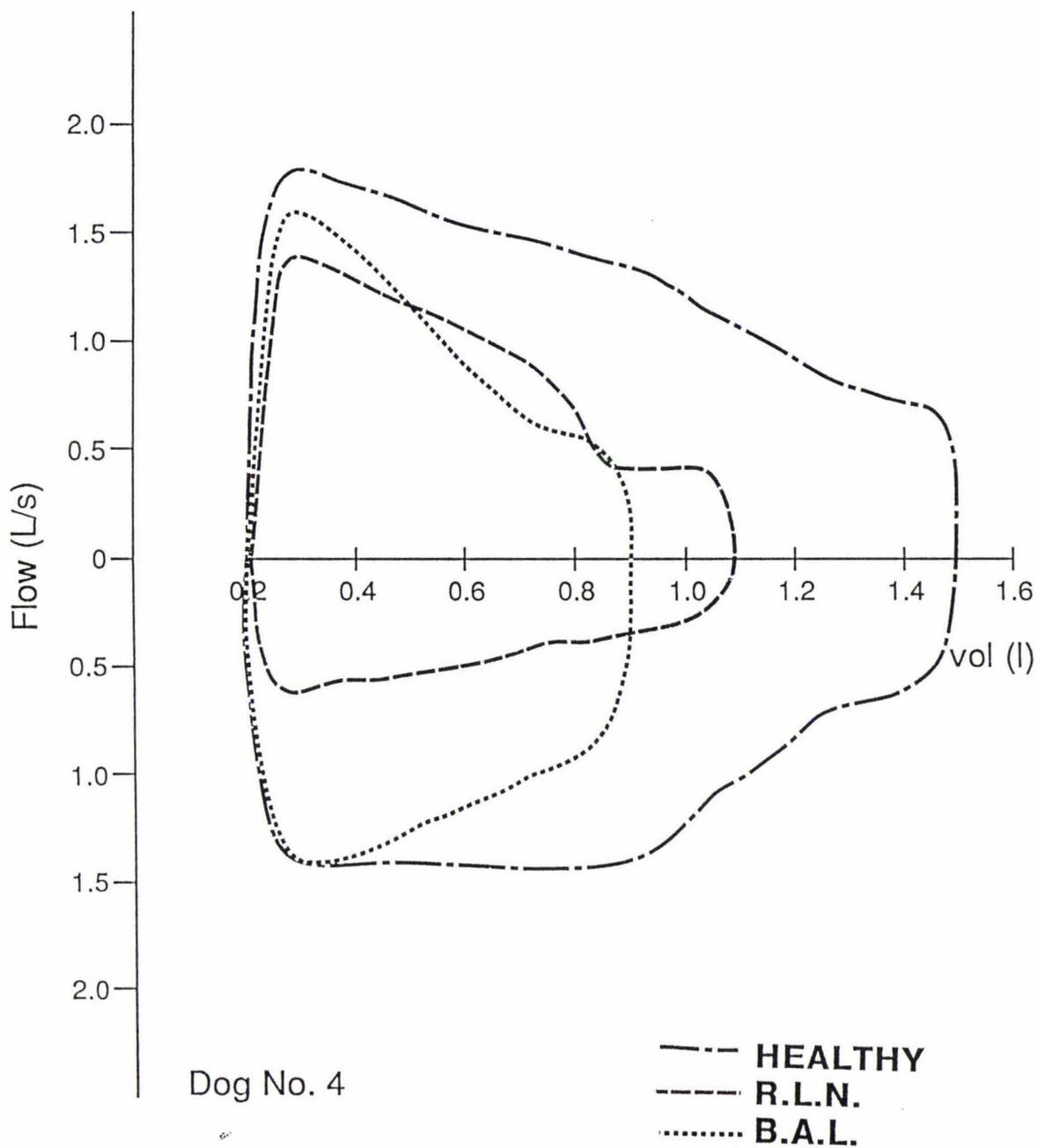
L or l = LITRES

s = SECONDS



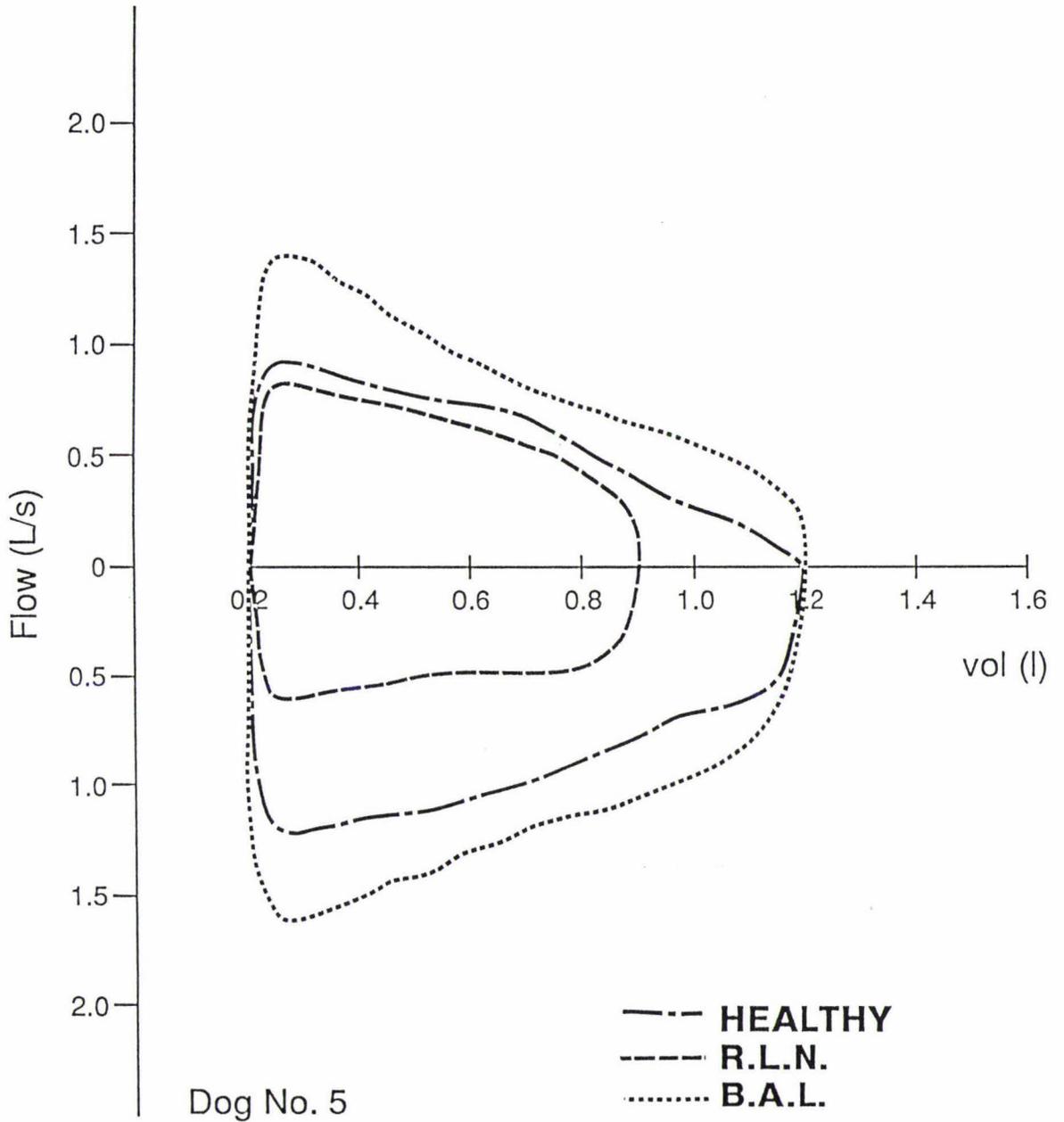
## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
B.A.L.	=	DOG AFTER BILATERAL ARYTENOID LATERALISATION
L or l	=	LITRES
s	=	SECONDS



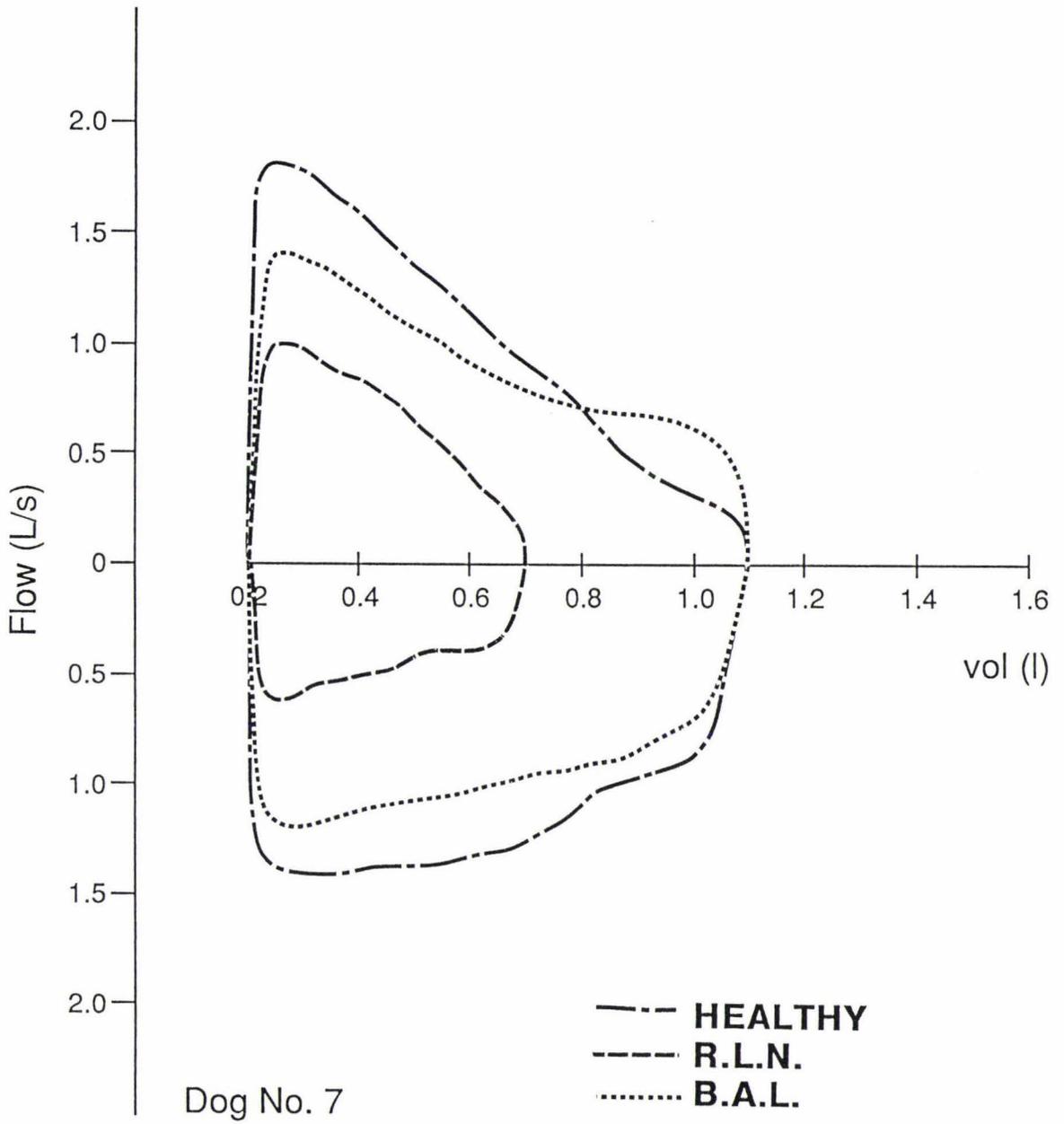
### LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
B.A.L.	=	DOG AFTER BILATERAL ARYTENOID LATERALISATION
L or l	=	LITRES
s	=	SECONDS



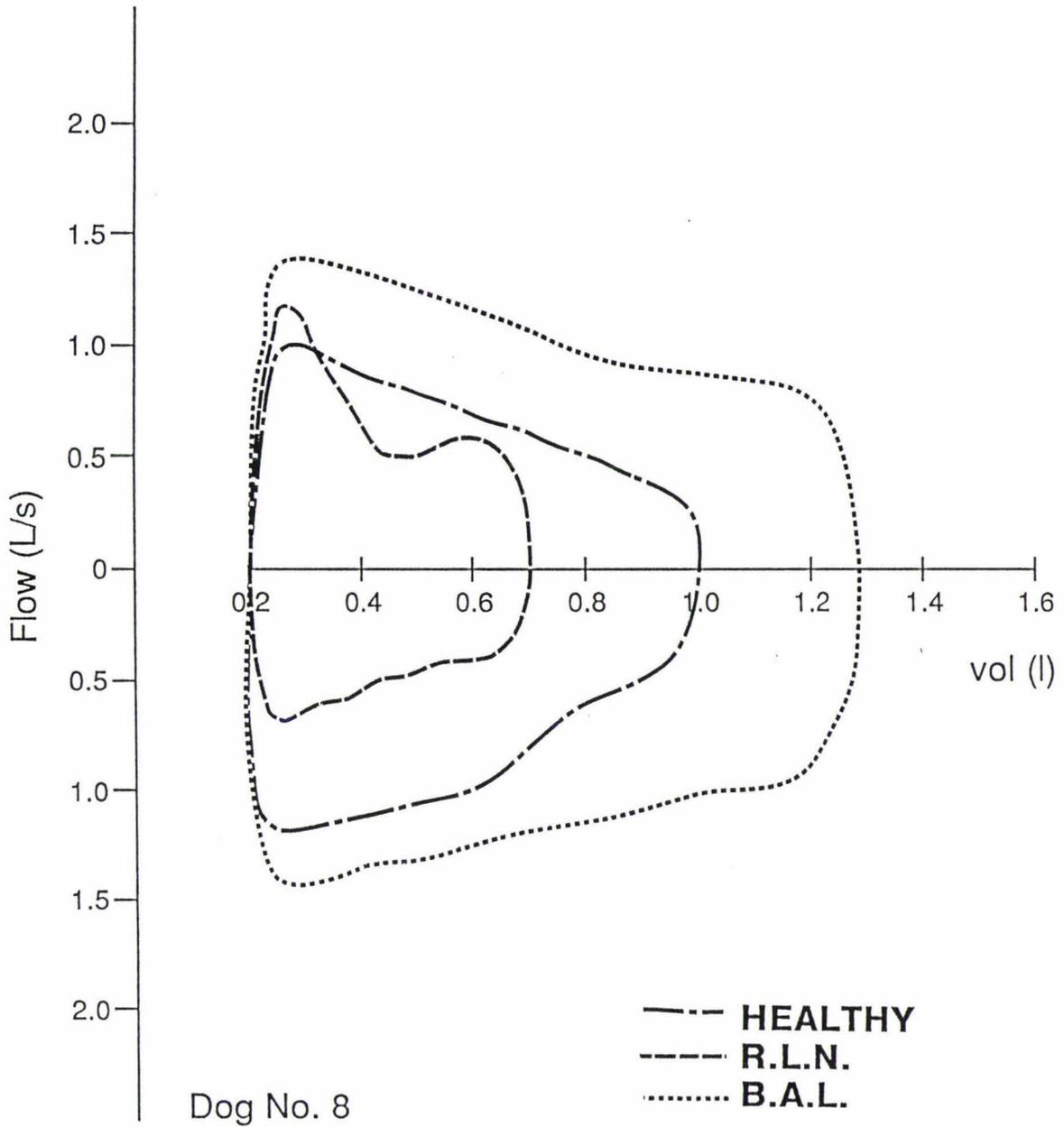
## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
B.A.L.	=	DOG AFTER BILATERAL ARYTENOID LATERALISATION
L or l	=	LITRES
s	=	SECONDS



## LEGEND

HEALTHY	=	DOG PRIOR TO NEURECTOMY
R.L.N.	=	DOG AFTER RECURRENT LARYNGEAL NEURECTOMY
B.A.L.	=	DOG AFTER BILATERAL ARYTENOID LATERALISATION
L or l	=	LITRES
s	=	SECONDS



### III<sub>6</sub>. AUTOPSY EXAMINATION

In each dog, the site of recurrent laryngeal nerve resection was explored to ensure that the nerves had been severed. This was verified visually in each dog. Gross examination of the distal trachea, bronchi and lungs in all dogs revealed no significant abnormalities.

Inspection of the larynges demonstrated a change common to both groups of dogs. This was that the *rima glottidis* appeared smaller than the opening observed under laryngoscopic examination immediately prior to death (compare Figure 45 with Figure 58).

#### a) Laryngeal changes after castellated laryngofissure

In the dogs which underwent castellated laryngofissure, the following salient features were noted:

- i) when the *rima glottidis* was viewed rostro-caudally, it appeared to be wider ventrally, with the arytenoids positioned medially. In all cases, the mid ventral commissure was displaced to the left side, resulting in asymmetry (Figure 56).
- ii) the castellated incision had healed by fibrosis.
- iii) no web of granulation tissue was present within the laryngeal lumen at the site of the vocal fold resection.

The only other abnormality found in this group of dogs was at the site of the tracheostomy. Here the severed tracheal rings, although united by fibrous tissue, were markedly deformed when compared to the normal tracheal ring above and below this site (Figure 57).

#### b) Laryngeal changes after bilateral arytenoid lateralisation

In the group of dogs which underwent bilateral arytenoid lateralisation, examination of the larynges revealed:

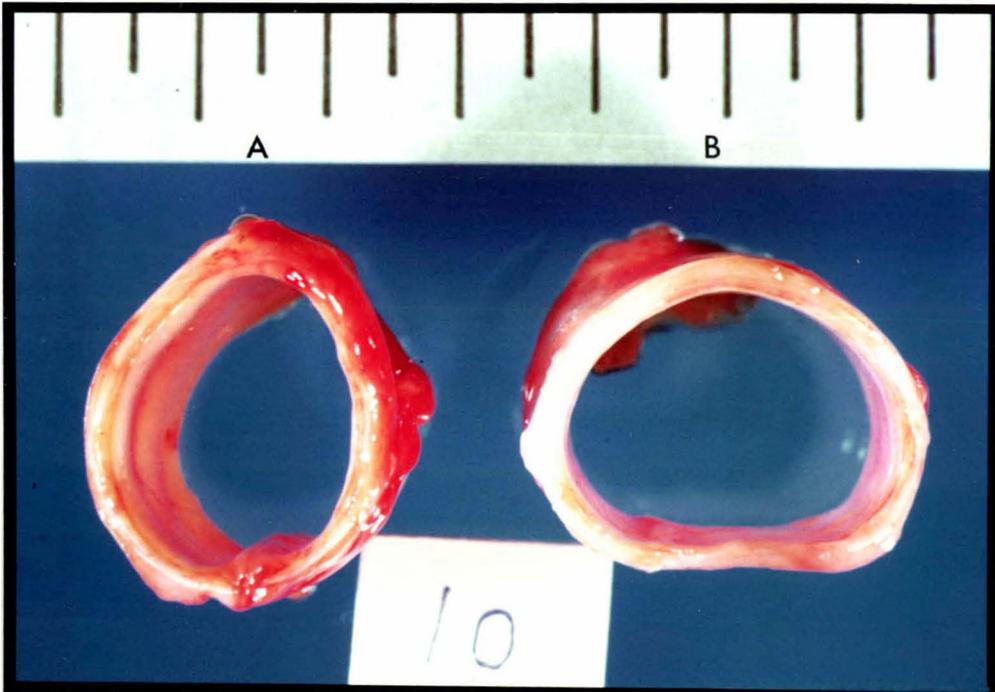
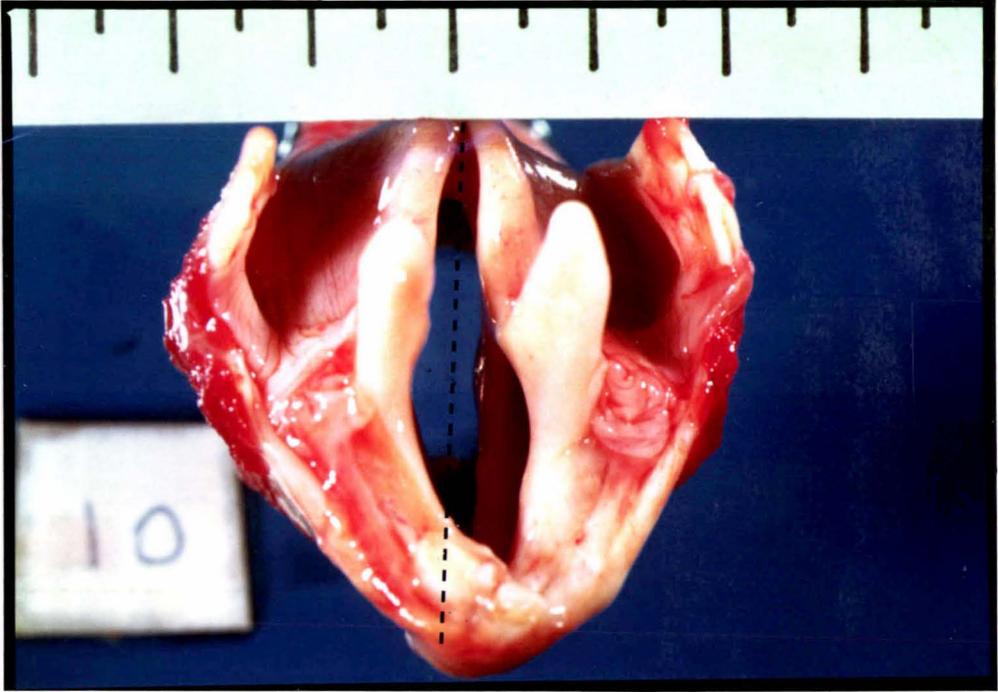
- i) The *rima glottidis* in dogs No. 4, 7 and 8 was symmetrical with the arytenoids being similarly positioned (see Figure 58). However, in dogs 3. and 5. this was not observed. In dog No. 3, the left arytenoid was less abducted than the right, and in dog No. 5 the right arytenoid was less abducted than the left (see Figure 60).

It was noted that, in these two dogs, the lateralisation suture had been placed in a more rostral position in relation to the caudal border of the thyroid cartilage (Figure 61) compared to the others (Figure 59).

- ii) The muscular process of the arytenoid cartilage had united with the thyroid lamina by fibrous tissue at the site of the lateralisation suture.
- iii) There was no visual evidence of penetration of the laryngeal mucosa in dogs No. 5 and 7.
- iv) The lateralisation sutures had remained secure and unbroken in every case.

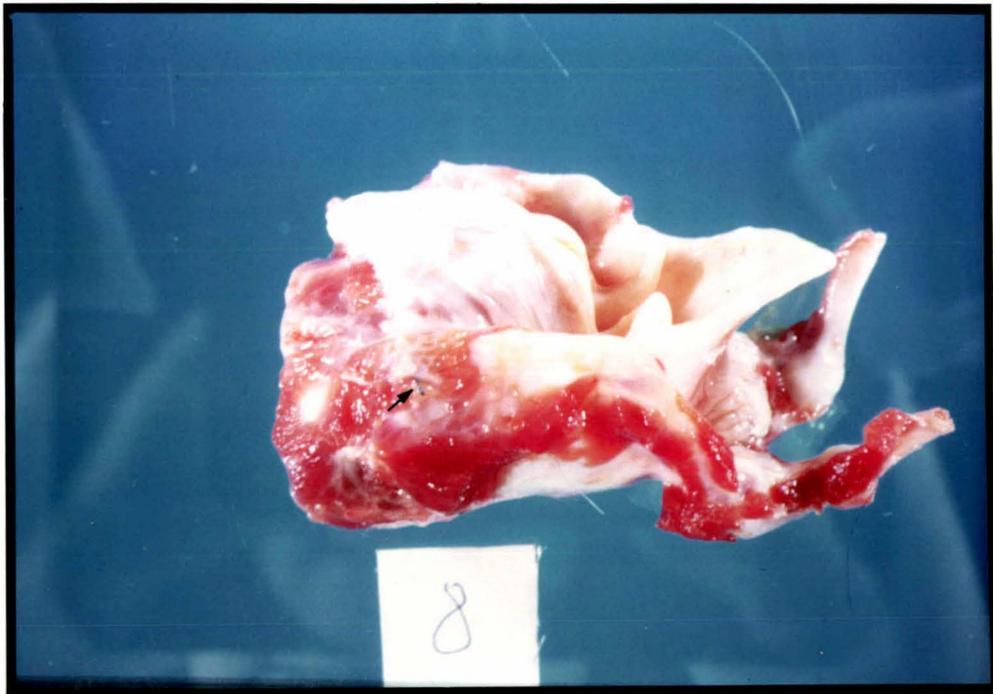
**Figure 56** Photograph of larynx from cadaver after castellated laryngofissure demonstrating asymmetry of the *rima glottidis*. The ventral portion of the laryngeal opening is deviated to the left.

**Figure 57** Photographs of two trachea rings illustrating the effects of tracheostomy on the tracheal shape. A) the site of the tracheostomy shows narrowing and change in shape when compared to the normal tracheal lumen, B) immediately distal to the tracheostomy site.



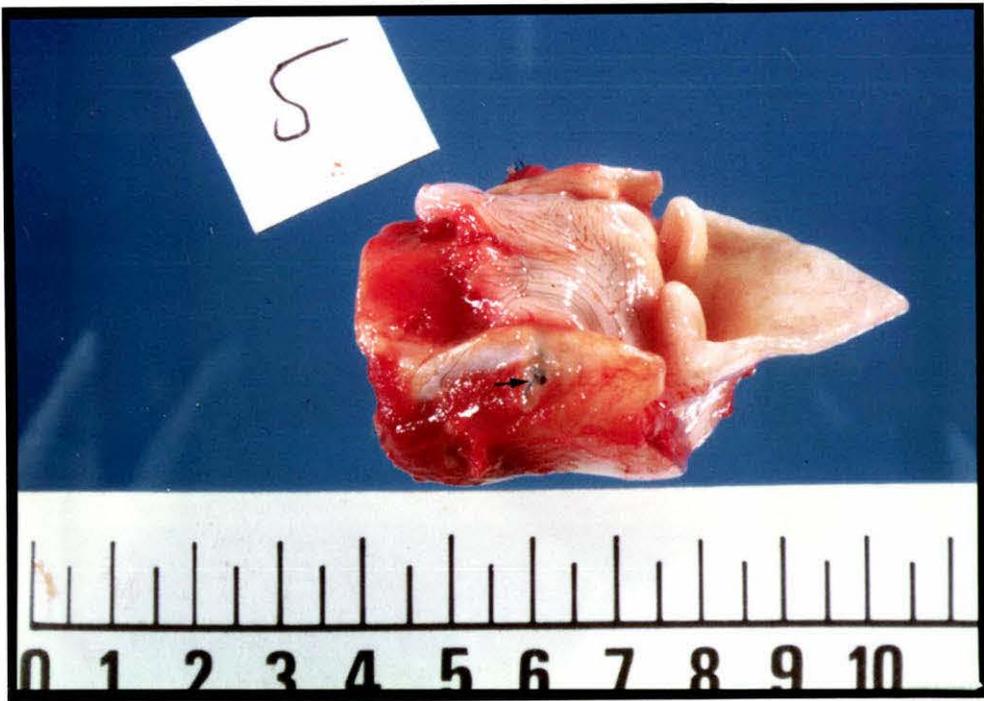
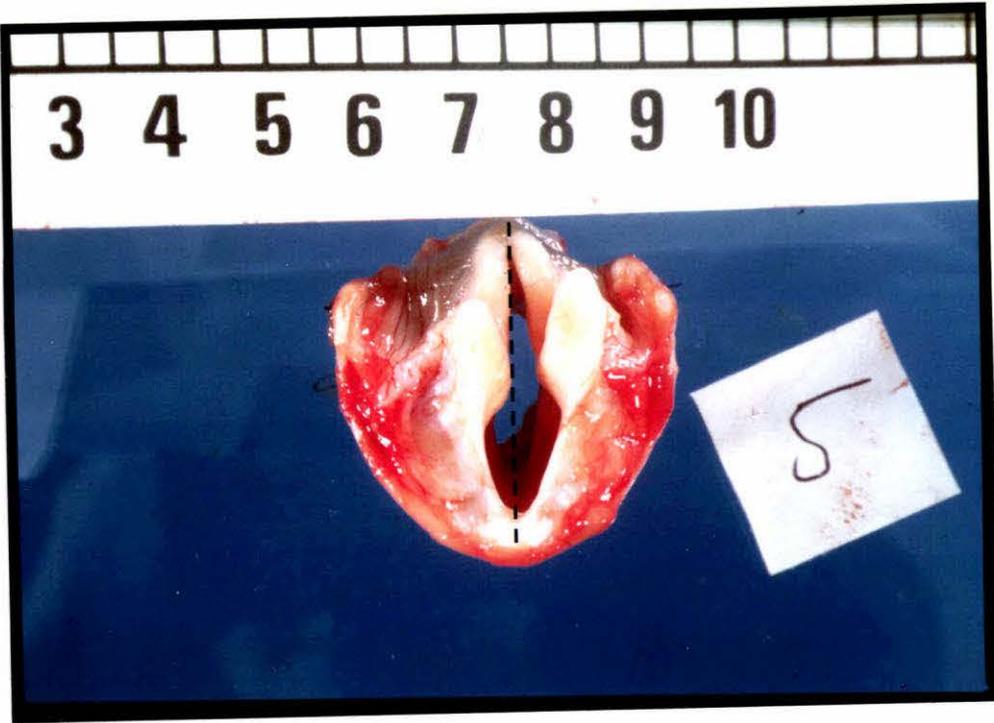
**Figure 58** Photographs of oral view of larynx from cadaver after bilater arytenoid lateralisation showing symmetrically positioned arytenoid cartilages.

**Figure 59** Photograph of lateral view from cadaver after bilateral arytenoid lateralisation showing caudal position of lateralisation suture (arrow).



**Figure 60** Photograph of larynx from cadaver after bilateral arytenoid lateralisation showing asymmetry of the *rima glottidis*.

**Figure 61** Photograph of larynx from cadaver after bilateral arytenoid lateralisation showing the more rostral position of lateralisation suture (arrow).



## CHAPTER IV.

## DISCUSSION

The results of this investigation are discussed in five sections. These are:

- i) the effects of bilateral recurrent laryngeal nerve resection.
- ii) the efficacy of castellated laryngofissure and vocal fold resection in alleviating the effects of experimentally induced laryngeal paralysis.
- iii) the efficacy of bilateral arytenoid lateralisation in alleviating the effects of experimentally induced laryngeal paralysis.
- iv) the evaluation of the diagnostic techniques used.
- v) critical evaluation of the experiment.

#### IV<sub>1</sub> THE EFFECTS OF BILATERAL RECURRENT LARYNGEAL NERVE RESECTION

Bilateral recurrent laryngeal nerve resection produced a number of clinically obvious effects in the experimental dogs. These included coughing, a husky reduced bark and inspiratory stridor, and were similar to the clinical signs described in dogs with naturally occurring idiopathic laryngeal paralysis (O'Brien *et al*, 1973; Venker-van Haagen *et al*, 1978; Gaber *et al*, 1985; and Love *et al*, 1987). However, some clinical signs which are associated with idiopathic laryngeal paralysis such as gagging and vomiting (Gaber *et al*, 1985; LaHue 1989), were not observed in the neurectomised dogs. Thus it seems that these signs are unassociated with recurrent laryngeal nerve damage, but are, as Harvey (1983) postulated, possibly caused by a co-existing neuromuscular disease affecting the swallowing mechanism. The nerves governing swallowing are the glossopharyngeal and the pharyngeal branch of the vagus. Dogs with clinically apparent dysphagia are believed to have a polyneuropathy affecting these nerves, with either the lesion in the *nucleus ambiguus* or, specifically, within the motor neurons (Venker-van Haagen, Hartman and Wolvekamp, 1986). Since dogs with idiopathic laryngeal paralysis may also show signs of dysphagia, and the glossopharyngeal, vagus and recurrent laryngeal nerves all have their nuclei in the *nucleus ambiguus*, it seems plausible that the disease may involve a lesion within this structure.

Another similarity between the experimentally produced and the naturally occurring laryngeal paralysis is that, whilst at rest, the dogs appeared to cope well with the laryngeal dysfunction. It was only when they were vigorously exercised or stressed that clinical signs of respiratory obstruction became apparent.

In addition to the clinical effects produced by bilateral recurrent laryngeal neurectomy, endoscopic, blood gas and tidal breathing flow-volume loop changes were observed.

In this experiment, the larynges, after neurectomy, appeared endoscopically similar to that described by authors studying both experimentally induced laryngeal paralysis (Dedo, 1970) and naturally occurring idiopathic laryngeal paralysis (O'Brien *et al*, 1973; Wykes, 1983; Greenfield, 1987). Unlike the laryngeal action seen in many of the idiopathic laryngeal paralysis dogs, no paradoxical movement was observed in the experimental animals. The absence of this movement indicates that the vocal cords are not drawn into the laryngeal lumen during inspiration and must therefore be stabilised by some factor not present in some of the dogs with the naturally occurring disease. It may be that, in the recently denervated dorsal cricoarytenoid muscles, sufficient random fibre contraction occurs to counteract the negative pressures pulling the vocal cords into the lumen. Another possibility is that the unaffected cricothyroid muscle may act to tense the vocal cord during inspiration, thus preventing collapse.

The overall reduction in arterial oxygen tension found in the neurectomised dogs in this study is similar to the findings recorded in dogs with idiopathic laryngeal paralysis (Love *et al*, 1987). The cause of this reduction in PaO<sub>2</sub> is probably due to the paramedian position of the vocal cords and the arytenoids obstructing the upper airway and restricting airflow. The actual value of the mean PaO<sub>2</sub> recorded in the experimentally induced laryngeal paralysis dogs (mean PaO<sub>2</sub> 100mmHg) and idiopathic laryngeal paralysis cases (mean PaO<sub>2</sub> 77mmHg) previously reported by Love *et al* (1987) differed substantially. This is most likely to be due to the result of administration of sedative drugs and the greater mean age of the dogs in the latter study.

The characteristic tidal breathing flow-volume loop shape indicating a variable extrathoracic obstruction in eight of the ten dogs following neurectomy, was consistent with previously reported findings in dogs (Amis *et al*, 1986) and human neonates (Abramson *et al*, 1982). The inspiratory flow is primarily affected as evidenced by flattening of the inspiratory arc. This pattern apparently occurs because the paralysed arytenoid cartilages and partially paralysed vocal cords cause narrowing of the *rima glottidis*. This is likely to have several effects. Firstly, it causes increased resistance and thereby a reduction in inspiratory flow. Secondly, the speed of the inspired air is increased at the site of the narrowing resulting in a reduced intra-luminal pressure (combined Bernoulli and Venturi effects). Since the paralysed dorsalcricothyroid muscles are no longer able to resist these forces, collapse of the unstable arytenoid cartilages and vocal cords into the laryngeal lumen, results.

The reduced peak inspiratory and expiratory flow found after neurectomy in the two remaining dogs, indicated that a fixed lesion was present causing an airway obstruction. This indicates that bilateral recurrent laryngeal neurectomy can cause, in some cases, a reduction to both inspiratory and expiratory air flows. Similar findings have been recorded in dogs (Amis *et al*, 1986) and humans (Miller and Hyatt, 1973) in clinical cases of laryngeal paralysis.

The rapid oscillations or "noise" contained in the tracings of the reduced inspiratory arcs in the tidal breathing flow volume loops of the neurectomised dogs are thought to be due to marked flow turbulence (Miller and Hyatt, 1973) and probably reflect the oscillations of the partially paralysed vocal cords.

One difference between the loop pattern produced in the neurectomised dogs and those produced by dogs with the naturally occurring disease is the presence of a relatively high peak inspiratory flow value recorded in the former. This could be due to several factors. The first, is that the dogs may have increased their inspiratory effort at this point in the breathing cycle, perhaps by utilising the accessory respiratory muscles. Indeed, the dogs were observed to forcibly expand their chest using their intercostal and diaphragm muscles during the latter phase of inspiration. This would result in an increased negative pressure within the lower respiratory tract and hence an increased flow rate, providing that the *rima glottidis* remained unobstructed. It would appear that dynamic collapse does not occur during quiet respiration in the neurectomised dogs, since paradoxical movements of the vocal cords were not observed in any of these animals (q.v.). However, in dogs with idiopathic laryngeal paralysis, the negative pressure created within the laryngeal lumen during reposed inspiration is sufficient to pull the arytenoids and vocal cords medially resulting in obstruction and hence cessation of airflow. Furthermore, it has been demonstrated that maximum inspiratory flow rates in man are determined by, not only airway resistance, but also, the forces of the inspiratory muscles (Hyatt and Flath, 1966). In these maximal inspiratory flow-volume curves it is the midrange of vital capacity that is predominately reduced (Miller and Hyatt, 1973) in extrathoracic airway obstructions. The second cause of the relatively high peak inspiratory flow may be due to the erroneous measurement of the peak point. Many of the inspiratory tracings in the neurectomised dogs showed undulating oscillations and it was difficult to determine exactly where the peak flow point lay.

The increase in inspiratory time recorded in the neurectomised dogs is similar to that reported in dogs with idiopathic laryngeal paralysis (Amis *et al*, 1986). A greater inspiratory duration is apparently required to compensate for the reduced airflow resulting from the laryngeal obstruction.

## IV<sub>2</sub> THE EFFICACY OF CASTELLATED LARYNGOFISSURE WITH VOCAL CORD RESECTION IN ALLEVIATING THE EFFECTS OF EXPERIMENTALLY INDUCED LARYNGEAL PARALYSIS

Although castellated laryngofissure with vocal cord resection was moderately effective in alleviating the effects of laryngeal paralysis, clinical signs of upper airway obstruction were still present in three out of the five dogs after this procedure. In two of these dogs, this respiratory stridor was noticeable even at rest. A similar persistence of stridor has been recorded in dogs with naturally occurring laryngeal paralysis after this operation (Gourley *et al*, 1983). The remaining dog with signs of airway obstruction following this surgical technique, showed marked signs of respiratory distress when excited or stressed. The cause of this was not established, but the reduced inspiratory flow shown in the post-operative tidal breathing flow-volume loop indicated that a functional respiratory obstruction remained after surgery in this animal. This dog also showed compromised respiratory function in cold climatic conditions which may have been the result of an increased exposure of the cold receptors in what remained of the partially resected vocal cords. Increased exposure of the receptors may have lead to a greater stimulation and, as a consequence, reflex apnoea.

Another unfavourable effect of this surgical procedure is that an intermittent cough is present during the immediate post-operative period. The origin of this cough was not determined but could have been due to upper airway irritation from the severed laryngeal and tracheal mucosae.

The inconsistent improvement in upper airway function, as determined by endoscopy, blood gas analyses, and tidal breathing flow-volume loop analyses, following this operation may have been due to a number of reasons. Firstly, the degree of widening of the *rima glottidis* could have been insufficient for normal breathing requirements. Secondly, the operation may have failed to stabilize the arytenoid cartilages which were therefore still responsive to the negative pressures within the laryngeal lumen during inspiration, resulting in dynamic collapse of these structures. Thirdly, the operation produces an asymmetry of the glottis which probably creates air flow turbulence and increases resistance to airflow.

The surgical technique used in this experiment varied slightly from that in the original description by Gourley *et al* (1983). The two modifications made were:

- 1) three to five millimeters of the vocal folds were preserved ventrally. This possibly reduced the risk of a web of granulation tissue forming across the ventral commissure of the *rima glottidis* during the healing process (Aron and Crowe, 1985).

and

- 2) the tracheostomy tube was removed as soon as the dog was standing. This early removal of the tube was not associated with any clinical problems and indeed had the benefit of minimising patient discomfort following surgery.

#### IV<sub>3</sub> THE EFFICACY OF BILATERAL ARYTENOID LATERALISATION IN ALLEVIATING THE EFFECTS OF EXPERIMENTALLY INDUCED LARYNGEAL PARALYSIS

A comparison of the clinical, endoscopic, blood gas and tidal breathing flow-volume loop results following this operation compared with those taken after castellated laryngofissure indicated that bilateral arytenoid lateralisation was more effective in alleviating the obstructive effects of bilateral recurrent laryngeal neurectomy. Moreover, when care is taken to ensure correct positioning of the lateralisation suture, more dependable results can be obtained using this technique.

Consistent improvement in the clinical signs of laryngeal obstruction was achieved by this operation. In addition, post-operative endoscopic observations showed that, on average, a wider and more symmetrical *rima glottidis* could be achieved with this procedure than after castellated laryngofissure.

Further proof of bilateral arytenoid lateralisation's superiority was demonstrated by the significant improvement in post-operative PaO<sub>2</sub> levels and inspiratory airflow. These increases were probably due to the combined effects of producing a wider *rima glottidis* and stabilising the arytenoid cartilages.

The operation, however, does have some disadvantages. The tidal breathing flow-volume loops, whilst demonstrating an improved inspiratory flow also showed a diminution of the expiratory airflow braking mechanism of the larynx. This was probably the result of fixed abduction of the arytenoid cartilages produced by the surgery. The procedure, therefore, seemingly causes a loss of the larynx's ability to regulate expiratory flow and duration.

In addition, bilateral arytenoid lateralisation is a relatively difficult surgical technique for the inexperienced surgeon. It requires a thorough knowledge of the larynx and surrounding structures. Even when performed by experienced surgeons, some inconsistency in placement of the lateralisation suture can result. This may produce an asymmetrical laryngeal airway and a narrower *rima glottidis* than is desirable. The two dogs in this experiment where one of the lateralisation sutures was placed more rostral in the thyroid cartilage had not only a narrower glottic opening, but also a lower post-operative Pao<sub>2</sub> level than those of the other dogs on which this procedure was used.

The ideal position of the lateralisation suture to achieve the most advantages arytenoid abduction is unknown. Maximum abduction is achieved by placing the suture caudally and dorsally either, within the thyroid cartilage as in this study, or as recommended by some authors within the cricoid cartilage (LaHue, 1989; White, 1989).

Various suture materials have been used for the lateralisation procedures. Theoretically the material chosen should

- : be capable of maintaining tensile strength for a long period of time.
- : have minimal ability to cut cartilage under tension.
- : have excellent tissue compatibility.
- : be incapable of harbouring bacteria.
- : have good knot security.
- : be readily available and inexpensive.

A non-absorbable monofilament suture such as nylon or polypropylene is used by most surgeons (Rosin and Greenwood, 1982; LaHue, 1989; White, 1989) and appeared to act satisfactorily in this study. Stainless steel, however, has a tendency to cut through the cartilage (White, 1989).

The advantages of bilateral arytenoid lateralisation over unilateral lateralisation were not addressed in this study. Harvey and Venker-van Haagen (1975), Love *et al* (1987), LaHue (1989) and White (1989) advocated the former, whilst Rosin and Greenwood (1982) supported the latter. Certainly, observations on larynges from cadavers by Harvey (1983) revealed that vastly increased glottal cross sectional area was achieved when the bilateral operation was used. It could be argued that dogs with permanently abducted arytenoids would be likely candidates for aspiration pneumonia. This hypothesis was not supported in this study, since none of the dogs exhibited a cough or showed any evidence of pneumonia at autopsy. Indeed, it seems preferable that both sides should be stabilised in cases of bilateral laryngeal paralysis so that the effects of dynamic arytenoid collapse during inspiration can be minimised.

#### IV<sub>4</sub> EVALUATION OF DIAGNOSTIC TECHNIQUES USED

##### i) Laryngoscopy

The advantages of laryngoscopy in the diagnosis of laryngeal paralysis and in the assessment of the effects of surgery are, that it permits direct evaluation of the size of the *rima glottidis*, it is easy to perform and requires little specialised equipment. Although, in

this study, fiberoptic endoscopic examination of the larynx was found to be preferable, it was possible to gain similar information by using a simple lighted laryngoscope. Since the width of the *rima glottidis* is clearly observed by laryngoscopy, the degree of dilation achieved by the two restorative surgical techniques can be readily compared.

Although both adductor and abductor muscles are involved in laryngeal paralysis, diagnosis by laryngoscopy is primarily based on observing abductor dysfunction. This involves correlating any movement of the arytenoid cartilages and vocal cords seen with the phase of respiratory cycle. In this study, it was essential to observe the larynx throughout several deep inspirations, in order to appreciate the range of movements that occurred prior to neurectomy. The characteristic immobility of the arytenoid cartilages and vocal folds, reduced width of the *rima glottidis* and the loss of abduction during inspiration seen after neurectomy, were easily recognised. Similarly any confusing movements such as the larynx being pulled caudally by the action of the *sternothyroideus* muscles, or the tensing of the vocal cords by the action of the cricothyroid muscles, were promptly detected. Laryngoscopy can, therefore, be recommended, for assessing laryngeal movements as it is simple and informative and has been observed to correlate accurately with electromyographic findings (Venker-van Haagen, 1980).

The main disadvantage of laryngoscopy is that it requires general anaesthesia. This may present a hazardous risk in the respiratory distressed patient and requires some knowledge, on the part of the observer, of the relationship between depth of anaesthesia and possible laryngeal movements. One other associated problem of laryngoscopy in the dog is the inability to observe the laryngeal movements during exercise. The degree of dynamic collapse that might occur under these circumstances is therefore unknown.

#### ii) Radiography

In this study, radiographs of the chest were of little value for assessing the effects of laryngeal function on the remainder of the lower respiratory tract. They do, however, have a diagnostic role in idiopathic laryngeal paralysis cases for detecting other causes of respiratory embarrassment and for determining the presence of any concurrent chest disease (Gaber *et al*, 1985; La Hue, 1989).

#### iii) Blood Gas Analysis

The assessment of arterial oxygen and carbon dioxide tensions provides a measurement of respiratory efficiency. Lowering of PaO<sub>2</sub> or elevation of PaCO<sub>2</sub> may indicate either ventilation/perfusion inequalities within the lung, or airway obstruction, or both.

Several factors seem to affect the PaO<sub>2</sub> level in healthy conscious dogs at rest. These include hyperventilation, excitement (Clark *et al*, 1977) and sedation (Love *et al*, 1987). In comparison, the number of variables in PaO<sub>2</sub> sampling in dogs after exercise are increased, making interpretation of the results difficult. These include the fitness of the dog, the duration and exertion of the exercise and the time interval between ceasing exercise and arterial puncture. Some or all of these factors may account for the variable PaO<sub>2</sub> measurements recorded in this study, where some of the dogs had a elevated PaO<sub>2</sub> after exercise, whilst, in others, the value remained unchanged or decreased.

It has also been reported that dogs with clinical signs of respiratory disease may have blood oxygen tensions within the normal range (Clark *et al*, 1977; Love *et al*, 1987) and that major interference with respiratory function must occur before the arterial blood tension falls. In this study, the PaO<sub>2</sub> parameters recorded in dogs after neurectomy, although significantly lower than those measured in the healthy state, would not be regarded as indicating significant hypoxia.

However, despite the limitations of this technique, blood gas analysis in dogs at rest was useful in assessing firstly, the degree of reduced PaO<sub>2</sub> in laryngeal paralysis and secondly the scale of improvement after surgery. Blood gas analyses after exercise does not appear to be as helpful, because of the inability to standardise the protocol.

Clarke *et al*, 1977 established that measurement of PaO<sub>2</sub> is a more useful evaluation of respiratory failure than the determination of PaCO<sub>2</sub>. Thus, the lack of statistically significant differences in the PaCO<sub>2</sub> measurements in this experiment were not surprising. In general, PaCO<sub>2</sub> rises only with respiratory depression or with an overall fall in ventilation (Clarke *et al*, 1977), and only where there is a severe increase in airway resistance will PaCO<sub>2</sub> values increase (Nunn, 1977).

iv) Tidal breathing flow-volume loop analysis

Tidal breathing flow-volume loops in this study were easily obtained in untrained, unsedated dogs. This co-operation from dogs was also reported by Amis *et al* (1986). For each dog, the loop shape was consistent and reproducible. Distinct tidal flow volume loop patterns were produced by the changes in the extrathoracic airway obstruction after neurectomy and later, after laryngeal surgery. This finding was in agreement with other investigations in dogs (Amis *et al*, 1986) and in human neonates (Abramson *et al*, 1982). Thus, tidal breathing flow volume loops can be helpful in identifying the level of the airway obstruction and the assessment of the response to treatment.

There are two main disadvantages of tidal breathing flow loop analysis. Firstly, in contrast to maximum, forced expiratory and inspiratory flow loops, the flow rates are relatively low and therefore they are less sensitive to the detection of early or minor obstructive disease. Secondly, they are effort dependant and therefore they may be markedly altered in the conscious dog that is able to change its breathing pattern (Amis *et al*, 1986). The loop shape obtained can be altered by a change in the respiratory rate, and a change in the driving pressure. An increase in the latter can increase the airflow during tidal breathing, which may obscure a flow limitation. For these reasons, it can be difficult to standardise measurement of tidal breathing flow-volume loops thus making numerical interpretation difficult. Amis *et al* (1986) attempted to normalise the above effects by calculating the ratios of the absolute flow measurement. This technique was tried in this experiment with varying success. One problem appeared to be the variation in shape of the loops for each individual dog. Four loop types have been described in healthy dogs (Amis *et al*, 1986) and therefore the pooling of data in this study from such a small number was likely to make statistical analyses unreliable. It is also known that body size has an influence on tidal breathing flow volume loop shape and this factor was minimised in the present study by selecting dogs of similar size and breed.

Tidal breathing flow volume loops are unable to indicate the severity of respiratory tract obstruction (Abramson *et al*, 1982; Amis *et al*, 1986). It has been shown that even maximum forced expiratory and inspiratory flow ratios provide no reliable correlation between the numerical value obtained and the degree or severity of obstruction present (Topham and Empey, 1974). Nevertheless, qualitative assessment of tidal breathing flow-volume loops in individual patients can be useful in diagnosing upper airway dysfunction and, by evaluating the change in loop shape, the "success" of surgery.

Other potential sources of errors with tidal breathing flow-loop analysis relate to the sensitivity and calibration of the measuring equipment. Marked flow turbulence produces "noise" on the loops and atypical loops are produced by the presence of more than one obstructing lesion within the airways.

v) Autopsy examination

The size of the *rima glottidis* at autopsy gave no direct indication of its ante-mortem dimensions. In the live animal, the positioning of the arytenoids and vocal cords is dependant on the combined effect of the intrinsic laryngeal muscles and the accessory muscles of respiration. A wider opening of the glottis is achieved during inspiration with abduction of the arytenoids by the dorsal cricoarytenoid muscle coupled with a caudal movement of the laryngotracheal complex by the *sternothyroideus* muscles. The loss of this latter effect in the larynx probably caused some of the reduction in glottal size seen at

at autopsy when compared to the final endoscopic examination.

Nevertheless, inspection of the larynges at autopsy did allow assessment of the symmetry of the *rima glottidis* and conformation of suture placement and security.

#### IV<sub>5</sub> CRITICAL EVALUATION OF THE EXPERIMENT

This experiment studied the efficacy of two surgical procedures in alleviating the effects of bilateral recurrent laryngeal nerve resection in otherwise healthy dogs. The experimental dogs were used to simulate the conditions of idiopathic laryngeal paralysis. Dogs with the naturally occurring disease, however, appear to have, in many cases, neurological problems affecting other nerves in addition to the recurrent laryngeal nerves. Thus, the experimental dogs did not mimic idiopathic laryngeal paralysis exactly. Nevertheless, the surgically induced laryngeal paralysis in the experimental dogs did serve as a suitable model for the assessment and comparison of the two surgical techniques.

Each diagnostic technique used throughout this experiment was also subject to variation. Laryngoscopy provided the most accurate assessment of the width of the *rima glottidis* and degree of laryngeal obstruction.

The results of blood gas analyses were sensitive to variation depending on whether the dogs were excited, hyperventilating or breath holding at the time of sampling. Certainly there was some difficulty in standardising the degree of exercise undertaken by the dogs and the time of arterial sampling after that exercise rendering the post-exercise blood gas analyses open to discrepancy. Resting blood gas samples would appear to give a more accurate reflection of the animal's respiratory status.

Tidal breathing flow-volume loops were subject to variation in two aspects. Firstly, there were problems related to equipment sensitivity, such as the pneumotachography having an inadequate frequency response, and the effects of turbulence causing distortion of the basic loop (Finucane, Egar and Dawson, 1972). Secondly, the co-operation of individual dogs undergoing this functional test, varied. Changes in breathing pattern, posture or excitement had an effect on the tidal breathing flow-volume loop obtained and an effect on the calculated ratios, making it difficult to standardise and numerically interpret the loops, particularly between dogs. Nevertheless each dog did appear to have a typical loop pattern and comparison of the loops shape in individual dogs was meaningful and helpful.

The two surgical procedures studied were also subject to slight variation in technique in individual dogs. This was minimised by the operations being performed by the same team of personnel, but nevertheless, these differences are probably reflected in the results.

The number of dogs used in this experiment was kept to a minimum because of ethical considerations, but was sufficient to establish a significant difference between efficacy of the two surgical procedures.

Castellated laryngofissure with vocal fold resection and bilateral arytenoid lateralisation, both alleviate some of the airway obstruction caused by bilateral recurrent laryngeal neurectomy. However, the results from this study indicate that bilateral arytenoid lateralisation is superior for this purpose. The operation produced more consistent clinical improvement, a wider *rima glottidis* and significant increases in post-operative PaO<sub>2</sub> levels and inspiratory airflow. The technique also has the advantage of causing less patient discomfort in the immediate post operative period, because the laryngeal mucosa is usually not incised and there is no need to use a tracheostomy tube during the procedure. Bilateral arytenoid lateralisation did not, however, allow the larynx to resume normal function. It had the disadvantage of diminishing the regulatory control of the larynx by preventing the expiratory braking mechanism.

Laryngoscopy, blood gas determination at rest and tidal breathing flow-volume loop analyses are all useful techniques for (a) diagnosing laryngeal dysfunction and (b) for evaluating the success of surgery to alleviate laryngeal obstruction.

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