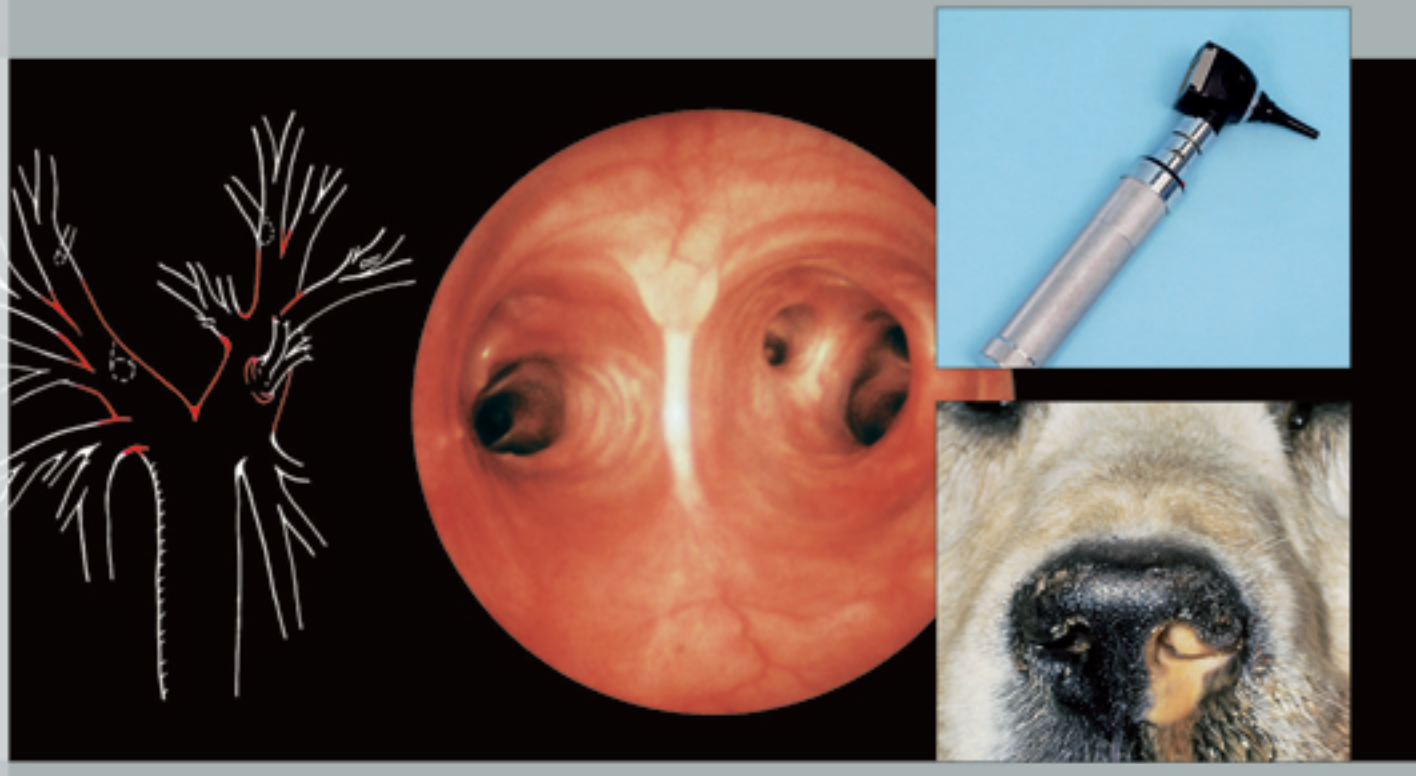
Anjop J. Venker-van Haagen

Ear, Nose, Throat, and Tracheobronchial Diseases in Dogs and Cats



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Abbreviations

ADC	Analog-to-digital converter
B.O.S.	Brachycephalic obstructive syndrome
BAER	Brain stem auditory evoked response
BERA	Brain stem evoked response audiometry
CPG	Central pattern generator
CRDs	Complex repetitive discharges
CT	Computed tomography
DAC	Digital-to-analog converter
dB SPL	Decibel sound pressure level
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyogram/Electromyography
F generations	Offspring generations
FISH and RH mapping	Methods for gene mapping used for association studies
Hz	Hertz
i.d.	Inside diameter
Ig	Immunoglobulin
kHz	Kilohertz
MRI	Magnetic resonance imaging
Nd-YAG laser	Laser using Yttrium-Aluminum-Garnet with Nd ions
NSAIDs	Nonsteroidal anti-inflammatory drugs
NTS	Nucleus tractus solitarius
$p < 0.01$	The probability that the result is due to chance is less than 1 in 100 (highly significant)
P generation	Parent generation
SLN	Superior laryngeal nerve/Cranial laryngeal nerve
T-tube	T-shaped tracheal tube
V	Trigeminal nerve
VII	Facial nerve
IX	Glossopharyngeal nerve
X	Vagus nerve
Xph	Pharyngeal branch of the vagus nerve
XII	Hypoglossal nerve

Preface

Among my fellow members of the International Veterinary Ear Nose and Throat Association (IVENTA), the reason for marking out specialization in diseases of the ear, nose, throat, and tracheobronchial tree is clear. They recognize that many of the diseases of these organs have similar clinical signs, similar infectious etiology, or similar regulatory mechanisms, and that these organs share cranial nerves for the execution of their functions.

Most of the knowledge in this field has been provided to veterinarians—and regularly updated by—the major textbooks of small animal internal medicine and small animal surgery. The textbook presented here aims not only to provide a more complete overview of diseases of the ear, nose, throat, and tracheobronchial tree but also to increase understanding of the functions of the respective organs for hearing, olfaction, swallowing, vocalization, and conditioning inspired air for gas exchange in the lung.

Each chapter begins with functional considerations of its subject and ends with a clinical topic chosen for its uncommon complexity. The book is readily accessible through a detailed list of contents and an elaborate index. It is intended to provide information of interest to academics as well as practitioners and students.

I am grateful to Dr. Bruce Belshaw for editing the English language with care and experience. Mr. Joop Fama handled the figures and made them shine, and I am grateful both for his knowledge and for the time and care he gave to the work. Dr. Ulrike Oslage at Schlütersche Verlagsgesellschaft invited me to write this textbook and I thank her not only for the opportunity but also for the free hand which I had in preparing it. Dr. Simone Bellair at Schlütersche Verlagsgesellschaft fine-tuned the written material and the various pictures into a book and I am grateful for her professional skill.

I hope that readers will find this a pleasant and useful book and that interest in this field will continue to develop the science of ear, nose, throat, and tracheobronchial diseases in dogs and cats.

Utrecht, May 2005
Anjop Venker-van Haagen

Cat. China, second half of the 18th century. Cat shows signs of severe dyspnea: mouth breathing and fear. (From: Rijksmuseum, Diary 1999, week 44, with permission).

1 The Ear

1.1 Functional considerations

1.1.1 The ear as sensory organ

The ear is a sensory organ that has evolved to receive and transform the air waves or vibrations that we call sound into a code of neural impulses to be conveyed to the brain. The resulting distinct patterns of neural activity in the brain are then integrated with information from other sensory systems to guide behavior.[52] The first stage of this transformation occurs in the external and middle ear, which collect sound waves and amplify their pressure, so that the sound energy can be successfully transmitted from air to the fluid that fills the cochlea of the inner ear. In the inner ear the signal is divided into simpler, sinusoidal components, with the result that the frequency, amplitude, and phase of the original signal are faithfully converted by the sensory hair cells into encoded electrical activity in the auditory nerve fibers.[52] In the brain the earliest stage of central processing occurs in the cochlear nucleus, where the peripheral auditory information diverges into a number of parallel central pathways. These include the superior olivary complex, where the information from the two ears interacts to aid in localizing the sound in space. The cochlear nucleus also projects to the inferior colliculus of the midbrain, a major integrative center and the first place where auditory information can interact with the motor system. The inferior colliculus is an obligatory relay for information traveling to the thalamus and cortex, where more complex aspects of sound are processed.[52]

External ear. The external ear is the portion lateral to the tympanic membrane. It consists of the external auditory canal and its cartilaginous extension, the auricle. The medial part of the auditory canal is surrounded and supported by the temporal bone. The auricle is covered with skin which continues as the lining of the auditory canal. This skin is thin and in the medial part of the auditory canal it has little subcutaneous tissue, but in the lateral part it bears numerous hair follicles and ceruminous and sebaceous glands. Both the bony and the cartilaginous parts of the auditory canal provide an open passageway for air to the tympanic membrane. The tympanic membrane is the medial boundary of the auditory canal and its lateral component is formed by the epithelium of the skin lining the auditory canal. In mammals the auricle and the auditory canal are together regarded as a simple funnel that collects and crudely filters sound. In humans, however, the auricle and auditory canal increase the acoustic pressure at the tympanic membrane of sounds in the 1.5 kHz to 5 kHz range, which is the frequency range most important for speech perception.[37] In the dog and cat the auricle can be turned toward the source of sound; right and left auricles can move independently so that each ear can focus on separate sounds. Hence the animal does not have to turn its head to localize sounds, as humans do. It is not clear to what extent the shape of the auricle—large and erect like that of the German shepherd or folded like that of the cocker spaniel—influences hearing capacity, but the latter might seem to be disadvantageous, at least in theory.

Tympanic membrane. The tympanic membrane terminates the ear canal and covers the entrance to the tympanic cavity, thereby separating the external from the middle ear. The membrane is composed of three layers, the outer squamous cell epithelial layer being a continuation of the epithelial layer of the skin of the external ear canal, the inner mucosal layer being a continuation of the mucosa of the middle ear or tympanic cavity, and the intervening fibrous layer or tunica propria. The tympanic membrane is thin, slightly oval, semitransparent, and concave, owing to traction on its

medial side by the tensor tympani muscle. There are three ossicles (malleus, incus, stapes) in the middle ear, the manubrium of the malleus being fixed in the tunica propria of the tympanic membrane. The tensing of the tympanic membrane makes it ideal for the conversion of sound waves into vibrations of the malleus.

1.1.2. Middle ear matches different impedances

The major function of the middle ear is to match relatively low impedance airborne sounds to the higher impedance fluid of the middle ear. The term impedance in this context stands for a medium's resistance to movement. Because of the difference in impedance of the two media, 99.9 % of the sound energy is reflected at the interface between air and fluid and only 0.1 % is converted into pressure changes in the fluid. The middle ear overcomes this problem and ensures transmission of the sound energy across the air-fluid boundary. The first and major boost is achieved by focusing the force impinging on the relatively large diameter tympanic membrane onto the much smaller diameter membrane of the oval window, where the stapes, the last of the three ossicles, is attached and where the vibration of the tympanic membrane is conveyed to the fluid of the inner ear. A second and related process involves the mechanical advantage gained by the lever action of the three interconnected ossicles which link the tympanic membrane to the oval window.[47, 52]

Auditory ossicles. These are also attached to the wall of the epitympanum or dorsal part of the tympanic cavity by several ligaments. While the manubrium of the malleus is embedded in the tympanic membrane, the head is suspended in the epitympanum and is fused with the incus in a rigid joint. The long process of the incus is then linked to the stapes by another joint, one that is rigid in the direction of the piston-like movement of the stapes but flexible perpendicular to this movement. The stapes is suspended in the oval window of the cochlea by two ligaments. The stapedius muscle—the smallest striated muscle in the body—is attached to the head of the stapes. It pulls the stapes in a direction perpendicular to the piston-like motion and is innervated by the facial nerve. The other muscle of the ossicles is the tensor tympani muscle attached to the muscular process of the malleus. It pulls the manubrium of the malleus inward, tensing the tympanic membrane. This muscle is innervated by the trigeminal nerve. One of the functions of the two muscles of the middle ear is to support and stiffen the ossicular chain. In addition, because loud sounds are attenuated by the actions of the acoustic reflex—the contraction of both muscles in response to loud sounds—it is likely that another function of the reflex is to protect the inner ear from damage due to overexposure to excessive sounds. In addition to their protective function, the two muscles may attenuate low-frequency masking sounds that might otherwise interfere with auditory function. Contraction of the muscles during chewing would attenuate the associated sounds, which are largely low frequency, while preserving sensitivity to high-frequency external sounds.[37, 47]

Tympanic cavity. The ventral part of the tympanic cavity forms the tympanic bulla. Although its function is not known with certainty, it may be to improve the perception of sounds of very high and very low frequencies.[17] The middle part of the tympanic cavity, the mesotympanum, includes the tympanic membrane in its lateral wall and opens rostrally into the nasopharynx via the auditory (eustachian) tube. The auditory tube is short and its narrow lumen is compressed laterally and usually not open. The tube is confined by an inverted cartilaginous trough except along its ventral border.

The pharyngeal openings of the left and right auditory tubes are located in the lateral walls of the nasopharynx and are marked by accumulations of lymphoid tissue. The cartilage of the auditory tube extends into the medial wall of the pharyngeal opening and stiffens it. The auditory tubes facilitate equalization of the pressures on the opposite sides of the tympanic membrane. They open temporarily during each swallow and yawn. This permits escape of the slight secretion from the goblet cells and the glands in the lining of the tympanic cavity.[17]

Inner ear. The inner ear is housed in a bony labyrinth in the petrous portion of the temporal bone. It contains the membranous labyrinth with its sensory organs of hearing and balance. The membranous labyrinth consists of an interconnecting series of epithelial-lined tubes and spaces containing endolymph. There are three functionally-related parts: (1) the semicircular ducts, containing hair cells that detect acceleration of the endolymph caused by rotation of the head; (2) the utricle and saccule, containing hair cells with a membrane, the macula, that responds to linear acceleration of the head and its static position; and (3) the cochlear duct, which is the auditory portion of the labyrinth, resembling a snail shell and containing the hair cells involved in hearing, in the organ of Corti.

Cochlea. This is the bony shell surrounding the cochlear duct in a spiral of 3 $^1/_4$ turns (in the dog) around a hollow central core of bone, the modiolus, which contains the cochlear nerve. The osseous spiral lamina that winds around the modiolus, much like the thread of a screw, divides the lumen of the cochlea into the tympanic and vestibular canals, both containing perilymph. The osseous spiral lamina begins within the vestibule, the ovoid space that communicates with the cochlea rostrally and with the semicircular canals caudally, and ends at the apex. The vestibular canal communicates with the vestibule and hence the fluid within, the perilymph, is acted upon by the foot plate of the stapes resting on the membrane in the oval window. The round window is the opening, also covered by a membrane, by which the tympanic canal communicates with the middle ear. Both windows are at the basal end of the cochlea. The membranous cochlear duct completes the separation of the two canals but they communicate at the apex of the modiolus via a small opening, the helicotrema. Perilymph gains access from the subarachnoid space to the vestibule, cochlea, and semicircular ducts via the perilymphatic duct.[19]

1.1.3 Organ of Corti, sensory organ for hearing

The organ of Corti in the cochlear duct is the sensory organ for hearing. It contains many different cells, of which the hair cells are the most directly involved with hearing. The hair cells, so-called because of the hair-like bundles of cilia that project from their apex, are arranged in rows along the basilar membrane, the connective tissue that forms the floor of the cochlear duct. There are two main types of hair cells, outer and inner. The outer hair cells—about 12,000 in the human cochlea—are arranged in 3 to 5 rows along the basilar membrane, while the inner hair cells—about 3,500 in the human cochlea—are arranged in a single row. The outer hair cells are cylindrical and the inner hair cells are shaped like a flask or pear. The outer hair cells are incompletely surrounded by supporting cells (Deiter's cells on the basilar membrane side and Hensen's cells laterally) and they lie free in the perilymph covering the organ of Corti. The inner hair cells are tightly surrounded by supporting cells. The stereocilia of the outer hair cells form an inverted »W« and a basal body representing a rudimentary cilium (kinocilium). The inner hair cells have stereocilia arranged linearly and also a rudimentary cilium.[21, 47]

Stereocilia/hair cells. These are linked together by specific structures. The tips of the tallest outer hair cell stereocilia are embedded in the overlying tectorial membrane, whereas the tips of the inner hair cell stereocilia are free of the membrane. The tectorial membrane is anchored medially at the limbus, medial to the cochlear duct, and laterally to Hensen's cells by a fibrous net. The basilar membrane is attached to the modiolus at a different site and when the basilar membrane and the tectorial membrane are displaced vertically by the traveling wave created by sound energy delivered to the oval window, the displacement of the basilar membrane creates a shearing action between the cuticular plate, the base of the stereocilia, and the tectorial membrane. The stereocilia of the outer hair cells which are attached to both structures bend. The streaming movement of the fluid between the cuticular plate and the tectorial membrane may bend the inner hair cell cilia which are not attached to the tectorial membrane.[37] It is the bending of the stereocilia which initiates the electrical current in the hair cells and the formation of the electrical potential in the fibers of the cochlear nerve.

The resting potential of the hair cell is between −45 mV and −60 mV relative to the fluid that bathes the basal end of the cell. At the resting potential, only a small fraction of the potassium-selective transduction channels at the tip of the stereocilia are open. When the hair bundle is displaced in the direction of the tallest stereocilium, more transduction channels open, causing depolarization as K^+ enters the cell. Depolarization in turn opens voltage-gated calcium channels in the hair cell membrane, and the resultant Ca^{2+} influx causes more transmitter release from the basal end of the cell into the auditory nerve endings. Because some of the transduction channels are open at rest, the receptor potential is biphasic: movement toward the tallest stereocilia depolarizes the cell, while movement in the opposite direction leads to hyperpolarization. This allows the hair cell to generate a sinusoidal receptor potential in response to a sinusoidal stimulus.[52]

The basal and apical surfaces of hair cells are separated by tight junctions. The apical end with stereocilia is exposed to the potassium-rich, sodium-poor endolymph produced by the stria vascularis. The basal end is bathed in perilymph, the same fluid that fills the tympanic canal, and is K^+-poor and Na^+-rich. The endolymph is about 80 mV more positive than the perilymph, while the inside of the hair cell is about 45 mV more negative than the perilymph. The resulting electrical gradient across the membrane of the stereocilia (about 125 mV) drives K^+ through the open transduction channels into the hair cell.[52]

It is the inner hair cells that are the sensory receptors and 95 % of the fibers in the auditory nerve that project to the brain arise from this subpopulation. The terminations of the outer hair cells are almost all from axons that descend from cells in the brain. The outer hair cells have a function in changing the stiffness of the tectorial membrane by actively contracting and relaxing. In this way the outer hair cells sharpen the frequency-resolving power of the cochlea at particular locations, and thereby account for the cochlea's extreme sensitivity.[52] The basilar membrane is stiffer at the basal end than at the apex. The gradual change in stiffness causes sounds reaching the ear to create a wave on the basilar membrane that travels from the base toward the apex of the cochlea. This traveling motion is the basis for the frequency separation that the basilar membrane provides, higher frequencies activating sensory cells at the base of the cochlea and lower frequencies activating the sensory cells at the apex. The outer hair cells interact actively with the motion of the basilar membrane.

1.1.4 Ascending and descending pathways for hearing

The auditory nervous system contains an ascending and a descending pathway. The ascending auditory nerve extends from the organ of Corti to the cochlear nucleus in the brain stem and its bipolar cell bodies are in the spiral ganglion, located in the modiolar region of the cochlea. Fibers cross over from the cochlear nucleus to the contralateral superior olivary complex and from there the bundle continues as the lateral lemnicus before ascending to the inferior colliculus, in which most of its fibers terminate. The bilateral inferior colliculi are connected by commissural fibers and fibers also project to the medial geniculate body. From the medial geniculate body fibers project to the primary auditory cortex.[48, 49] Of the two descending pathways, the corticocochlear system connects the primary auditory cortex with the inferior colliculus and the periolivary nucleus, while the olivocochlear system connects these pontine nuclei with hair cells of, mainly, the contralateral cochlea, as described in the cat.[27]

1.1.5 Vestibular organ, the key to postural reflexes and eye movement

The vestibular organ and the cochlea are joined and the common membranous labyrinth that forms the auditory cochlea also comprises the utricle, the sacculus, and the semicircular canals of the vestibular organ. The vestibular membranous labyrinth within the osseous labyrinth is filled with endolymph. Like the cochlear endolymph, it is high in K^+ and low in Na^+. The space between the osseous labyrinth and the membranous labyrinth is filled with perilymph, similar in composition to that in the vestibular and tympanic canals of the cochlea, low in K^+ and high in Na^+. As in the cochlea, the cell bodies of the vestibular hair cells are embedded in perilymph and their stereocilia are in endolymph. Depolarization of these cells is similar to that of the cochlear hair cells (see above). Movement of the endolymph in the direction toward the tallest stereocilium causing an influx of K^+ via the top of the stereocilia, which in turn opens the voltage-gated calcium channels. The calcium influx causes more release of transmitter from the basal end of the cell. Movement away from the tallest stereocilium causes hyperpolarization of the hair cell and thus reduces nerve transmission. The vestibular hair cells are located in the utricle and the sacculus and in the three ampullae at the base of the semicircular canals.[53]

Vestibular hair cells. These hair cells provide the basis for vestibular function. The hair bundles have a specific orientation in each part of the vestibular organ. The accelerating movement of the endolymph in the semicircular canals causes the cap of the ampullary crest, the organ consisting of hair cells and their supporting cells, to bend following the movement of the fluid. Sensory receptors in the macule of the saccule and the utriculus consist of hair cells and associated supporting cells. Overlying the hair cells is the otolithic membrane, in which crystals are embedded. A shearing motion between the macule and the otolithic membrane occurs when the head undergoes linear acceleration.

Vestibular function is a key component in both postural reflexes and eye movements. Damage to the system affects balance, the control of eye movements when the head is moving, and the sense of orientation in space. The dysfunction of the vestibular system will be illustrated in the section on ototoxicity.

1.2 History and clinical signs

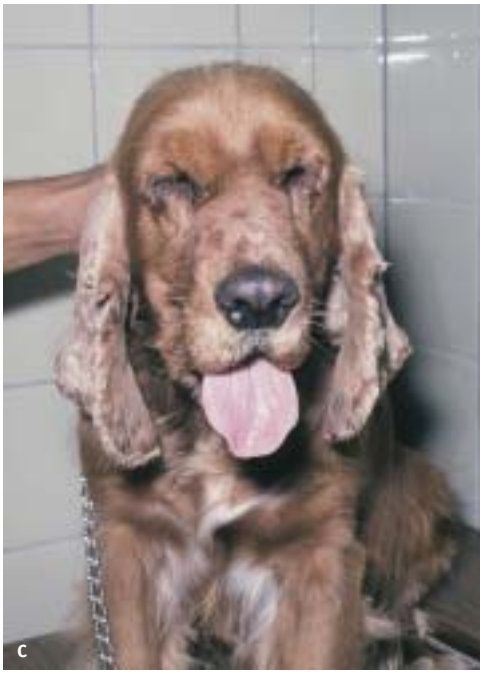
1.2.1 History

The medical history in diseases of the ear is characterized less by hearing disorders than by pain. Pain can be caused by disease of both the external ear and the middle ear, and can be unilateral or bilateral. When the inner ear is involved in the dog or the cat, vestibular dysfunction is more commonly mentioned in the history than loss of hearing. Diseases of the ear are usually presented as disorders affecting one or both ears exclusively. However, questioning may reveal signs of a more generalized skin disorder of which inflammation of the external ear is a part, or recurrent periods of fever and other signs of infection together with middle ear disease, or other signs of neurogenic disease rather than a vestibular problem alone. It is therefore essential that additional questions be asked about the animal's general condition, appetite, drinking, and physical activity; whether there have been changes in its habits; and whether there have been similar problems in the past, in either or both ears. The onset of ear problems may be sudden or gradual. The onset of signs caused by a foreign body in the external ear is often sudden and recognized by the owner. In contrast, inflammation of the external ear often begins gradually but becomes progressively worse; with such a history it is useful to ask what treatments have been tried. There are many ways of »treating« inflammation of the external ear which cause the inflammation to persist or even to increase. If parasitic infection is suspected, questions should be asked about contacts with other animals, of the same or different species.[79] Vestibular dysfunction is usually sudden in onset and the signs are usually dramatic, but hearing loss may go unnoticed, especially if unrelated to a specific event. In some cases of sudden deafness an associated event is mentioned by the owner, but it is not always easy to find a logical relation between this and the hearing loss. Unilateral hearing loss is often masked by normal hearing in the other ear.

Figure 1.1 a–c: This cocker spaniel was shaking its head continuously. Its ear canals were clean and not inflamed. (a) The auricles are long but were normal on visual inspection. (b) Palpation of the auricles revealed several heavy lumps of hair with accumulated dirt and food. (c) Clipping away the hair stopped the shaking.

1.2.2 Clinical signs

Pain is an »unpleasant sensory and emotional experience associated with actual or potential tissue damage«.[1, 44] It is a complex subjective experience, depending on the severity of the noxious damage, but also on a variety of additional cognitive and emotional aspects, and is therefore difficult to measure. It is even more complicated when a dog or cat is in apparent pain as described by the owner or caretaker. Ear pain is usually recognized if the animal tries to prevent handling of the ear, is less alert than usual, and sometimes very carefully scratches the ear or shakes the head. Pain and pruritus are not easily distinguished by casual observation, and in dogs and cats the signs of both can be suppressed by analgesic drugs. Pain caused by external ear disease may be severe and may change the dog's or cat's behavior, something often better recognized in retrospect when the pain disappears with successful treatment.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Signs of external ear disease. These are predominantly pain and pruritus, the pain sometimes causing the dog to turn its head slightly with the painful ear downwards. In dogs the auricle may be in an uncharacteristic position for the breed, and in cats it may be folded and turned backwards. The concave side of the auricle is usually thinly haired and inflammation of the external ear can be recognized by swelling and lesions of the skin, often with excess cerumen and exudate. Long hair on the auricle can become heavy with accumulated dirt and food, also causing the dog to shake its head in the absence of ear inflammation (Figure 1.1 a–c). Rubbing or scratching of the ear which injures the skin can lead to bacterial infection, increasing the inflammation and pain. Other signs of inflammation include scaling, hyperpigmentation, and tissue proliferation, the latter particularly at the base of the auricle on the concave side and around the entrance to the ear canal. Thickening of the auricle can result from acute or chronic dermatitis or perichondritis. The auricle can also be extremely thickened by a hematoma within the cartilage layer, presenting as a bulge on the concave side. In contrast to an abscess, which occurs most often in cats, a hematoma does not result in pain or general malaise.

Temperature of the auricle. The temperature of the auricle varies with the flow of blood, best appreciated on the concave side by the accompanying variation in its pink color. The color can vary not only with the ambient temperature but also with the balance between sympathetic and parasympathetic influence on blood flow in the auricle. A »red and warm« auricle may be a normal and transient finding, and is then usually bilateral.

Cerumen, or ear wax, is formed on the concave side of the base of the auricle and in the external ear canal. It is a mixture of the secretions of the sebaceous and the ceruminous glands. It has a waxy consistency and varies in color from

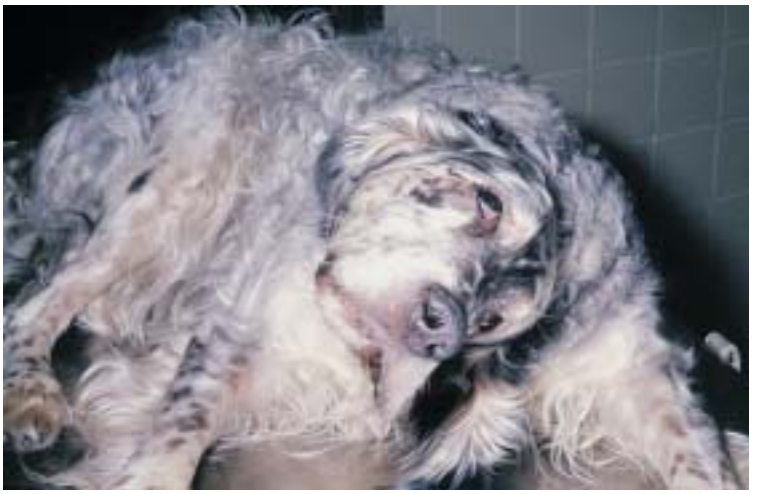


Figure 1.2:
An English pointer with signs of acute left-sided labyrinthitis: rotation of the head and cranial portion of the body, with the affected ear down and the eyes turned toward the affected side also.

yellow to brown. The secretion of the sebaceous glands being gray to white and that of the ceruminous glands being brown, the color of cerumen varies with the relative contribution of each. A thin layer of cerumen is normally present in the areas where the glands are located and sometimes small lumps are found at the base of the auricle at the entrance to the ear canal. The odor of cerumen is usually described as aromatic, but if the skin of the base of the auricle and the ear canal is inflamed, the production of cerumen can be increased and its composition can be changed. An increase and alteration in the bacterial flora can change the appearance of the cerumen and give it a more penetrating odor.[79] When combined with pus and detritus, its appearance and odor may become overwhelming and repulsive.

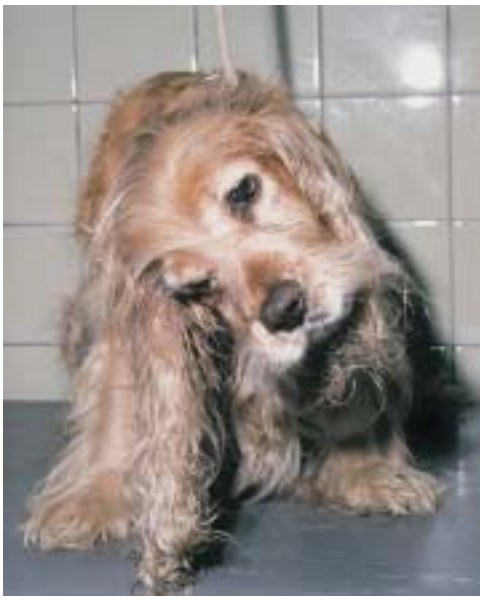


Figure 1.3:
A cocker spaniel, 3 months after ototoxic injury to the labyrinth of the right ear. The dog could stand and walk, but the rotation of its head was permanent.

Primary inflammatory disease of the middle ear mainly causes pain. It is usually unilateral and appears to be severe. The animal shuns petting of its head and loses alertness and appetite. Hearing loss is to be expected but is almost never mentioned by the owner. If the inflammation is purulent and the tympanic membrane is ruptured, there can be purulent discharge from the external ear canal.

Vestibular dysfunction is most apparent when it is unilateral. The signs of acute vestibular labyrinthitis are loss of equilibrium, inability to stand or walk, and falling to the affected side when trying to stand; rotation of the head and cranial portion of the body with the affected ear down; deviation of the eyes toward the affected side (Figure 1.2); and horizontal nystagmus with the rapid phase toward the unaffected side. The animal is severely disoriented, nauseated, and refuses food. The vestibular system responds immediately with central compensatory mechanisms (a process of neuroplasticity), sensory substitution (vision and proprioception), and learning processes. Within 3 days the nystagmus disappears, within a week the animal can stand with assistance, and within 3 weeks it can walk. The head rotation is usually permanent (Figure 1.3), but can be masked by compensation so that it is only observed when the animal's interest is absorbed by an event. Although the vestibular dysfunction is permanent, the persisting clinical signs vary according to the progress of compensation. However, those disabilities still present at 3 months will remain. In this regard, the clinical history may be helpful in determining the time of onset of the vestibular dysfunction if it is reviewed with an appreciation of the effects of compensatory mechanisms.

1.2.3 Physical examination

The auricles are inspected for symmetry and uniformity, and for abnormalities of the skin and hair. They are palpated to discover temperature differences and structural changes. They may be cooler than normal as a result of poor circulation, as in shock, or warmer because of hyperemia associated with inflammation. Structural changes can be due to tumor or ossification, which is a common reaction of the auricular cartilage to trauma. The entrance to the ear canal is inspected to evaluate its width. Normally the outer part of the canal, which is subcutaneous, is wide enough to be inspected without instruments. Both the medial part turning toward the temporal bone and the part within the temporal bone can only be inspected with an otoscope. The outer part is vertical when the animal's head is positioned to look straight ahead and is thus termed the vertical part, while simultaneously the medial part is horizontal and is therefore termed the horizontal part of the ear canal.[79] The external ear canal can be examined by palpation. The tip of the auricle is taken by one hand and stretched laterally while the first three fingers of the other hand are curved around the outermost part of the ear canal and then the tips of the fingers explore the cartilage medially. Pain elicited by this indicates inflammation. The lumen of the canal can be checked by softly compressing the cartilage. Proliferation of the lining increases the width of the canal and prevents it from being compressed. The cartilage can become ossified in chronic inflammation, resulting in a palpably hard and rigid tube. This is a painful disease, as the procedure will reveal.[79] A tumor that is invisible to inspection can be presumed (but not diagnosed) if there is a palpable local increase in the diameter of the ear canal. Special diagnostic techniques are required for examination of the parts of the ear inside the temporal bone.

Figure 1.4:
Otoscope with a rechargeable battery, an ear speculum with interchangeable cones (various sizes available), and a magnifying lens. This model has glass fibers incorporated in the wall of the speculum, transmitting a bright circle of light at its tip.

1.3 Special diagnostic techniques

Inspection of the external ear canal is very important in diagnosis of diseases affecting it. The entrance can be inspected with the naked eye but the remainder of the canal can only be inspected with the help of special illumination techniques and instruments. An otoscope is used for this purpose in the dog and the cat. This instrument consists of an ear speculum with interchangeable cones of several sizes, a small light source, and a magnifying lens (Figure 1.4). The best otoscopes have glass fibers incorporated in the wall of the speculum which transmit a bright circle of light at the tip while

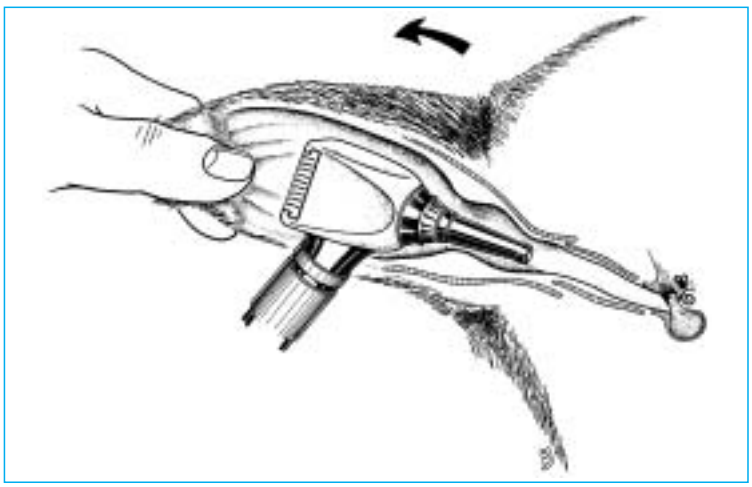


Figure 1.5:
Otoscopic examination. The left hand of the examiner grasps the right auricle firmly, pulling it out laterally and ventrally. This brings the vertical and horizontal parts of the ear canal into line, forming a straight, horizontal tube. The otoscope is held in the right hand and as it is inserted into the ear canal, the examiner looks through the magnifying lens.

keeping the light source out of the visual path. By choosing the appropriate size of cone for each, the same otoscope can be used to examine the ear canal and tympanic membrane in a small cat as well as a very large dog.

1.3.1 Otoscopic examination

The technique of otoscopic examination is the same in the dog and cat. The animal is held on the examination table in a sitting position or resting on its sternum, restrained by an expert assistant. Its head is held looking straight ahead. If possible, its mouth should not be tied closed during this examination, particularly with something passing behind the ears, for this usually presses the ear canal shut, or fixes it tightly against the head. When the animal is held securely, either sitting or lying on its sternum, the left hand (if the examiner is right-handed) is used to grasp the auricle securely and pull it out firmly, laterally and ventrally. This brings the vertical and horizontal parts of the ear canal into line, to form a straight, horizontal tube (Figure 1.5). The otoscope is then taken in the right hand, and while looking through the otoscope, so that everything is carried out under visual control, the examiner carefully inserts the otoscope into the ear canal. The otoscope should not be advanced unless the lumen of the ear canal is clearly in view. Bringing it into view is accomplished by moving the stretched auricle and with it the ear canal, first dorsally, then rostrally, ventrally, and caudally, while looking through the otoscope. Hence the ear canal and the otoscope are moved together and in alignment rather than moving the otoscope within the ear canal. This allows the entire canal and the tympanic membrane to be inspected with the least possible discomfort for the animal. The skin lining the ear canal is very sensitive and hence pressing upon it with the otoscope should be avoided.

Figure 1.6:
An apparatus developed for ear flushing. It consists of a small heater in which tap water is warmed to body temperature and held there. The water is sprayed into the ear canal through a short cannula, at the pressure indicated on the display.

Examination of the ear canal of dogs is occasionally hindered by excessive hair in the entrance. The hair can be plucked in bunches with a short jerk, using a round-tipped Péan forceps. It is not noticeably painful for the animal and scarcely results in hyperemia of the skin of the ear canal. If there is excessive scaling or cerumen or exudate, the ear canal must be flushed before a satisfactory otoscopic examination can be carried out. If microscopic examination for parasites or bacteriological examination of exudate is indicated, material must be collected for this purpose before flushing. Water or 0.9 % NaCl solution can be used to flush the ear. The fluid must have a temperature of 35 to 39 °C in order to prevent dizziness and even a shock-like reaction. The stream of water must be thin and forceful in order to wash out the long and narrow ear canal. The canal should be stretched out as described for otoscopy, so that the vertical and horizontal parts form a straight tube. An apparatus developed for ear flushing in humans is excellent for use in dogs and cats. It consists of a small heater in which tap water is brought to body temperature and held there (Figure 1.6). The water is sprayed into the ear through a short cannula at the pressure in the water supply pipe. The strength of the stream can be regulated by a lever on the handle but is limited to a physiologically

acceptable pressure. Most dogs and cats tolerate the flushing without sedation. Every ear can be flushed in this manner, even if it is not known whether the tympanic membrane is intact, with one exception: if after recent trauma there is blood in the ear canal (hence possible fracture of the base of the skull and the risk that contaminated water could reach the cranial cavity!), the ear may *not* be flushed.

After the ear canal has been flushed it is dried most effectively by letting the animal shake its head. This requires no encouragement, except occasionally in the cat, because shaking water from the ear canals is a natural reflex in dogs and cats. The otoscopic examination is then resumed. Sometimes it will reveal that the flushing must be repeated to clear away debris sufficiently to allow inspection of the entire wall of the canal and the tympanic membrane. The ear canal may be found too narrow due to inflammatory swelling and proliferation of the lining skin. Epithelial lesions may be found and tumors may also be recognized. Foreign bodies and bunches of hair (usually loose and coming from outside the ear canal) must not escape attention.[79]

Tympanic membrane. The tympanic membrane as it appears through the otoscope is a transparent membrane with the manubrium of the malleus visible as a clear white structure (Figure 1.7). The tympanic membrane is not oriented perpendicular to the long axis of the ear canal, but is tilted further inward, so to speak, ventrally and rostrally. The pars tensa is grayish-blue in the dog and the cat, with stripes fanning out from the manubrium embedded in the transparent lamina propria. There are small blood vessels along the manubrium. The pinkish-red pars flaccida is dorsal to the pars tensa and bulges into the ear canal if the pressure in the middle ear is increased (Figure 1.8). The tympanic membrane may be less transparent in older dogs and the manubrium is more slender in cats than in dogs (Figure 1.9).

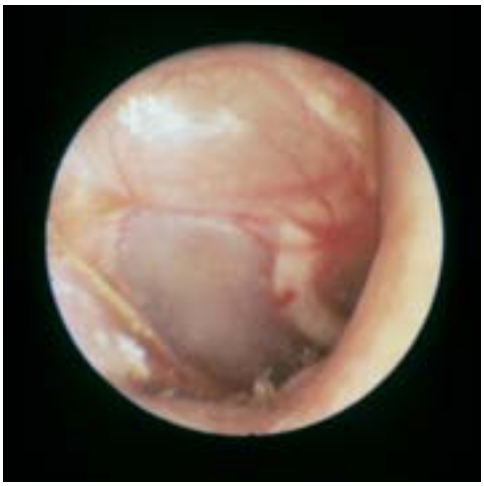


Figure 1.7:
Otoscopic view of the right tympanic membrane in a dog. The pars tensa is grayish-blue and transparent, with stripes fanning out from the manubrium, embedded in its lamina propria. There are small blood vessels along the manubrium. The pinkish-red pars flaccida is dorsal to the pars tensa.

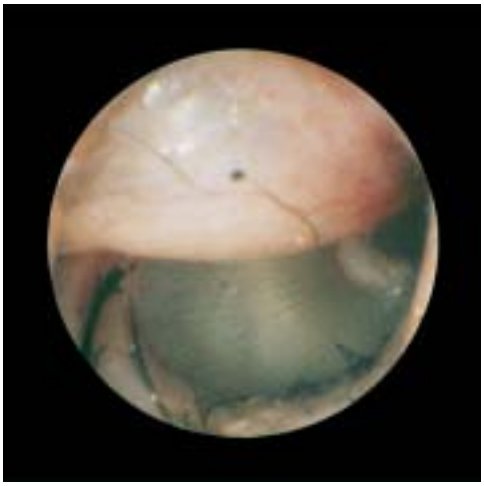


Figure 1.8:
The pars flaccida bulges into the ear canal when the pressure in the middle ear increases.

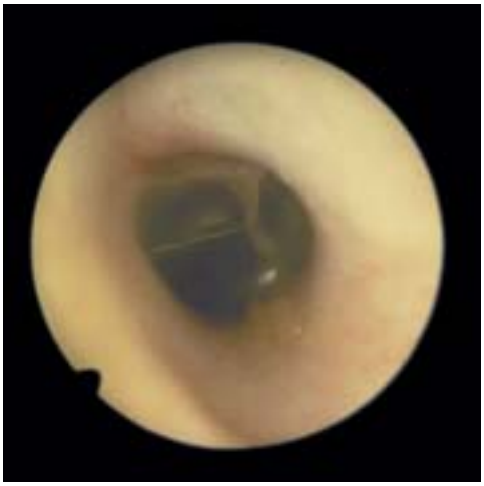


Figure 1.9:
Otoscopic view of the right tympanic membrane in a cat. The features are similar to those in dogs, but the manubrium is more slender in cats.

Rupture and other disorders of the tympanic membrane are diagnosed by otoscopy. Otoscopy is also very useful in the diagnosis of inflammation of the middle ear, for the inflamed mucosa lining the middle ear and the inner side of the tympanic membrane has a distinct redness and the transparency of the membrane is lost. Polyps growing in the middle ear in cats may rupture the tympanic membrane and be seen during otoscopy as round red masses in the medial part of the external ear canal. They may fill the entire ear canal when the diagnosis is delayed.

1.3.2 Diagnostic imaging of the ear

Diagnostic imaging of the external part of the ear canal seldom adds to information obtained by physical examination and otoscopy, but accurate visualization and assessment of the part embedded in the temporal bone can be very useful. Because of the difference in density between the bony components and the air-filled and fluid-filled spaces around and within the middle and inner ear, conventional radiography, computed tomography (CT), and magnetic resonance imaging (MRI) all lend themselves to this purpose.

Radiographic examination. The standard radiographic examination of the skull consists of a lateral and a dorsoventral projection. These radiographs may provide all the information that is needed or they may serve as a primary inventory examination. However, these standard projections are usually of limited value for examination of the temporal bones, and special projections such as rostrocaudal open-mouth projections, or radiographs with intraoral film, are required to avoid superimpositions. Radiography of the skull demands the most stringent and careful radiographic technique. Any unintended obliquity in positioning will hinder evaluation of the radiographs and thus deep sedation or general anesthesia is mandatory. When the diameter of the skull exceeds 10 cm to 12 cm, a grid must be used to diminish the unfavorable effect of scattered radiation on radiographic quality. A grid is not used when part of the skull is radiographed in open-mouth projections or when radiographs are made with intraoral film. Both nonscreen film in a light-proof envelope for intraoral application and screen film in a cassette with intensifying screens should be available. Nonscreen films require a much longer exposure time but produce radiographs with greater detail.

Radiographs provide a complete overview of all structures of the part of the skull being examined, with high spatial resolution. However, superimposition of structures on the film may make it difficult and sometimes impossible to distinguish a particular detail, especially when structures differ only slightly in density, which is often the case in temporal bone disorders. Even the tympanic bulla, a remarkable structure, is not shown in sufficient detail to distinguish features adequately for diagnosis and surgery.

Computed tomography (CT) is a radiographic technique that allows measurement of small absorption coefficients and differentials not recognizable by conventional radiography. With the x-ray beam collimated to a narrow fan shape, the x-ray tube revolves around the object during the exposure and the beam is altered as it penetrates the object. An array of sensitive detectors on the opposite side of the object quantitates the x-rays passing through, thereby determining the x-ray attenuation in different parts of the object, in all projections. Computer analysis of this collection of attenuation measurements results in a cross-sectional image of the object, which is displayed on a monitor with high spatial resolution and higher contrast resolution than provided by conventional radiographs. An intravenously administered radiographic contrast medium will increase the contrast between normal and abnormal tissue and facilitate the recognition

of blood vessels. Since the images represent slices of the object, they do not suffer from superimposition, but neither do they provide a survey view.[81]

The positioning of the patient is no less important in CT than in conventional radiography. The position of the object within the gantry determines the scan plane. The patient support is perpendicular to the opening of the gantry, and thus perpendicular to the scan plane. CT of the body is thus always axial, perpendicular to the long axis of the body, with some adjustment possible through angulation of the gantry relative to the patient support. Other scan planes can be achieved if the object can be positioned differently relative to the gantry opening. This is important, because interfaces between organs or structures can only be imaged satisfactorily when they are perpendicular to the scan plane. The head in particular can easily be examined in different scan planes. Transverse (coronal) scans of the head are made with the animal in prone or supine position and the head extended. Dorsal (axial) scans are made with the animal in supine position and the nose pointing upward. Sagittal scans are made with the animal in lateral recumbency and the head raised on a support.[81] The temporal bone can be studied in detail with regard to the bony details as well as soft issue density. The external auditory canal, the middle ear, and the inner ear can be studied, but identification of the type of substance producing an abnormal density is very limited. Densitometric readings obtained with a cursor of variable size are often unreliable because of partial volume averaging within the small cavities of the ear.[74]

Magnetic resonance imaging (MRI) is based on the magnetic properties of atomic nuclei which have an odd number of protons. Since protons are in continuous rotation, called nuclear spin, and have an electrical charge, they may be thought of as tiny magnets. Because the hydrogen nucleus consists of a single proton and hydrogen is abundant in living tissues, it is eminently suitable for magnetic resonance imaging.

In the body, the magnetic moments of the protons point in all directions, thus canceling each other. When the body is placed in a strong, homogeneous magnetic field (in clinical imaging, usually between 0.15 and 1.5 Tesla, 1 Tesla being 10,000 times the strength of the earth's magnetic field), the protons are forced into positions parallel to the axis of this magnetic field, not only spinning around their own axes, but also precessing around the axis of the magnetic field, like a child's spinning top. The frequency of precession is called the Larmour frequency and it depends on the strength of the external magnetic field.[81]

By use of radio waves of the same frequency as the precession frequency, the protons can be made to resonate: they will precess around the axis of the external magnetic field at a larger angle and all protons will be in phase. When the radio frequency wave is switched off, pulse relaxation occurs through two phenomena: the protons realign in the magnetic field (T1-relaxation) and they go out of phase (T2-relaxation). During the process of relaxation, the protons emit weak radio signals and it is these signals that are used to create the images. The images may be dominated by the concentration of protons (proton density), by T1-relaxation (T1-weighted images), or by T2-relaxation (T2-weighted images).

By using gradient coils to create small variations in the x, y, or z direction of the external magnetic field, certain scan planes and slices can be selected in which the precession of protons has exactly the right frequency to be susceptible to the radio wave pulses, and images can be made in different scan planes without having to reposition the patient. These images are influenced by the concentration of hydrogen nuclei in the part of the body being examined, by the chemical nature of the environment around the nuclei, and by the interactions between nuclei. MRI provides detailed anatom-

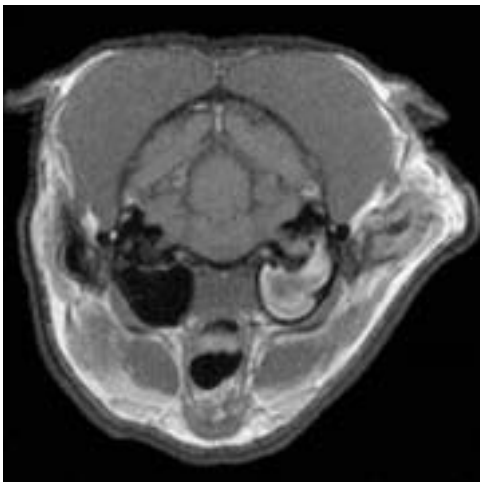


Figure 1.10:
MRI of the middle ears in a dog. The middle ear on the right side is filled by a mass that extends from the tympanic bulla to the epitympanum. The left middle ear is unaffected. The exact location, extent, and involvement of bony structures such as the ossicles and the labyrinthine capsule cannot be determined. (Courtesy of Dr. G. Voorhout, Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University).

ical images with soft tissue contrast that is superior to that of CT. The soft tissue contrast may be further enhanced by the intravenous administration of a contrast medium which is a paramagnetic substance (usually gadolinium). Its predominant effect is to shorten T1 and thus the regions that take it up are bright on T1-weighted images.[81]

Fluid, blood, and soft tissue masses within the temporal bone are readily identified by MRI as areas of abnormally high signal intensity. The exact location, extent, and involvement of bony structures such as the ossicles and the labyrinthine capsule cannot be detected, however (Figure 1.10). For this reason, CT remains the technique of choice for assessment of intratemporal abnormalities, with the exception of the petrous apex.[74]

Ultrasound imaging of the canine tympanic bulla has been described as an experimental procedure.[16] Fluid was introduced into the tympanic bullae of cadavers and the sonographic images were compared with those in living dogs without ear disease. It was possible to differen-

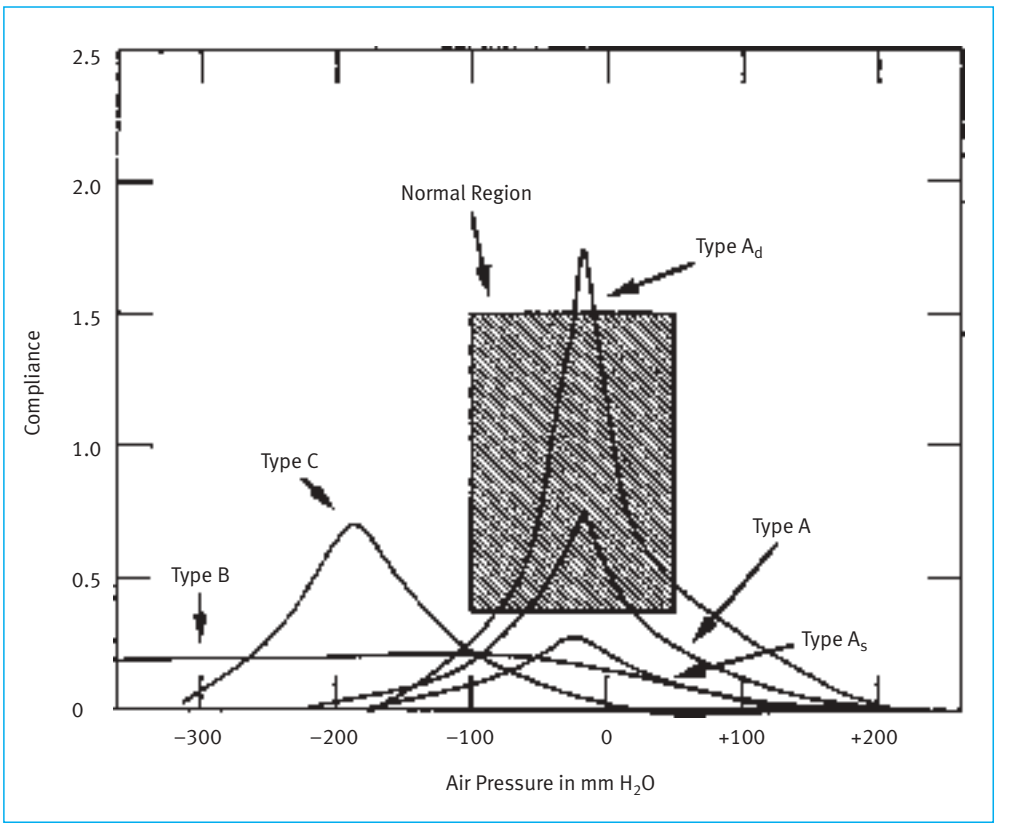


Figure 1.11:
Categorization of tympanograms in humans. Compliance (the reciprocal of stiffness) of the middle ear is on the vertical axis and the applied pressure is on the horizontal axis. Type A is a normal tympanogram, Type B is usually associated with fluid in the middle ear, and Type C is usually associated with eustachian tube dysfunction. Type A_s is a shallow variant of type A associated with fixation of the ossicular chain and Type A_d is the deep variant associated with disruption of the ossicular chain. (From: Hall III JW, Lewis MS. Diagnostic audiology, hearing aids, and habilitation. In: Snow Jr JB, Ballenger JJ, editors. Ballenger's Otorhinolaryngology Head and Neck Surgery. Hamilton: BC Decker Inc, 2003: 134–160, Fig. 5-2).

tiate between gas and fluid within the bulla. The method is attractive because ultrasound imaging is available in many veterinary practices and the examination was well tolerated in non-sedated dogs.

1.3.3 Tympanometry

Tympanometry is the continuous recording of middle ear impedance as air pressure in the ear canal is systematically increased or decreased. The technique is a sensitive measure of tympanic membrane integrity and middle ear function. Compliance—the reciprocal of stiffness—of the middle ear is the vertical dimension of the tympanogram and the varied pressure is the horizontal dimension (Figure 1.11). Tympanometry is a popular clinical technique in humans because it requires little specialized training and less than a minute to perform.[28] Immittance measurement is an electrophysiological method and does not depend on cooperation of the patient, other than accepting a soft pressure in the ear canal.

Tympanometry involves complete closure of the ear canal with a rubber plug containing three channels, one for introduction of the sound stimulus from the tympanometer, one for recording the sound reflected after application of the stimulus, and one for the programmed variation of pressure by the tympanometer (Figure 1.12). The compliance of the tympanic membrane is derived from the variation in the reflected sound in response to the programmed variation in pressure. The procedure evaluates the flexibility of the tympanic membrane and leads to the diagnosis of a change in pressure in the middle ear (usually a parameter of eustachian tube function) and an abnormally flaccid tympanic membrane. The absence of flexibility of the membrane can be due to its perforation, the filling of the middle ear with fluid, or adhesion of the tympanic membrane to surrounding structures (Figure 1.13).

Tympanometry was performed in an experimental setting in 7 conscious dogs, 50 dogs under anesthesia, and 43 under sedation.[20] In another study, 114 dogs (228 ears) were examined under anesthesia to compare the otoscopic and tympanometric evaluations of tympanic membrane integrity, and tympanometry proved to be reliable.[35] Comparisons of the tympanographic findings in 35 mixed-breed dogs and 21 laboratory-bred beagles revealed that the volume of the ear canal and peak compliance were both smaller in the beagles and that ear canal volume was dependent on body weight. All measurements were carried out under sedation or anesthesia.[12]

Comparison of tympanographic findings in cats with those in humans revealed that in cats the ascending and descending pressure runs differed, the descending runs being simpler in configuration and less influenced by the rate of pressure change. The stability of the tympanogram in cats and humans was similar in that the acoustic admittance increased sequentially during the first three pressure sweeps of a repetitive testing sequence in most individuals in both groups.[50]

In cats with intact tympanic cavities, measurements of acoustic input impedance were

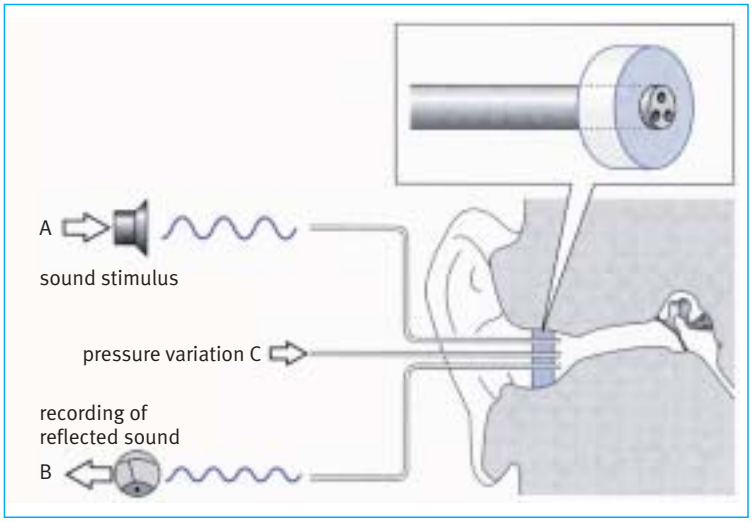


Figure 1.12: Tympanometry involves complete closure of the ear canal by a rubber plug that has three channels: one for introducing the sound stimulus from the tympanometer (A), one for recording the sound that is reflected (B), and one for the programmed variation of pressure by the tympanometer (C). (From: Huizing EH, Snow GB, editors. Leerboek keel-, neus- en oorheelkunde. Houten: Bohn Stafleu Van Loghum, 2003: Fig. 2.16).

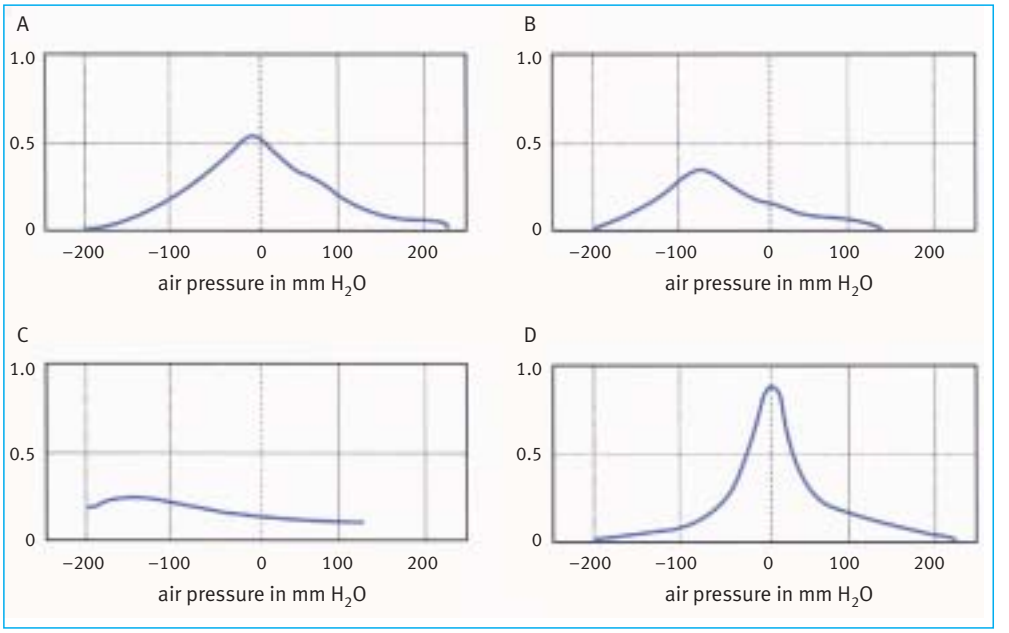


Figure 1.13: Examples of tympanograms recorded in humans. (A) normal; (B) left shift of the peak due to eustachian tube dysfunction; (C) a flat curve indicating fluid in the middle ear, perforation of the tympanic membrane, or adhesion of the tympanic membrane; (D) a high peak indicating abnormal flexibility of the tympanic membrane or rupture of the ossicular chain. (From: Huizing EH, Snow GB, editors. Leerboek keel-, neus- en oorheelkunde. Houten: Bohn Stafleu Van Loghum, 2003: Fig. 2.17).

found to be effective using a test signal of 300 Hz to 4 kHz.[38] The standard in human tympanometry is 226 Hz.

1.3.4 Neurological examination for vestibular dysfunction

The vestibular system, consisting of the vestibular organs in the labyrinths, the vestibular nerve, the vestibular nuclei, and the vestibular part of the cerebellum, provides orientation in space, stabilization of visual imaging during movement of the head, and stabilization of the body at rest and during movement. Other sensory contributions to normal equilibrium include visual information, proprioception, and auditory information. For functional evaluation of the vestibular system more sensory systems should be examined. In visual disturbances the head may be held in an abnormal position, as also occurs with severe pain in the eye. This causes the patient to move carefully and insecurely and to lift its feet too high. Abnormal positioning of the eyes may be caused by vestibular disorders or diseases of the eyes and adnexa, while nystagmus is usually caused by vestibular or central disorders. Testing the postural reflexes and reactions may reveal more abnormalities than can be explained by a vestibular disorder alone. In diseases of the vestibular organ, the hearing impairment is often ipsilateral to the affected labyrinth. To distinguish between central and peripheral vestibular dysfunction, examination of hearing ability in each ear separately will provide adequate information. Otoscopic evaluation of the tympanic membrane belongs to the tests and should be performed during the clinical examination.

1.3.5 Hearing tests

Several methods have been employed to test hearing in dogs and cats. A behavioral test is useful if the animal's response to sound in a clinical or home setting answers the questions of the owner and the veterinarian. The animal is placed in a familiar environment and a sound is produced out of its sight. The tone and intensity

of the stimulus should be within the range of the animal's normal hearing ability and it should be considered loud by the observers. A whistle used by sport referees is a good choice. The response is considered positive if the animal is startled by the sound and looks around to find its source, and negative if the animal is not disturbed and its attitude is unchanged. A positive response indicates only that the patient is able to hear the sound, while a lack of response indicates that it is deaf. Further classification of hearing would require that the patient be placed in a special soundproof room and conditioned to respond in, for example, a Pavlovian manner. The simple test does not detect a unilateral hearing disability, only a complete bilateral hearing deficit, although this is sufficient in cases of congenital deafness. When hearing appears to be defective and it is necessary to know the relative impairment of hearing in each ear separately, there are more sophisticated tests for use in dogs and cats.

Brain stem Evoked Response Audiometry (BERA) or Brain stem Auditory Evoked Response (BAER) hearing test. This is the test with the most accurate reference values in dogs; the difference is only semantic. The test has also been used in cats but appropriate frequency and volume limits have not yet been established. Each ear is stimulated separately with a sound that is well defined in intensity (sound pressure level in decibels, dB SPL) and frequency (Hz), while the waveform of the evoked potential in the brain stem is displayed and the amplitudes and latencies of its multiple peaks are recorded and analyzed. The stimulus is delivered within one ear canal and the opposite ear is plugged to prevent hearing. The evoked response is recorded over the brain stem by carefully positioned subcutaneous recording electrodes. Different methods have been described following these principals and others will undoubtedly be published. The details of the method which we have developed at Utrecht University are given here. This method was used to establish a normative data base for the combined evaluation of both intensity-specific and frequency-specific hearing in dogs.[73]

This study was carried out in 10 clinically-healthy dogs, 3.5 to 7.0 years of age (mean, 5.7 years) and weighing 12.5 to 21.3 kg (mean, 17.8 kg). Seven were mixed-breed littermates and 3 were beagles. Five were intact males, 1 was a castrated male, and 4 were intact females. None of the dogs had any clinical signs of neurological or otic disease, or showed abnormal behavior suggestive of hearing impairment. Otoscopic examination under anesthesia verified normal external ear canals and normal tympanic membranes in all 20 ears.

A light plane of anesthesia was induced with medetomidine (100 µg/kg IV), followed by propofol (1 mg/kg IV) within 5 minutes after medetomidine sedation. Anesthesia was maintained with medetomidine (25 µg/kg IV every 30 minutes) and propofol (1 mg/kg IV every 15 minutes during the first hour and every 20 minutes during the remainder of the session). At the end of the procedure, anesthesia was antagonized with atipamezole (250 µg/kg IV). Body temperature as indicated by rectal temperature was maintained at 36.5 to 37.5 °C by means of circulating water heating pads placed under the dog. With the dog in sternal recumbency, 3 needle electrodes were inserted subcutaneously: a recording electrode at the base of the auricle of the ear to be stimulated, a common (ground) electrode at the base of the auricle of the other ear, and a reference electrode over the occipital protuberance on the midline. The electrodes were cleaned mechanically and electrolytically before each session. The non-stimulated ear canal was plugged with an adjustable wax ear plug. The electrodes were attached to an amplifier having a total gain of 20,000 to 50,000 and a bandpass filter of 3 to 300 Hz. Positive electrical activity at the recording electrode produced an upward deflection on the recording.

Click stimuli were generated as rectangular waves 0.2 ms in duration at a nominal repetition rate of 10 Hz; phase-synchronized triggering was used to reduce power-line-derived noise. The generation of click stimuli was triggered by the data acquisition software and the stimuli were amplified by a variable headphone attenuator. They were delivered to the ear via a high-frequency bandpass speaker (tweeter) with a frequency response up to 38 to 40 kHz. The tweeter was connected to the ear via a flexible transmission tube, 4.5 cm long with an internal diameter of 8 mm, extending 1 cm into the vertical ear canal. Because of the flexibility of the tube, a tight fit in the ear canal could be accomplished visually without significantly altering or obstructing the internal dimensions of the tube or the ear canal.

Toneburst stimuli of 1, 2, 4, 8, 12, 16, 24, and 32 kHz were generated by software, transmitted to a digital-to-analog converter (DAC), and delivered to the ear in the same way as click stimuli. Two or more sine waves were used for rise and decay times, and one was used for plateau time, with a total duration of at least 1 ms. The 1, 2, and 4 kHz toneburst stimuli therefore had a 2-1-2 configuration, the 8 kHz toneburst stimuli had a 4-1-4 configuration, the 12 kHz toneburst stimuli had a 6-1-6 configuration, and so on. Stimuli alternated between rarefaction and condensation with a nominal repetition frequency of 10 Hz.

The intensity of the output of the tweeter was measured at the end of the flexible tube using a sound pressure meter to determine the stimulus sound pressure levels in decibels (dB SPL). The measurements were made with a linear filter setting of 1 Hz to 70 kHz. The output of the tweeter also was checked with a microphone, from which the electrical output was displayed on an oscilloscope to visually confirm the shape and duration of the stimulus and the number of peaks.

All amplified response signals were fed to an analog-to-digital converter (ADC) interfaced to a personal computer. Signal acquisition, averaging, and analysis were controlled by dedicated software developed in-house. Signals were recorded for 12.8 ms, using a sampling time per data point of 0.05 ms. Responses to 256 stimuli were averaged. During the recordings, the running average was displayed in real time. Results were stored for subsequent analysis using the dedicated software developed in-house.

For each ear, hearing was assessed by delivering click stimuli, beginning at 80 dB SPL and decreasing in intensity in steps of 10 dB until the threshold was reached. Threshold was defined as the intensity 5 dB above that at which no visually recognizable brain stem wave V was elicited. Two recordings were made for each set of stimulus variables to confirm reproducibility. The entire procedure was then repeated, frequency-by-frequency, using toneburst stimuli of 1, 2, 4, 8, 12, 16, 24, and 32 kHz to determine the threshold at each frequency. For all of the 80 dB SPL recordings (click stimuli and toneburst stimuli), peak latencies and interpeak latencies were measured. The latency of each wave was considered to be the time from stimulus onset to the maximal amplitude of that wave. The time between 2 peaks was the interpeak latency. Interpeak latencies were calculated for pairs I to III and I to V. Amplitudes of waves I and V were measured from apex to nadir. The wave amplitude ratio (A-ratio) was calculated by dividing wave V amplitude by wave I amplitude.

In each dog, a single ear was assessed during a session lasting about 2 hours. The second ear was tested 3 to 12 weeks later. (The results of these tests are discussed in Chapter 1.9.)

Industrial audiometers suitable for BEAR examinations are available, and are practicable for use in dogs and cats. However, their frequency limits are designed for testing the human ear, which is deficient at the higher frequencies that dogs (and presumably cats) can hear. In spite of this shortcoming, such

audiometers make the separate testing of each ear in dogs and cats feasible for many veterinarians.

1.4 Congenital diseases of the ear

The auricle and the external ear canal are derived from mesenchymal condensations from branchial arches 1 and 2. The middle ear cavity is formed as a lateral extension of the first pharyngeal pouch. The eustachian tube develops from an entodermal extension of the first pharyngeal pouch. The ossicles arise from branchial arches 1 and 2. The tympanic membrane is a three-layered structure consisting of an inner mucosal layer of entodermal origin, a middle layer of mesodermal origin, and an outer layer of ectodermal origin. The inner ear consists of a membranous and a bony labyrinth. The membranous labyrinth is derived from the ectoderm, whereas the bony labyrinth (otic capsule) is derived from the mesoderm and neural crest surrounding the primordial membranous labyrinth.[92]

1.4.1 Congenital deformity of the external ear

Congenital malformation of the auricle is rare in dogs and cats, or at least affected pups and kittens are rarely met in veterinary practice. It is usually not medically necessary to correct a defect of the auricle, and thus these difficult, esthetically unsatisfactory, and painful reconstructions are ethically undesirable. Veterinarians in the Netherlands do not encourage surgical intervention when the auricle is healthy and »ear cropping« without a medical indication is prohibited.

Congenital atresia of the ear canal is rarely detected because the malformation is not visible from the outside. In most cases it is the medial part of the ear canal that is undeveloped and a patent canal does not reach the tympanic membrane. The abnormality is usually unilateral and the resulting unilateral deafness is not always detected early in the animal's life. If atresia of the ear canal is found without clinical signs, it is advisable to refrain from intervention, but if there is an abscess of the middle ear, it requires drainage followed by antimicrobial therapy. Middle ear disease may recur and the owner should be advised to return the animal promptly if it shows signs of ear pain. Surgical reconstruction is not a justifiable option.[80]

1.4.2 Congenital deafness

Deafness that is detected soon after birth may be acquired or inherited and it may occur in any puppy or kitten, whether purebred or of mixed breeding. Acquired deafness may result from viral infections, severe malnutrition, or the ototoxic side effects of drugs or other chemicals. Because all dogs and cats are born deaf, deafness in a puppy or kitten is not abnormal up to a certain age. The earliest discriminating hearing tests reported in cats were performed at the age of 7 days. Measurements of cochlear potentials via an electrode in the round window were found to be conclusive about the presence or absence of hearing in cats over 7 days of age.[39] In 4 puppies (2 beagles, 2 wolfhounds) brain stem auditory evoked potentials were recorded from surface electrodes at the base of the auricles and on the midline over the parietal bone. The click stimulus was a rectangular wave 0.2 ms in duration at a repetition rate of 10 per second, delivered via two microphones (acting as speakers) at opposite ends of the cage. The intensity was 80 dB SPL and 90 dB SPL as measured at the center of the cage, in which the puppies were placed without sedation. For the recording of bone-conducted brain stem evoked potentials the puppies were taken out of the cage, leaving the electrodes in place. The same stimulus was used but was applied via a bone conductor pressed against the skull by

hand, maintaining the pressure that resulted in the best response signal. Testing was performed every other day from the 3rd to the 27th day after birth. Brain stem evoked potentials were first observed on the 7th day after birth in the 2 beagles and on the 11th day in the 2 wolfhounds, in both cases using the bone-conducted stimulus. Using air-conducted stimuli, the first brain stem evoked potentials were observed on the 25th day in the beagles and on the 27th day in the wolfhounds. While the number of puppies in this pilot study was small, the results indicate that, technically, puppies may be able to hear by 5 to 7 days after birth.[80]

Congenital deafness caused by a genetic defect has been thoroughly investigated for the white cat [9, 39, 51, 70] and the Dalmatian dog.[41] These investigations were in part motivated as animal model studies for the Waardenburg syndrome in humans, which is characterized by hereditary deafness associated with disorders of pigmentation of hair, skin, and the iris.[82] The Waardenburg syndrome in humans is now recognized to have 4 clinical types (WS1 to WS4), of which both WS1 and WS3 are caused by mutations in PAX3.[65] Congenital deafness associated with disorders of pigmentation has been described in the cat [9, 39] and in the dog.[2, 32, 41] In dogs deafness is associated with merle pigmentation (Shetland sheepdog, collie, harlequin Great Dane) and carriers of the piebald gene (bull terrier, Samoyed, Great Pyrenees mountain dog, Sealyham terrier, greyhound, bulldog, Dalmatian dog). In this form of inherited deafness, the animal is completely deaf in one or both ears.[80]

Most who have studied the histology of the development of the cochlea in deaf white cats have agreed that the primary degeneration of the epithelial and sensory elements occurs in the first week after birth, after which secondary degeneration of the neural structures follows.[9, 39, 70] Normal neural structures were found together with an advanced stage of degeneration of the epithelial elements in deaf white cats.[55] In a later study, however, it was reported that the spiral ganglion neurons in 2 of 11 white kittens (7 and 16 days old) were completely degenerated. It was concluded that the degeneration of the cochlea in white cats may be considered to be a process affecting both sensory and neural structures with a variety of features and variable timing.[51]

The earliest postnatal histological studies of the cochlea in deaf Dalmatian dogs were at the age of 4 weeks. At that age, a volume reduction of the saccule and the cochlear duct had already occurred.[41] This volume reduction is a consequence of the descent of Reissner's membrane toward the organ of Corti and, at a later stage, the covering of the organ of Corti by this membrane. Other signs of degeneration in the cochlear duct were a decrease in thickness of the stria vascularis and of the cellular components of the organ of Corti. In older deaf Dalmatian dogs the degeneration was more severe and was accompanied by loss of ganglion cells.[41] In a histological comparison of the cochleas from hearing and deaf Dalmatian dogs at the age of 6 weeks, Maïr's findings were confirmed for the deaf dogs, whereas the cochleas of the dogs with normal hearing were completely normal.[10] The relation between variation in pigmentation and inner ear dysfunction is recognized in several hereditary syndromes in several species of mammals, including humans. The pigmentation abnormalities are always of the »white spotting« kind, also known as hypopigmentation. When the entire coat is white, the animal must be regarded as having one very large spot rather than as being an albino.[80]

The difference between the two types of whiteness, albinism and hypopigmentation, is fundamental. Melanocytes can be identified in the hair follicles of albinos but they are incapable of forming melanin due to a biochemical block, whereas no melanocytes can be identified in hypopigmented animals or in spotted regions of pigmented animals.[7] There is no pig-

ment whatever in albinos, but in hypopigmented animals pigment is present in the retina, showing that the genetic capacity to produce melanin is not lacking.[15] The abnormalities in the inner ear are confined to the cochlea and saccule. There is severe degeneration of the organ of Corti, the stria vascularis, the spiral ganglion, and the macula of the saccule. The relation between hypopigmentation and inner ear abnormalities was investigated in mice.[15] The main conclusions were that »spotting« of the coat was always associated with hypopigmentation of the stria vascularis in the cochlea, usually a heavily pigmented area. Hypopigmentation of the stria vascularis was always associated with degeneration of the cochlea, but there was no correlation between the severity of this hypopigmentation and that of the coat.[15] These findings may also be applicable to the dog and the cat.

The relation between the piebald gene and »spotting« was established in mice,[42] and the abnormality was localized in the neural crest. The abnormal parts developed abnormal melanoblasts in the differential areas, resulting in local hypopigmentation. The abnormal areas varied in location and size, as did the pigmentation in the stria vascularis. The degeneration of the cochlea varied in severity within the cochlea. Hearing was not tested in these animals.

From these findings it may be concluded that if an animal with »spotting« or hypopigmentation is born with unilateral or bilateral deafness, the deafness is based on a hereditary defect. Furthermore, when hypopigmentation is a canine breed characteristic, deafness will occur in the breed.[80]

In a recent study, 20 of the genes already found to be the cause of sensorineural hearing impairment in humans or mice were considered as candidates for causing deafness in dogs, and chromosomes were assigned by FISH and RH mapping. The mapping data, which confirm the established conservation of synteny between canine and human chromosomes, provide a resource for further association studies in segregating canine populations and the basis for insights into this common canine and human disease.[54]

1.5 Inflammatory diseases of the ear

Inflammatory diseases of the ear in dogs and cats are usually painful when the external ear and the middle ear are involved. Labyrinthitis, however, is characterized by signs of severe vestibular dysfunction, usually unilateral, and unilateral deafness. Otitis externa is the most common ear disease in dogs, while in cats otitis media is the most common. As long as the tympanic membrane is intact, otitis externa does not result in middle ear disease. Otitis media can lead to labyrinthitis, but the frequently occurring vestibular signs associated with otitis media can also result from ototoxicity. They may be milder than in infectious labyrinthitis, and secondary meningitis does not occur in ototoxicity.

1.5.1 Primary and secondary skin diseases of the auricle

Dermatitis on the auricle can be part of generalized skin diseases and also have the same appearance.[59] Hypersensitivity—including atopy, food hypersensitivity, and contact hypersensitivity—can also cause dermatitis on the auricles. Atopy and food hypersensitivity cause generalized skin disease and hence the history and clinical signs are those of generalized dermatitis. Atopy has a high incidence in dogs and cats, and atopic dermatitis affects the auricle more often than does hypersensitivity. Erythema of the auricle is a common feature of atopic dermatitis. Chronic inflammation may eventually lead to secondary bacterial or yeast infections. Food hypersensitivity is the second

most common hypersensitive reaction. In the majority of patients, and these are mostly dogs, the auricles are also affected. It has been suggested that particularly in cocker spaniels and Labrador retrievers the auricles and the external ear canals may be the only sites of expression of food hypersensitivity.[59]

Contact hypersensitivity can be caused by medications used to treat external ear disease, either an active component such as an antibiotic or glucocorticoid or a vehicle such as propylene glycol. The history is most indicative of the cause when the existing dermatitis is worsened the first time the topical medication is used.[59] To confirm the suspicion, the topical medication should be stopped and replaced by a different type of medication. Dermatological olive oil is useful for this purpose.

Keratinization disorders. Dermatitis of the auricles can be caused by keratinization disorders of the skin, which alter the surface and appearance of the skin.[60] The epidermis of dogs and cats is replaced at a high rate, in spite of which it maintains its normal thickness, has a barely perceptible surface keratin layer, and loses its dead cells invisibly into the environment. If the delicate balance between cell death and renewal is altered, epidermal thickness changes, the stratum corneum becomes noticeable, and the normally invisible sloughed cells of the stratum corneum become obvious. There are numerous causes of keratinization defects affecting proliferation, differentiation, or desquamation of the epidermis, or some combination of these, and there can be accompanying alterations in epidermal formation and deposition.[60] Breeds prone to primary idiopathic seborrhea, e.g., West Highland white terriers, cocker and springer spaniels, Irish setters, and Doberman pinchers,[61] tend to have scaling on the auricles and secondary dermatitis. Endocrinopathies such as hypothyroidism and hyperestrogenism may result in altered keratinization and secondary dermatitis that also involves the auricles. In dogs and cats seborrhea is usually resolved by removal or correction of the cause. Regular bathing with shampoos is usually helpful, but not all of the components of antiseborrheic shampoos for dogs are harmless to cats, and adequate information about the contents of a shampoo and its safety for cats should be obtained before it is used.

Parasites causing dermatitis in dogs and cats may also affect the auricles. The primary locations of canine scabies (*Sarcoptes canis*) are the ears, head, ventral abdomen, and distal aspects of the extremities. The lesions are papules, crusts, and scales and the principal clinical sign is extreme purities. The sarcoptic mange mite of cats (*Notoedres cati*) infests the auricles, face, and neck and causes alopecia, crusts, and secondarily infected excoriations.[89] Ivermectin is used to treat both cats and dogs, with the exception of collies, Shetland sheepdogs, and collie-related breeds, in which it has adverse effects. *Cheyletiella yasguri* in dogs and *Cheyletiella blakei* in cats may cause dandruff and hair loss, as well as mild to severe pruritus in cats, involving the auricles and other sites. The diagnosis is made by demonstration of the mites (Figure 1.14 a–c). Treatment consists of spraying or bathing with parasiticidal shampoos containing permetrin.[89] Demodicosis may affect large areas of the skin, not excluding the auricles in both dogs and cats.[62] Fly bites may be common during the fly season in certain areas. They produce pinpoint ulcers rapidly covered by black-red crusts on the auricles.[59]

Leishmaniasis is a protozoan infection that may include nonpruritic exfoliative dermatitis and dermatitis of the auricles among its clinical signs. In addition to erythema, there may be scaling, crusts, ulceration, and necrosis of the skin of the auricles.[84, 90]

Immune-mediated disorders such as systemic lupus erythematosus may affect the auricles of both cats and dogs.[64] Alopecia, crusting, depigmentation, and necrosis of the margins of the auricles are described among the multiple clinical signs. Pemphigus vulgaris may cause large bullae on the inner side of the auricle, and pemphigus foliaceus may cause dermatitis of the auricles with thickening of the skin at the margins.[85]

Sarcoidosis is a systemic disease of unknown cause, characterized by noncaseated epithelioid granulomas. In dogs nodules are found on the bridge of the nose and on the auricles. The disease is described in Chapter 2.5: Nasal sarcoidosis.

Alopecia of the auricles is observed most often in dachshunds, but also occurs in Chihuahuas, Boston terriers, whippets, and Italian greyhounds. It is important to differentiate this spontaneous alopecia from dermatosis, for it requires no treatment. It is a slowly progressive but benign problem and the remainder of the coat remains normal.[63, 86]

1.5.2 Perichondritis and chondritis of the auricle

Perichondritis or chondritis is a bacterial infection of the auricular perichondrium or cartilage, respectively. It may follow inadequately treated auricular cellulitis, acute otitis externa, or accidental or surgical trauma. The affected auricle is extremely painful, red, and swollen, with serous or purulent exudate. The most common pathogen is *Pseudomonas*. In the early stage in humans this condition is treated with systemic antibiotics such as ciprofloxacin,[34] but in dogs it is usually presented as a chronic disorder and there is so much painful perichondritis, chondritis, and cartilaginous ossification that surgical removal of the auricle or both the auricle and the external ear canal is necessary. Although radical, this treatment is effective. Before, during, and long after surgery, the systemic administration of antibiotics selected by repeated sensitivity tests will help to prevent spreading of the infection.

Polychondritis. Relapsing polychondritis of the auricular cartilage is a recognized autoimmune

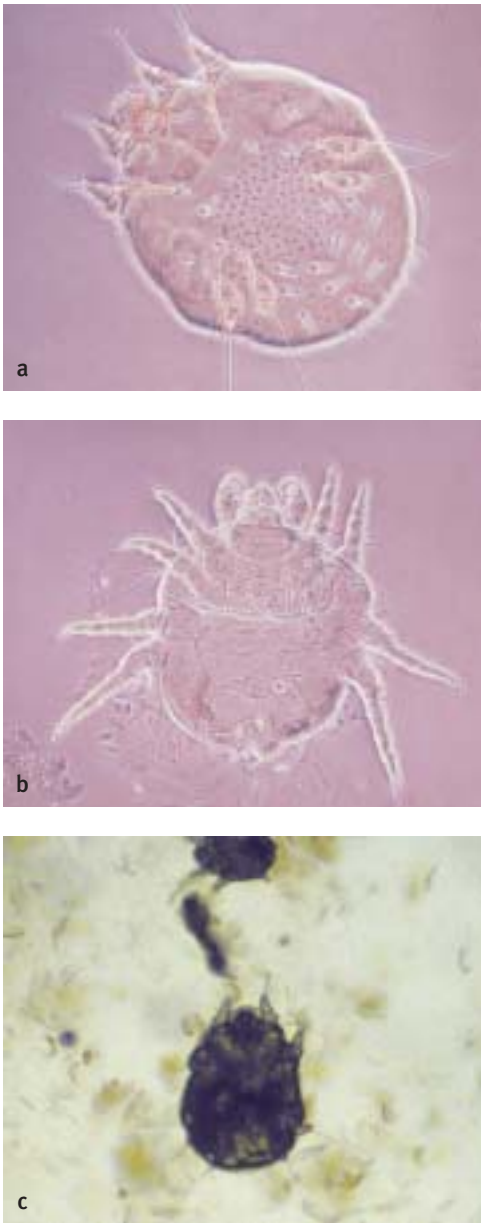


Figure 1.14 a–c: Three mites that cause dermatitis in dogs and cats, which may also involve the auricles. (a) Sarcoptes canis, (b) Cheiletiella, (c) Notoedres cati. (Courtesy of Dr. M.A. Wisselink, Division of Dermatology, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University).

disease in cats.[26, 87] As in humans, it is manifested by episodes of inflammation of cartilages throughout the body, but in cats these occur most prominently in the auricles. Affected cats are presented with swollen, erythematous, curled, and deformed auricles which are not apparently very painful but are sensitive to palpation. The disease can be unilateral but is usually bilateral. The cat is otherwise healthy. Histological examination reveals lymphoplasmacytic inflammation of the cartilage. The cat is usually FELV- or FIV-positive.[87] No curative therapy is known but topical treatment with a glucocorticoid may help to reduce the pain and swelling. Spontaneous recovery does occur, but the shape of the auricles may be permanently changed.

1.5.3 Cold agglutination and cutaneous vasculitis of the auricle

This disease is a type II autoimmune disorder in which cold-reacting (usually IgM) erythrocyte autoantibodies are formed. The lesions on the auricle, nasal plane, digits, and tail consist of erythema, macular purpura, necrosis, and ulceration. To differentiate cold agglutination from cutaneous vasculitis, it is necessary to obtain a thorough history, focusing on constitutional signs, previous infections, lymphoreticular neoplasms, frostbite, systemic lupus erythematosus, lead poisoning, and drug allergy. The histopathology may differentiate cold agglutination from vasculitis.[88]

1.5.4 Inflammatory diseases of the external ear canal

The base of the auricle and the external ear canal consist of a funnel-shaped fibroelastic cartilage covered by closely attached subcutaneous tissue and skin. The skin of the base of the auricle and the ear canal contains hair follicles and sebaceous and apocrine glands. The skin of the bony part of the ear canal is much thinner than that of the cartilaginous portion and is continuous with the epithelial layer of the tympanic membrane. There are no glands or hair follicles in the subcutaneous layer. Because of its thinness, the skin in the bony ear canal is more sensitive to trauma than that in the cartilaginous portion. The sensory innervation of the external ear is provided by the trigeminal, glossopharyngeal, and vagus nerves and the motor innervation is from the facial nerve.

Cerumen and inflammatory diseases. Cerumen is a combination of the secretions of sebaceous (lipid-producing) and apocrine (ceruminous) glands admixed with desquamated epithelial debris. It forms an acidic coating that aids in preventing external ear infection. Canine immunoglobulins A, G, and M have been identified in canine cerumen; IgG being the predominant immunoglobulin in both normal and inflamed ears.[31, 59] The external ear canal has a clearing mechanism in which masticatory muscle activity plays a role. The composition and quantity of the cerumen are important factors in the clearance of cerumen, dirt, and debris from the canal. Some dogs and cats have a scanty amount of cerumen and others form obstructive masses of it. The long and narrow ear canal in dogs and cats cannot be inspected without an otoscope and this may contribute to delayed recognition of accumulated cerumen. Irritation of the skin of the ear canal may alter the composition of the cerumen and when it becomes thick and sticky, the cleaning mechanism fails to function adequately. Inflammation may follow as a result of an increase in the microflora in the ear canal, in which bacteria and yeast are normally present. Trauma to the skin—caused by mechanical cleaning techniques and scratching—will almost certainly lead to otitis externa. Otitis externa has many causes in the dog and cat, but the timely recog-

nition of obstructive cerumen and its removal by flushing of the ear canal will eliminate one of the causes.

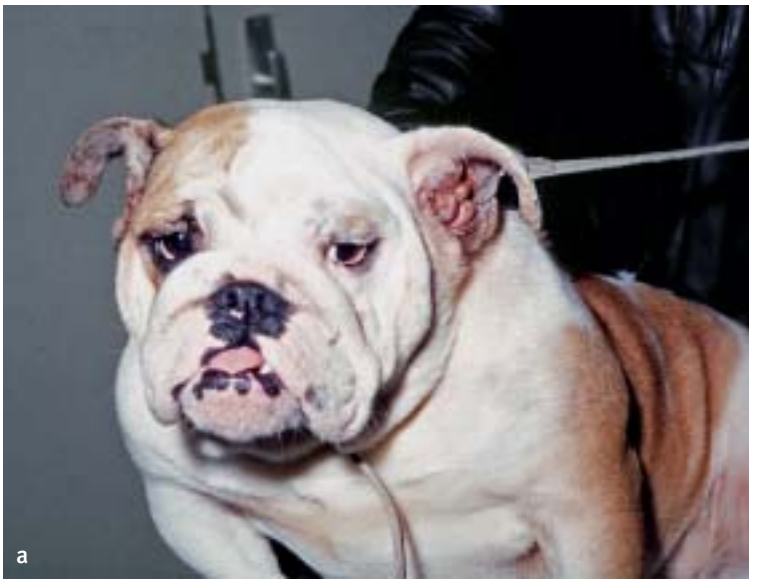
Acute otitis externa caused by foreign bodies in the external ear canal is most common in dogs. Plant parts are regularly found in the ear canal, especially during the season when certain grasses produce their seeds that have awns. Their barbs aid attachment to and movement through the dog's coat. They are found in the ear canal with their free filament directed outwards and the seed end inwards, often near the tympanic membrane.

The clinical signs of a foreign body in the external ear canal are acute irritation and pain, shaking of the ears, and rubbing the affected ear over the ground. Otoscopic examination easily reveals the grass awn in this early state. By use of a foreign body forceps through the otoscope, the grass awn can be removed under visual guidance. The dog should be sedated if it is restless and the ear is painful. If the grass awn or other foreign body is not removed promptly, inflammation narrows the canal and exudate obscures vision, delaying diagnosis. Cleaning of the canal by flushing may be sufficient to reveal the foreign body so that it can be removed, but it is usually necessary to first reduce the swelling and the overgrowth of bacteria. Daily administration for one or two weeks of a soft ointment containing a glucocorticoid and a broad-spectrum antibiotic, followed by again flushing the canal and repeating otoscopic inspection, almost always results in recognition and removal of the foreign body.

Acute otitis externa caused by ear mites. *Otodectes cynotis* causes acute inflammation of the external ear canal in both dogs and cats. The clinical signs are rubbing and scratching of one or often both ears, especially during the animal's resting period. During this period the temperature in the ear canal rises and the mites begin moving. Otoscopic examination reveals the mites, especially as the light of the otoscope begins to warm the inside of the ear canal. The presence of the mites especially activates the ceruminous glands and the resulting dark brown cerumen is an ideal background against which to observe the movement of the white, spider-like parasites. Treatment consists of flushing the external ear canal to remove the abundant cerumen and administering antiparasitic medication within the ear canal or systemically. Before antiparasitic medication is administered in the ear canal, the tympanic membrane should be inspected with care to ascertain that it is intact, because antiparasitic drugs are ototoxic if they gain entrance to the middle ear and consequently, the inner ear (Chapter 1.8). Ear mite infestation is contagious among dogs and cats, so all contact dogs and cats should also be examined with an otoscope.

If an ear mite infestation remains undetected, the skin of the ear canal undergoes proliferative inflammation, sometimes with overproduction of dark brown cerumen and sometimes with thin, moist detritus. The former is probably a hypersensitivity reaction[59] and can be treated by flushing, antiparasitic treatment, and use of soft ointment containing a glucocorticoid and a broad-spectrum antibiotic. The moist detritus, on the other hand, is caused by overgrowth of bacteria. When there is fluid in the external ear canal the mites will have left. The treatment is then simply flushing followed by use of a soft ointment containing a glucocorticoid and a broad-spectrum antibiotic, as described for bacterial otitis externa.

Acute diffuse bacterial otitis externa in dogs and cats is usually the result of removal of the protecting lipid film from the canal, which allows bacteria to invade the skin. This lipid film can be removed by mechanical cleaning of the ear canals (usually by the owners) or by using inappropriate ear drops. The use of a cotton swab or other objects to clean the canal causes abrasion of the skin, and the application of



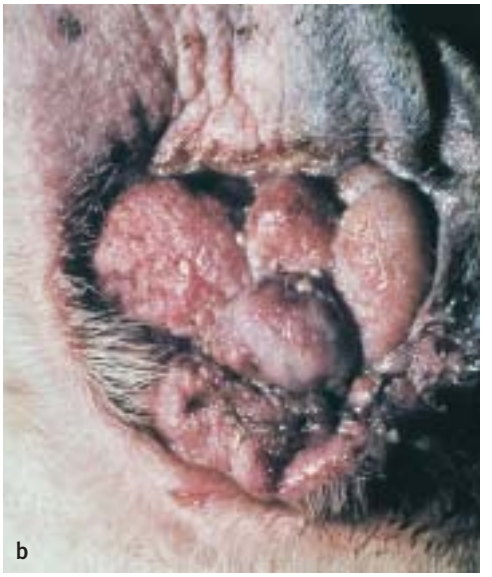


Figure 1.15 a, b: English bulldog, 9 months old, with chronic bilateral proliferative otitis externa (a). The bulging deformities at the base of the auricle continue inside the external ear canal (b).

drops can result in maceration. Other predisposing factors include frequent swimming, a warm and humid climate, a narrow and hairy ear canal, trauma, undetected foreign bodies, and undetected mite infestation. The usual pathogens associated with acute diffuse bacterial otitis externa are *Pseudomonas aeruginosa, Proteus mirabilus,* and *Staphylococcus aureus*. There is a remarkable similarity with disease in the human external ear canal.[34]

Treatment consists of flushing the ear canal when there is purulent material and debris. Both dogs and cats shake the head vigorously after ear flushing, which is very effective in drying the entire canal without trauma. This reaction is missing when the ears are flushed under sedation or anesthesia, which is why this should be avoided (Chapter 1.3). After the ears are flushed and are dried by head shaking, they are treated with a soft ointment containing a broad-spectrum antibiotic, or specific antibiotics if the pathogens are known from previous culture, and a glucocorticoid for its anti-inflammatory effects, which also reduce the pain. The long, narrow ear canals of dogs and cats need to be completely filled by the ointment to insure contact of the medication with all of the skin of the external ear canal. A good ointment consists of 2.5 mg neomycin and 1.0 mg triamcinolone acetonide per ml in a base of petroleum jelly (Vaseline) and paraffin. If this ointment is soft enough it can be injected into the ear canal with a soft-tip plastic injector.

Chronic otitis externa is a low-grade diffuse infection and inflammation of the external ear canal that persists for months or years. It is characterized by pruritus, dryness, and hypertrophy of the skin of the external ear canal and base of the auricle. It results in thickening of the skin of the external ear canal and thus, progressive narrowing of the lumen. Although bacterial or fungal infection is the main cause of chronic otitis, it can also result from such conditions as seborrheic dermatitis and sensitization to otic medication.[34] An antibiotic and a glucocorticoid in a soft ointment will reduce the inflammation. This should be administered once daily until the ear canal becomes wider, and then it can be given every other day and then once weekly. The owner is advised not to clean the ears; instead, flushing by the veterinarian should be performed regularly every 2 weeks until the ear canal widens, then once every 4 to 6 weeks when the ear canal has become wider and the

skin thinner and softer. Not all ears will recover completely and sometimes treatment with ointment once weekly and continuing periodic examination and flushing by the veterinarian may be necessary to prevent recurrence of the disease. In these cases, the skin continues to be slightly dry and hypertrophic and the cerumen continues to be sticky, leaving the skin less protected and more sensitive to bacterial invasion.

Chronic proliferative otitis externa occurs in both dogs and cats. In breeds which are predisposed to this disorder it is often bilateral (Figure 1.15 a, b) and may begin at a very young age, from less than one year up to four years of age. The proliferative changes develop in a few weeks or months, progressing to bulging deformities at the base of the auricle and inside the external ear canal. The ears are painful and although the animal shakes its head, it avoids being touched. The entrance to the ear canal is difficult to find and the lumen is closed by the proliferation. Otoscopic examination is therefore difficult and often ineffective. There is often gray to yellow cerumen that is foul-smelling and sticky. The external ear canal is hard, thickened to 2 to 4 times its normal diameter, and cannot be compressed. Treatment with soft ointment containing an antibiotic and a glucocorticoid is only possible if the lumen can be found with an otoscope, but if it can be found, treatment with abundant amounts of the ointment may be tried for 2 to 3 weeks. The ointment is applied with an injector into the small lumen of the canal and the crevices between the proliferations. If this treatment results in some shrinking of the proliferations, it is worth continuing, for there may be a chance of preserving hearing. However, in the majority of cases removal of the entire ear canal and the base of the auricle is the only solution. Pathological examination of 58 surgically-resected external ear canals from dogs and 27 from cats formed the basis of an analysis of the many changes occurring in the external ear canal in this disease.[75] It was observed that otitis externa often begins with hyperplasia—acanthosis and hyperkeratinization—of the squamous epithelium (Figure 1.16). Below the epithelium there is an infiltration of lymphocytes, plasma cells, polymorphonuclear leukocytes, and macrophages. There

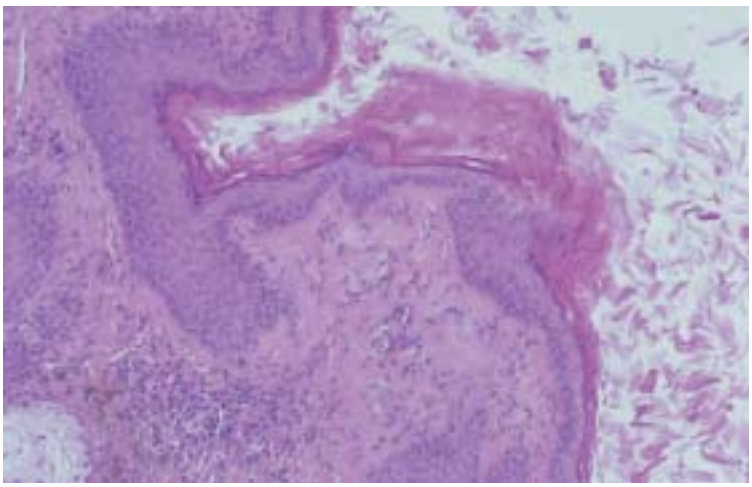


Figure 1.16:
Otitis externa in a dog: hyperplasia of the squamous epithelium, with acanthosis, hyperkeratosis, and inflammation. Plasma cells, lymphocytes, and macrophages containing ceroid pigment are present in the subepithelial connective tissue (H.E. x 10). (Courtesy of Dr. I. van der Gaag, Division of Pathology, Department of Pathobiology, Faculty of Veterinary Medicine, Utrecht University).

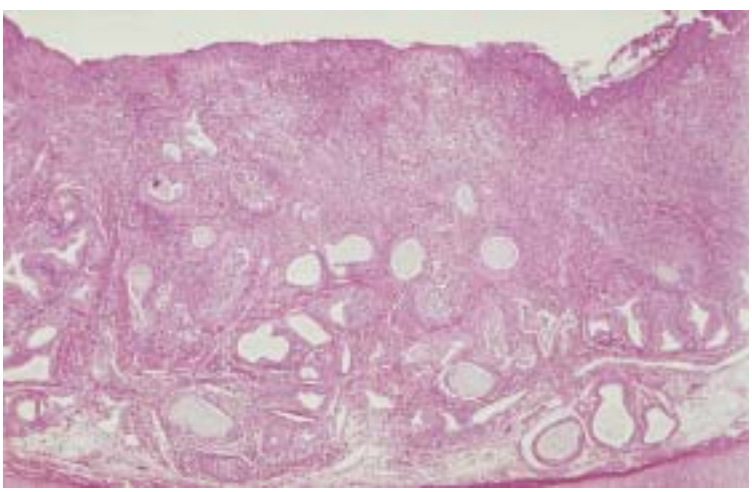


Figure 1.17:
Otitis externa in a dog: hyperplasia of the ceruminous glands, with cystic glands, dilated ducts, and erosions of the epithelium (H.E. x 2.5). (Courtesy of Dr. I. van der Gaag, Division Pathology, Department of Pathobiology, Faculty of Veterinary Medicine, Utrecht University).

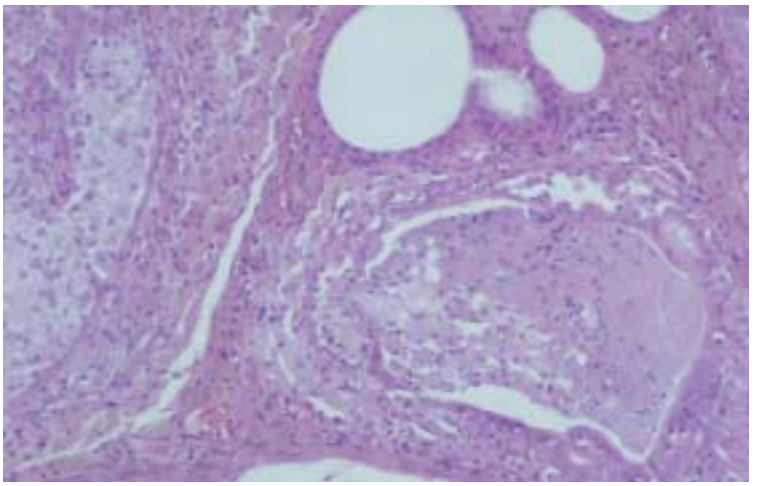


Figure 1.18: Otitis externa in a dog: a dilated duct filled with eosinophilic material, polymorphonuclear cells, lymphocytes, and macrophages and surrounded by round cell inflammation and macrophages with ceroid pigment (H.E. x 10). (Courtesy of Dr. I. van der Gaag, Division Pathology, Department of Pathobiology, Faculty of Veterinary Medicine, Utrecht University).

is often a large amount of ceroid pigment and there may be proliferation of fibrous tissue. In the early stage of otitis the sebaceous glands become hyperplastic, with dilated ducts. Later, the ceruminous glands also become hyperplastic, with cystic dilatation of the glands and ducts, and they can almost completely displace the sebaceous glands (Figure 1.17). The ceruminous glands and ducts are often filled with eosinophilic material combined with polymorphonuclear leucocytes, lymphocytes, and macrophages (Figure 1.18). There may be papillary proliferation of the cyst and duct epithelium, as well as proliferation of the myoepithelium. The proliferating tissues and dilated glands and ducts can more or less obliterate the external ear canal. Erosions and ulcers occur and in some dogs there is ossification of the external ear canal, even extending into the cartilage. In cats, circumscribed granulomatous inflammation can be found and the hyperplasia of ceruminous glands is particularly striking, but in general the histological features of otitis are very similar to those in dogs.[75] Similar findings were described in a study in which ear canals of dogs with external otitis were compared with those of normal dogs.[68]

Technique for resection of the entire ear canal. Our technique for resection of the entire ear canal and the affected part of the base of the auricle—without bulla osteotomy—has been described for the dog[76] and in less detail for the cat.[78] This surgical procedure is unusually painful if the anesthesia is inadequate and hence there should be continuous monitoring of the ECG, the pulse, and the adequacy of analgesia by an experienced anesthesiologist who is able to anticipate changes in ventilation and heart action.

With the animal in lateral recumbency, two incisions are made in the skin over the vertical part of the ear canal, one extending from the intertragic notch to the ventral limit of the vertical part of the ear canal (determined by palpation), and the second from the trachohelicine notch to the same ventral point. The resulting triangular flap of skin is dissected free to its base at the entrance of the external ear canal. The vertical part of the ear canal is then freed from the subcutaneous tissue and muscles. This dissection should proceed along the interface between the cartilage and surrounding tissue, i.e., on the surface of the cartilage itself, to avoid unnecessary hemorrhage. The cartilage and the skin of the medial wall of the ear canal are separated from the cartilage and the skin on the concave side of the base of the auricle using strong scissors; all proliferations being included with the ear canal. The horizontal part of the cartilaginous external ear canal is then also separated from the surrounding tissues by dissecting close to the cartilage. Appropriate care must be taken to avoid the facial nerve, which leaves the stylomastoid foramen and turns rostrally around the horizontal part of the external ear canal. In some cases the nerve is attached to the cartilage and great care must be taken to separate it. When the horizontal part of the cartilaginous external ear canal has been freed from the surrounding tissues, its attachment to the osseous external acoustic meatus is easily palpated during slight movements of the cartilaginous part of the ear canal. The cartilaginous part is then removed from the osseous part with

pointed scissors. The facial nerve must be protected during this procedure. Removal of the cartilaginous part of the ear canal is followed by a very important and time-consuming part of the operation, namely, the complete removal of all of the skin that lines the osseous external ear canal. This is best accomplished with a small curette. When the lining is being removed, the tympanic membrane is removed with it to complete the removal of all components of the external ear canal.

The next step is the remodeling of the auricle, the cosmetic objective being to make it fold over naturally. The caudal part of the auricle is folded forward, using the most natural folding point at the base of the auricle as the point of rotation. The most caudal part of the base of the auricle is then sutured to the rostral part of the base, leaving the ends of the sutures long and lying inside the folded auricle. A Penrose drain is placed between the osseous external meatus and the ventral part of the skin incision, leaving 1 cm protruding. The subcutaneous tissues and the skin margins of the initial triangular incision are then sutured together. The drain is left in place for 5 days, an analgesic is given for 5 days, and an antibiotic is given for at least 2 weeks. In a follow-up study of 78 ears of 50 dogs in which the above surgery was performed, 5 dogs had a partial facial paralysis and 5 had fistulas which could not always be resolved by long-term antibiotic treatment.

Chronic bacterial otitis externa is usually associated with a refractory *Pseudomonas* infection of the external ear canal. It is characterized by pain and purulent discharge from the severely inflamed canal. The infection causes lesions in the skin of the canal, and the entire skin, subcutis, and eventually even the perichondrium and cartilage can be affected. Treatment initiated in the early stages is often successful if the owner agrees to a long-term program of management. The program undertaken by the veterinarian consists of culturing the exudate, flushing the ear canal, and examining the tympanic membrane. The owner is shown how to how to fill the ear canal with soft ointment containing an antibiotic usually effective against *Pseudomonas* and a glucocorticoid for its anti-inflammatory and analgesic effects. The owner applies the ointment once daily for one week, then on alternate days for one week, and then the veterinarian reexamines and flushes the ear canal. If the response is questionable, bacterial culture and sensitivity testing are repeated and the antibiotic is changed if necessary according to the results. The owner applies the ointment twice weekly during the following three weeks and then the veterinarian reexamines and flushes the external ear canal. If the improvement is satisfactory, the frequency of the reexamination and flushing can be reduced to once in 6 weeks and application of the ointment can be reduced to once weekly. It is sometimes necessary to continue once-weekly administration of the ointment for several months in order keep the inflammation under control. If the infection is refractory, repeated culture and sensitivity testing are used to guide the choice of antibiotic, but following this program of treatment, most infections can be brought under control satisfactorily.

Complications of otitis externa. A serious complication of external otitis is the presence of a ruptured tympanic membrane, for it allows extension of the *Pseudomonas* infection into the middle ear. It necessitates flushing of the middle ear, under anesthesia, and systemic antibiotic treatment (see *Otitis media*). External otitis can also be complicated by ossification of the external ear canal, together with perichondritis and chondritis. This is very painful and difficult to cure without removal of the entire external ear canal and long-term systemic antibiotic treatment. A highly unusual complication of chronic bacterial infection is a secondary *Aspergillus* infection. The best results

have been obtained with systemic administration of fluconazole or a derivate for 6 to 8 weeks.

1.5.5 Inflammation of the tympanic membrane

The tympanic membrane may become inflamed together with the external ear canal. The first sign of this is loss of transparency and thickening of the tympanic membrane, sometimes with vascularization of the proliferating external epithelial layer (Figure 1.19). No change is required in the treatment of the otitis externa, but the tympanic membrane may not regain its transparency until long after resolution of the otitis externa.

In acute inflammation of the middle ear both the mucosal side of the tympanic membrane and the mucosa of the entire middle ear become deep red due to hyperemia and hypervascularization. These changes are resolved by treatment of the middle ear inflammation. Purulent inflammation of the middle ear may cause rupture of the tympanic membrane. The clinical signs are pain and a sudden, massive purulent discharge from the external ear canal. Material should be taken for culture and systemic treatment with a broad-spectrum antibiotic should be started. An affected cat should be kept in the house for at least 3 weeks and a dog should be kept essentially at rest for 4 weeks. The initial antibiotic is changed, if necessary, according to the results of the culture and sensitivity testing. After 4 weeks of antibiotic therapy, otoscopic examination is repeated to determine whether further treatment is necessary. The ruptured tympanic membrane will heal when the middle ear infection is resolved.

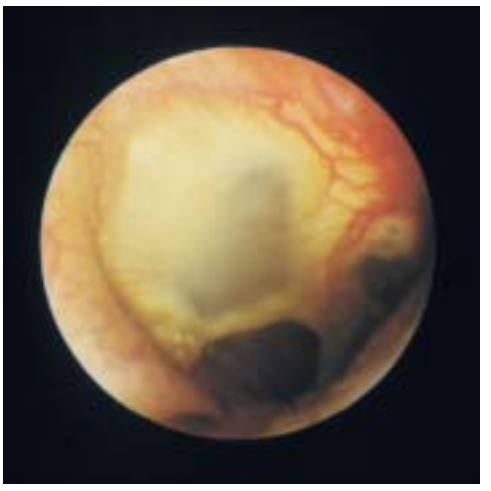


Figure 1.19: Chronic inflammation of the tympanic membrane in a dog, with a persistent lesion ventrally associated with chronic middle ear disease and chronic otitis externa. The loss of transparency and thickening, and the vascularization of the proliferating external epithelial layer are evidence of longstanding inflammation. Note the progression of vascularization from the surrounding epithelium of the external ear canal to the tympanic membrane.

1.5.6 Inflammatory disease of the middle ear

Otitis media denotes inflammation of the middle ear without reference to the cause. It may occur with or without effusion, which may be serous, mucoid, or purulent. In humans, otitis media with effusion is classified as acute (< 3 weeks), subacute (3 to 12 weeks), or chronic (> 12 weeks).[30]

Otitis media with or without effusion is more common in cats than in dogs. In both animals the history usually provides an accurate indication of the onset of pain in one ear, which is demonstrated by a slight head tilt with the affected ear down, and soft and cautious scratching and rubbing of the ear and shaking of the head. When the pain does not disappear spontaneously, veterinary consultation is sought. Otoscopic examination reveals no inflammation of the external ear canal but there is redness of the tympanic membrane. It is difficult to recognize effusion in this situation.

Numerous factors may contribute to the development of otitis media, but in humans dysfunction of the eustachian tube is considered to be the major etiologic factor.[30] Obstruction of the eustachian tube may hamper its functions of ventilating the middle ear, equalizing the air pressure between the middle ear and the nasopharynx, and draining middle ear secretions, all of which have direct consequences for functioning of the middle ear

mucosa. The major cause of dysfunction and obstruction of the eustachian tube may be swelling of the mucosa at its nasopharyngeal end, which is common in nasal and nasopharyngeal infections. That these so often occur in viral upper respiratory diseases in the cat may explain the rather common occurrence of otitis media in this animal.

When otitis media is diagnosed and the tympanic membrane is found to be intact, a broad-spectrum antibiotic, such as amoxicillin with clavulinic acid, should be given for 4 weeks. Otoscopic reexamination after 4 weeks will usually reveal a normal tympanic membrane. If there is still some redness the therapy should be continued for 2 more weeks. The clinical signs indicating pain will diminish within 2 weeks, but otoscopic evaluation of the tympanic membrane is the best indication of the state of the middle ear mucosa. When the tympanic membrane is ruptured in otitis media, purulent exudate is found in the ear canal and the membrane is not visible. The animal must be anesthetized so that under otoscopic control the pus can be removed by suction and a sample can be collected for culture. When the rupture in the tympanic membrane is located, the middle ear is gently flushed with physiological saline solution at body temperature, through a small cannula with the tip just inside the middle ear. A separate cannula in the external ear canal is used to aspirate the fluid, also under otoscopic control. Flushing and aspiration are repeated until the fluid returned from the middle ear is clear. Systemic broad-spectrum antibiotic treatment is given for 4 weeks, and if necessary, changed according to the results of the culture and sensitivity testing. After 4 weeks otoscopy usually reveals the disappearance of signs of otitis media and healing of the tympanic membrane, although the membrane may not yet be transparent. If the tympanic membrane is still reddened, the therapy should be continued for 2 more weeks.

Horner's syndrome can occur as a complication of otitis media as a result of acquired dysfunction of the sympathetic nerves that course through the middle ear cavity. The signs are miosis and retraction of the globe, which causes protrusion of the third eyelid and narrowing of the palpebral fissure. The syndrome disappears spontaneously in about 3 months.

Labyrinthitis can also develop as a complication, due either to bacterial toxins from the middle ear that pass into the labyrinth and affect the hair cells of the organ of Corti and the vestibular organs, or to extension of the infection itself into the labyrinth. The treatment of otitis media is not influenced by the presence of vestibular signs, but the prognosis for disappearance of the signs is guarded.

Chronic otitis media constitutes a structural change in the middle ear with a permanent defect in the tympanic membrane[72] Usually, but not always, mucosal changes of the middle ear are the cause of the chronic inflammation. The bony structures surrounding the middle ear cavity may also be involved. The possible causes of chronic otitis media are unresolved acute otitis media, detritus in the middle ear, a foreign body passing through the tympanic membrane, secondary fungal infection, ciliary dysfunction, a polyp arising in the middle ear mucosa, and development of a cholesteatoma. The history and clinical signs indicate long-standing ear disease with constant irritation and intermittent discharge. Otoscopic examination usually reveals that the skin of the external ear canal is swollen in the area of the bony external ear canal, and some discharge, usually purulent, obstructs further examination. If no tympanic membrane can be observed after flushing, further diagnosis is necessary, either by inspection and flushing of the middle ear under anesthesia, or by use of imaging techniques to evaluate the nature and extent of changes in the temporal bone.

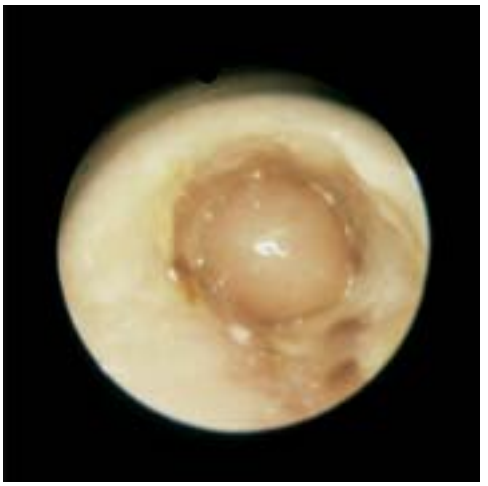
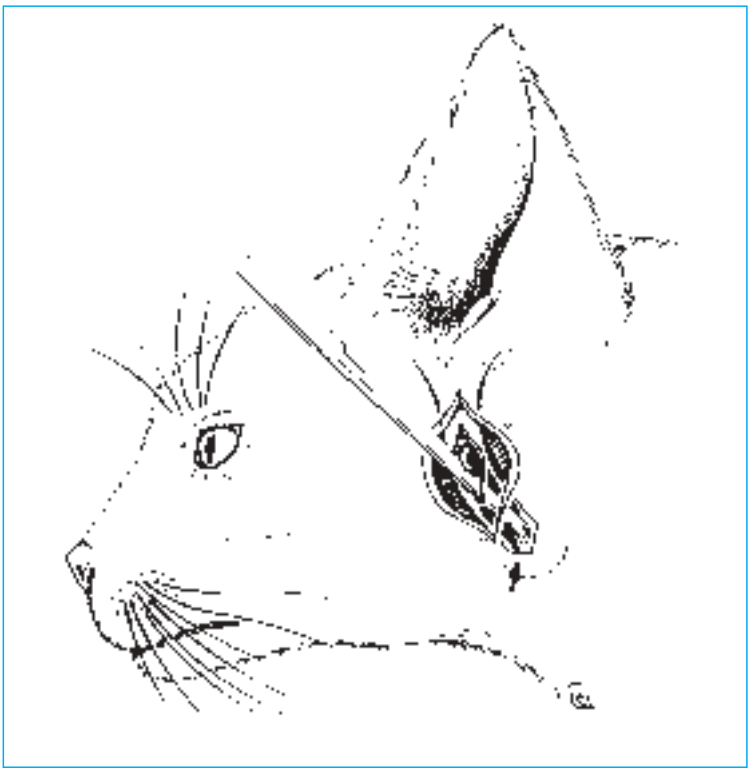


Figure 1.20: Otoscopic view of a middle ear polyp in a cat, which has protruded through the tympanic membrane into the external ear canal.

Figure 1.21: Through an incision in the external ear canal, the middle ear polyp is grasped with small forceps as close as possible to the osseus meatus, and is removed by a quick tug. The arrow indicates the stalk of the polyp. (From: Venker-van Haagen AJ. Diseases and surgery of the ear. In: Sherding RG, editor. The Cat: Diseases and Clinical Management. New York: Churchill Livingstone, copyright 1994: 1999–2009, Fig. 59-6, with permission from Elsevier).

Chronic otitis media in the cat. The most common cause is the development of one or more polyps in the mucosa of the middle ear. If a polyp expands within the middle ear and protrudes through the tympanic membrane, it will be found during otoscopic examination—after flushing of the external ear canal—as a round mass in the bony ear canal (Figure 1.20). Sometimes a polyp is of such size that it fills the entire external ear canal by the time it is diagnosed. It can be presumed to be a middle ear polyp if it can be easily moved with the tip of the cone of the otoscope and is not attached to the wall. However, if there is doubt, cytological or histological examination of a biopsy will confirm its identity. Histologically a middle ear polyp consists of proliferated submucosal connective tissue that is edematous and has a variable inflammatory infiltrate of eosinophils, plasma cells, and lymphocytes.[57] A middle ear polyp can also extend through the eustachian tube to expand in the nasopharynx, where it usually develops into a mass that obstructs the choanae (Chapter 3). When one or several polyps remain confined to the middle ear, diagnosis may be delayed, resulting in considerable damage to the middle ear and the surrounding structures.[13]



A polyp found in the external ear canal should be removed. An incision is made in the skin over the vertical part of the external ear canal ventral to the external orifice. The subcutaneous tissue and the parotid gland are dissected to free the cartilage of the vertical and horizontal parts of the ear canal, just dorsal and ventral to the junction between the auricular and the annular cartilages. After the facial nerve has been identified so that it will not be traumatized, a vertical incision is made in the external ear canal with a pointed scalpel. The polyp is then grasped with small forceps, as close as possible to the osseous meatus, traction is applied, and the polyp is removed by a quick tug (Figure 1.21). When the polyp has been removed »completely«, there will be a small stalk at its base (Figure 1.22). Hemorrhage following removal of the polyp is slight and stops spontaneously within a few minutes. The incision in the cartilage is closed by a continuous suture through the cartilage alone, using atraumatic absorbable material. The subcutis and skin are closed routinely. A broad-spectrum

antibiotic is administered for 3 weeks. If there is no persisting infection of the middle ear (a rare complication), the tympanic membrane will regrow in 3 to 4 weeks after surgery and will be found by otoscopic examination to be transparent after 4 weeks. In the rare cases in which the examination reveals reddish granulation tissue, indicating continuing middle ear inflammation, broad-spectrum antibiotic treatment is continued for 2 more weeks. In some cases another polyp develops and the procedure has to be repeated. If this occurs more than two times, a bulla osteotomy (see below) can be performed to remove the abnormally proliferating mucosa from the middle ear cavity.[78]

Figure 1.22:
The middle ear polyp, showing the indentation near the stalk where it was grasped by the forceps and the twisting of the stalk caused by rotation of the polyp before removal.

Unresolved otitis media is often due to detritus in the middle ear that causes continuous effusion and prevents healing of the tympanic membrane (Figure 1.19). Radiographs show a slight density in the middle ear and the wall of the tympanic bulla may be thickened. The approach to the middle ear should be via the external ear canal. Under anesthesia and otoscopic control, the middle ear is flushed with physiological saline solution at body temperature and the fluid is aspirated until all detritus is removed and the returning fluid is clear. It may be useful to save samples of the detritus for bacterial and fungal culture. When the tympanic membrane has the appearance of the opening of a fistula, with white edges (Figure 1.19), the edges should be removed in order to promote resumption of healing. After systemic antibiotic administration for 4 weeks, otoscopic examination should reveal an intact and almost transparent tympanic membrane. When the cultures reveal a specific bacterial or fungal pathogen, the systemic treatment is adjusted accordingly.

A density in the middle ear cavity can be a polyp, foreign body, cholesteatoma, or tumor. Of these, cholesteatomas and tumors cause the greatest damage to the bone surrounding the middle ear cavity. CT and MRI do not usually distinguish between malignant and benign masses, and hence surgical exploration of the middle ear is necessary. The bulla is usually approached ventrally. The animal is positioned in dorsal recumbency with the neck extended and the area between and around the mandibles and over the pharynx and larynx is prepared for surgery. A skin incision is made following the medial border of the rostral digastric muscle and extending caudally to a point lateral to the basihyoid bone. The rostral digastric muscle and the mylohyoid muscle lying medial to it are separated gently, taking care to avoid damage to the nerves of the pharyngeal plexus and the small vessels coursing to the mandibular lymph node. The bulla is found by tracing dorsally from the stylohyoid and tympanohyoid cartilages of the hyoid apparatus. The tissue ventral to the bulla is held aside with a blunt periosteum elevator. The bulla is opened with a hand-held trephine, making an opening sufficient for introduction of a suction cannula. The often whitish material and any fluid are removed, samples being saved for culture and histological examination as appropriate. Step-by-step, a large part of the middle ear cavity becomes visible, allowing a foreign body or polyp(s) to be removed or a biopsy of any apparent tumor to be obtained. A Penrose drain is then placed between the rostral digastric

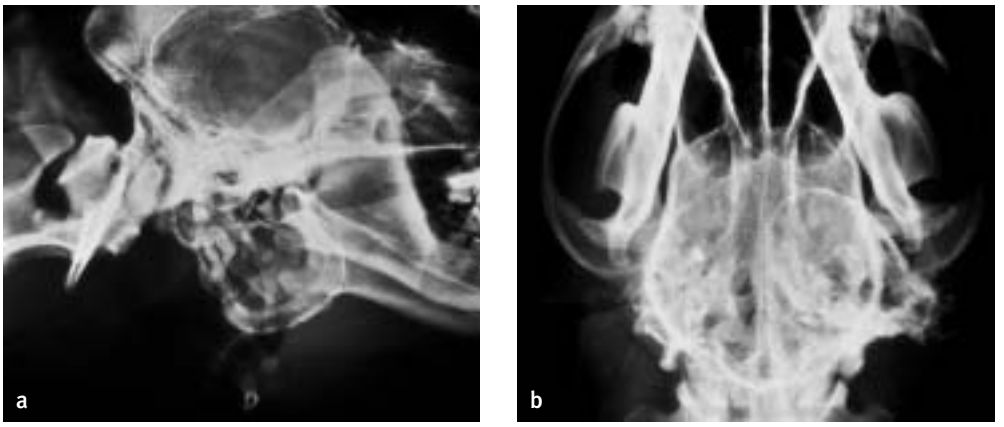


Figure 1.23 a, b: Radiographic changes caused by a cholesteatoma in the right middle ear of a 9-year-old dog. (a) Laterolateral radiograph, showing enlargement of the right tympanic bulla projected over the left tympanic bulla. (b) Dorsoventral radiograph, showing the circular bony ring over the right tympanic bulla.

muscle and the mylohyoid muscle, exiting outside the skin. The subcutis and skin are closed routinely. A broad-spectrum antibiotic is given for 3 weeks and otoscopic examination is repeated 4 weeks after surgery. In these more complex disorders there are no guidelines for the prognosis and the results of the diagnostic samples taken during the surgery will determine further management.

Cholesteatomas in the middle ear are masses composed of squamous epithelium originating from the external side of a ruptured tympanic membrane together with keratin formed by this tissue, and the annulus where the tympanic membrane attaches to the bony annulus.[11] Cholesteatomas in the middle ear contain no cholesterol crystals. In dogs they occur in chronic otitis media and result in considerable destruction of the surrounding bone via the pressure they exert as they expand, as well as chronic infection.[14, 36] They sometimes result in enlargement of the tympanic bulla (Figures 1.23 a, b and 1.24 a, b). Treatment consists of cleaning the middle ear cavity, removing the debris by suction through a ventral bulla osteotomy, and then 4 weeks of antibiotic treatment.

1.5.7 Labyrinthitis

Labyrinthitis can develop as a complication of chronic otitis media. It is recognized by acute and total loss of auditory and vestibular func-

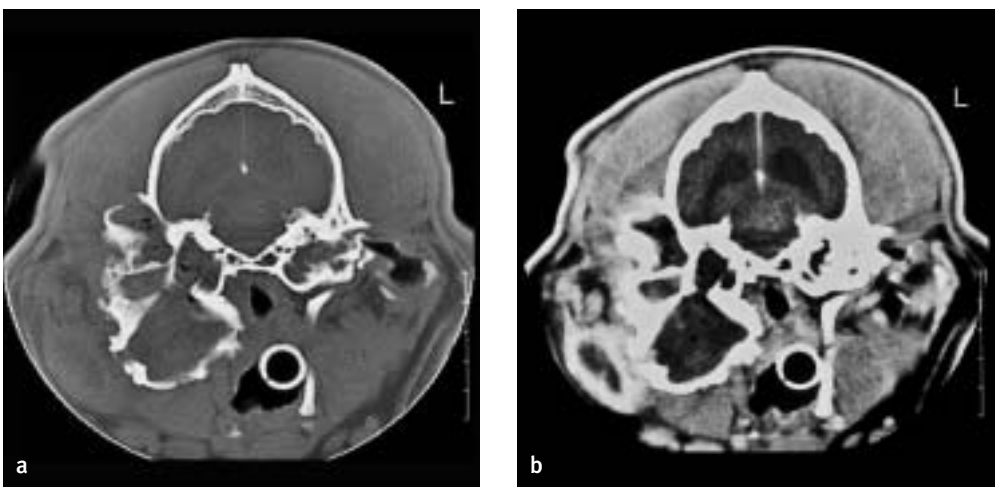


Figure 1.24 a, b: CT of the dog in Figure 1.23, showing (a) at bone settings, the right and left tympanic bullae do not contain air. There is a large mass with an irregular bony wall ventral, lateral, and dorsolateral to the right tympanic bulla and connected with the tympanic cavity. Small air bubbles can be seen in the soft tissue of the mass and in the tympanic bulla. The petrosal bone is involved in the process. (b) Contrast-enhanced CT, at soft tissue settings, reveals no enhancement of the contents of the tympanic bulla.

tion. It is a rare disease in dogs and cats and not easy to differentiate from ototoxicity clinically. Direct bacterial invasion of the labyrinth from chronic otitis media is the most likely cause. When the disease is recognized and otitis media is diagnosed no further diagnostic techniques are necessary. Although there is no possibility of reversing the clinical course, appropriate antibiotic treatment for 10 days is recommended to eradicate the labyrinthine infection and prevent propagation to the meninges.[29] When the bacterial infection has been suppressed, compensatory mechanisms may partly overcome the vestibular signs and hearing may not be completely lost.

1.6 Tumors of the ear

1.6.1 Malignant tumors of the auricle

Malignant tumors of the auricle include those involving the skin and those involving primarily the ceruminous glands. The most common malignant tumors of the auricle in the dog are sebaceous gland carcinoma, histiocytoma, and mast cell tumor, whereas those described in the cat are squamous cell carcinoma, basal cell carcinoma, hemangiosarcoma, and malignant melanoma.[59] Most of these tumors are presented as a proliferating, circumscribed mass on the auricle, but squamous cell carcinoma at the tip of the auricle often mimics granulomatous inflammation for months before irregular swelling and defects occur. Itching and epithelial erosions can be induced by the tumor, but are also often caused by scratching and shaking the head. Intermittent bleeding of the lesion is often the main problem mentioned in the history. Physical examination must include examination of the mandibular lymph nodes and the nodes at the base of the convex side of the auricle, which are normally too small to be palpated. If any of the nodes are enlarged, fine needle aspiration biopsies should be obtained for cytological examination. Depending on the character of the tumor, the auricle should be partially or totally removed. If the tumor lies near the edge of the auricle, it may be possible to simply excise a wedge that includes it. The opposing sides of the resulting triangle can then be sutured as illustrated for a tear in the auricle (Chapter 1.7). The skin is sutured on both sides of the cartilage, using interrupted sutures and beginning at the edge of the auricle. The cartilage is not sutured. Placing the first sutures at the edge avoids ending with a gap at the edge if the two sides are unequal. Absorbable sutures do not have to be removed.

Amputation of the entire auricle is often preferred for squamous cell carcinoma in the dog and the cat. The surgical procedure is easier in cats because vascularization of the auricle is less extensive and the auricle can be removed by simply cutting with scissors through both layers of the skin and the cartilage at one time. Bleeding is easily controlled by minimal use of thermocautery and ligation of one or two arteries. In dogs, however, the skin should first be carefully incised around the base of the auricle on the convex side so that all of the larger arteries and veins can be double ligated before the auricle is be removed. The suturing technique is similar in dogs and cats. The skin of the convex side of the auricle is sutured over the cartilage to the skin on the concave side. To avoid ending with unequal lengths of skin near the completion of the closure, 4 to 6 sutures are first used to divide the length of the wound into equal segments and then closure is completed with interrupted skin sutures between them (Figure 1.25 a–c). In dogs there is moderate swelling around the wound after surgery but in cats there is little or none. As in all ear surgery, administration of an analgesic for 5 days is indicated to reduce pain and prevent scratching. Since the surface of the tumor and the orifice of the ear canal and surrounding area cannot be disinfected adequately before surgery, antibiotic treatment for 10 days is advised.

1.6.2 Malignant tumors of the external ear canal

Malignant tumors of the external ear canal include, as described for the auricle, those involving the skin and those primarily involving the ceruminous glands. The most common malignant tumors involving the external ear canal in dogs are sebaceous gland carcinoma, histiocytoma, and mast cell tumor. In cats they are squamous cell carcinoma, basal cell carcinoma, hemangiosarcoma, and malignant melanoma; cats also have benign cystadenomas of the ceruminous glands.[59, 75] The history and clinical signs of a malignant tumor are those of a progressively enlarging lesion with a putrid exudate. The tumor gradually fills the ear canal and can be palpated by softly compressing the canal. Sometimes the circumference of the canal is increased by the growth of the tumor and sometimes only the tumor can be palpated because it has invaded through the wall of the ear canal into the surrounding area. Otoscopy is performed after flushing the ear canal and biopsies are obtained for histological examination to determine the tumor type. Fine needle aspiration biopsies are obtained from any enlarged lymph nodes, to search for metastases. Radiographs of the lungs are usually not indicated because tumors of the external ear canal rarely metastasize to the lungs. The treatment is complete surgical removal of the ear canal, including the tumor. If extension of the tumor beyond the external ear canal is suspected, imaging techniques can be used to reveal the extent of the process. CT is very helpful to evaluate local spreading of the tumor and radiographs of the thorax will clarify possible clinical signs of lung metastases. Tumors that extend beyond the external ear canal almost always recur after surgical removal, but those still contained within the ear canal are usually completely cured by surgery. The technique is similar to that for removing the ear canal in chronic proliferative otitis externa (Chapter 1.5). At present there is no satisfactory treatment, surgical or medical, for malignant tumors that have invaded beyond the external ear canal.

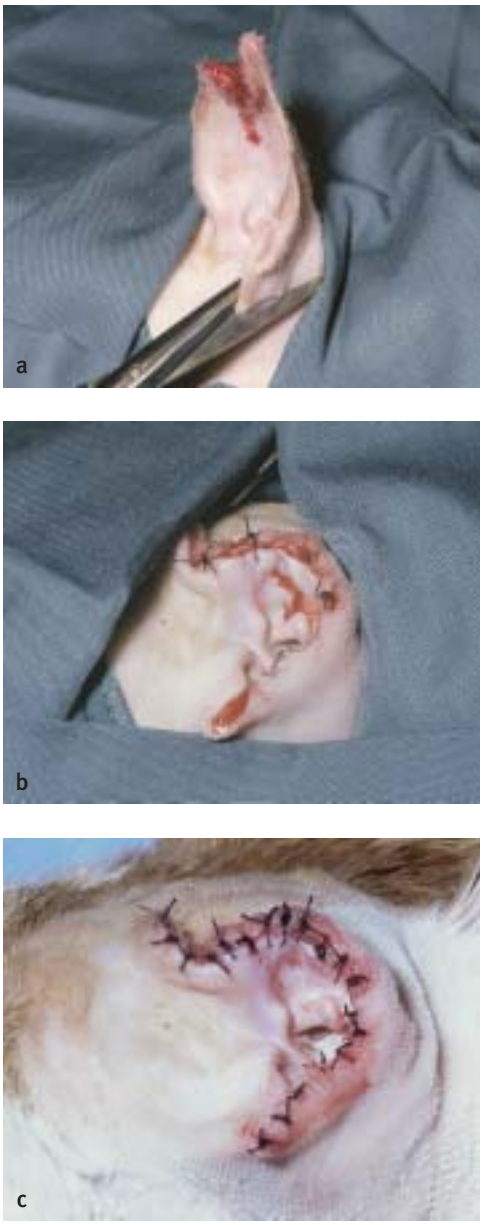


Figure 1.25 a–c: (a) Squamous cell carcinoma of the auricle in a cat: the auricle is removed by cutting with scissors through both layers of the skin and the cartilage at the same time. (b) The skin of the convex side of the auricle is sutured over the cartilage to the skin on the concave side, first by placing 4 to 6 sutures to divide the wound into equal segments. (c) Interrupted skin sutures are placed between these to complete the closure.

1.6.3 Tumors of the middle ear

Tumors of the middle ear are rare in dogs and cats. The history and clinical signs are similar to those of chronic middle ear inflammation and the same diagnostic procedures are followed. The growth of a tumor in the middle ear often leads to destruction in the surrounding temporal bone. The clinical signs may reflect dysfunction of the facial nerve (VII) and the vestibulocochlear nerve (VIII). Tumor has occasionally been discovered during diagnostic bulla osteotomy when it could not be differentiated from chronic middle ear disease by CT. The prognosis after removal of a malignant tumor from the middle ear is guarded. These tumors can extend into the labyrinth and can also eventually reach the meninges and brain. CT and MRI can reveal the location of the tumor and the extent of invasion. Treatment is usually not successful and damage to vital tissues is unavoidable.

1.7 Trauma to the ear

1.7.1 Trauma to the auricle

Trauma to the auricle is common in cats, especially those that roam outside and thus meet other cats. The trauma is usually caused by another cat's claw, which lacerates the auricle, causing hemorrhage. If the laceration is fresh, suturing the wound is preferred and results in more rapid wound healing and better cosmetic results than does healing by second intention. However, disinfection is needed, since many cats carry *Pasteurella multocida*. The cat is sedated and the wound is cleaned with 0.9 % NaCl solution and a disinfectant. Unless it is very short, the hair should be clipped and the wound should be débrided if necessary. Fine interrupted sutures with atraumatic delayed-solubility material are used to join skin to skin, avoiding the cartilage, first on the concave side and then on the convex side. Sutures are placed starting at the edge of the ear and proceeding inwards, to avoid an irregularity at the edge (Figure 1.26). In dogs laceration of the auricle often occurs in a fight and is the result of a bite by the canine teeth. The wounds must be assumed to be contaminated. Small tears can be sutured skin-to-skin without anesthesia but large wounds should be sutured in the manner just described for the cat, after any bleeding vessels have been ligated. Aftercare includes a broad-spectrum antibiotic for at least 5 days, analgesic treatment for at least 5 days, and use of an Elizabethan collar to protect the wound. Older wounds should be treated according to general principles of wound cleaning, débridement, and correction as described for superficial wounds.[83]

In cats an abscess may develop in the auricle after fighting, usually caused by *Pasteurella multocida*. The cat usually becomes depressed and febrile, and the auricle is obviously painful. The skin over the abscess should be opened and the pus removed by gentle compression,

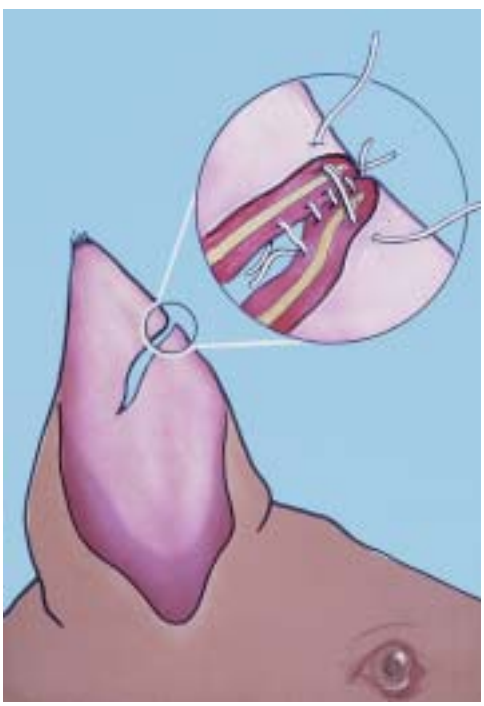


Figure 1.26:
A fresh laceration of the auricle is closed by suturing skin to skin on both sides, avoiding the cartilage. Suturing begins at the edge of the auricle and proceeds inward, so that the edge will be smooth.

followed by irrigation with 0.9 % NaCl solution. A broad-spectrum antibiotic is administered for 10 days. If this treatment is carried out within 1 or 2 days after appearance of the abscess, healing is uncomplicated and there is no scarring and shriveling of the auricle.[78]

1.7.2 Auricular hematoma

Auricular hematomas have long been thought to result from trauma, such as that caused by vigorous shaking or scratching of the ear(s). It has also been proposed that they may be the result of an autoimmune or immune-mediated lesion of the auricular cartilage, but the arguments in support of this have been conflictive and speculative.

Hematomas may involve the entire auricle or only part of it. They occur in both cats and dogs, often in association with inflammation of the external ear canal, which is thought to cause pruritus that results in head shaking. The hematoma develops within the cartilage and is therefore sterile. The diagnosis is based on the clinical appearance of the heavy auricle, bulging on the concave side, and the absence of clinical signs of an abscess, such as fever and pain. The hematoma should only be opened under sterile surgical conditions, to avoid contamination of the accumulated blood and the development of an abscess within the auricle. Therapy involves evacuation of the hematoma and application of pressure to prevent reaccumulation of blood. The bleeding within the cartilage separates it into two layers and these should be compressed together for about 2 weeks after removal of the blood. Simple needle aspiration is inadequate treatment and results in organization of the hematoma, with fibrosis and subsequent deformation of the auricle. The most effective treatment is to make an adequate incision for removal of the clotted blood and then to press the two layers together with through-and-through interrupted mattress sutures using slightly elastic, delayed-absorption suture material (polyglecapron). To avoid folding of the auricle, which would deform it, the incision is usually made in the shape of an S over the hematoma on the concave side. The sutures are tied on the convex side, where the skin and subcutis are thicker and more resistant to the pressure of the knots. The various surgical techniques for applying pressure over an auricular hematoma have comparable success as long as the pressure is applied equally and moderately over the entire hematoma, without interruption for 2 weeks. The cat should be kept in the house and the dog should be given rest and kept restricted for 2 weeks after surgery. A broad-spectrum antibiotic and an analgesic should be administered for 10 days and the sutures should be removed after 2 weeks. The dog should be kept quiet for one more week after the sutures are removed.

Inadequate treatment can lead to recurrence of the hematoma. A more serious complication is organization of an untreated or inadequately treated hematoma, resulting in fibrosis and ultimately a »cauliflower« ear. The contraction of the fibrous tissue leads to necrosis and ossification of the cartilage because it interferes with circulation. The result is continuous pain and scratching at the auricle, which can only be relieved by its amputation. If this surgery is delayed, the scratching and rubbing of the ear cause chronic inflammation of the external ear canal, in which case not only the auricle but also the ear canal will have to be removed to obtain relief.

1.7.3 Trauma to the external ear canal

Blunt trauma to the external ear canal is most often caused by attempting to remove ear wax with objects such as forceps, cotton swabs, or fingers covered with cotton. The skin of the ear canal is quite thin and it also has a very thin subepithelial layer. This tender skin is easily

abraded, which gives rise to secondary infection. The history and clinical signs and a few judicious questions will reveal the cause of the infection. The owner should be made aware of this sequence and should be advised to cease all attempts to clean the ear canal. The ear canals of most dogs and cats should be left alone and owners should be advised to ignore the small to moderate amount of cerumen that appears in the opening. Genuine overproduction cannot be resolved by simply removing that which becomes visible. Instead, otoscopic examination should be performed by the veterinarian to determine the reason for it.

Bleeding from the external ear canal after a dog or cat has been struck by an automobile may be caused by fracture of the temporal bone, in which case the blood is escaping through a ruptured tympanic membrane, or it may be caused by laceration of the external ear canal. An important rule is that the ear canal should not be flushed in such cases. Fracture of the temporal bone can open the way from the external ear canal to the brain. The patient should first be given a thorough clinical examination for signs of shock, such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilation of the pupils, hypothermia, muscle weakness, restlessness, and depression or even coma.[71] Then a search should be made for other fractures or wounds. Temporal bone fractures do not per se require immediate attention unless brain damage is suspected. In the absence of signs of brain damage, and when other traumatic injuries have been attended to, the nature and extent of the suspected temporal bone fracture should be examined by radiography and/or CT. Prolonged anesthesia is needed and thus these procedures are usually delayed for 24 hours or more following the accident. If fracture of the temporal bone can be excluded, the nature of trauma to the external ear should be determined. Interruption of the external ear canal usually occurs at the level of the fibrous connection between the auricular and the annular cartilage and it includes laceration of the skin lining the canal in that area. The diagnosis can be made by palpation of the ear canal and by otoscopic examination. The continuity of the external ear canal should be restored at an early stage, before spontaneous organization causes permanent stenosis. The skin is incised over the vertical part of the canal and dissection proceeds to the site of the rupture. The two cartilage ends are débrided and then brought together, taking care to oppose them in a natural position. They are first joined by a few sutures through the cartilage and skin of the ear canal and then the rupture is closed entirely by additional interrupted sutures. Fine suture material with delayed solubility is used. Healing takes about 3 weeks in this contaminated area. Otoscopic examination is delayed until 4 weeks after surgery. As after all ear surgery, an analgesic and a broad-spectrum antibiotic are administered systemically, in these cases for at least 10 days. The dog or cat should be given rest for 3 weeks; an Elizabethan collar should not be used. When surgery has been delayed, the approach and suturing are similar but much more tissue will have to be removed to reopen the ends of the ear canal. Stenosis of the external ear canal will often develop. If there is an abscess around the distal part of the external ear canal, the entire canal should be removed (Chapter 1.5), for first-intention healing cannot be expected and fistulas will be a continuing problem if it is not removed.

1.7.4 Trauma to the tympanic membrane

Trauma can cause rupture of a small or large part of the tympanic membrane. Ruptures usually occur in the pars tensa. The diagnosis is made by otoscopy. A fresh rupture is recognized by bleeding at the margins. Healing will be influenced by the presence of inflammation in either the middle or the external ear. Healing of the

tympanic membrane has been investigated in cats, rats, mice, hamsters, and guinea pigs.[8, 33, 56] These investigations revealed that traumatic perforations almost always close spontaneously. The healing of small perforations was studied in guinea pigs.[33] The tympanic membrane has no underlying tissue comparable to that beneath the skin. When the tympanic membrane is perforated, the healing of the connective tissue defect tends to lag behind the healing of the epithelial layer. The highly proliferative squamous epithelium spreads over the perforation, creating what is described as a snake-head appearance as the gap in the epithelial layer is closed before that in the connective tissue layer.[8, 56] The closure of the epithelial defect follows the progress of epithelial migration, hence from below upwards. The advancing edge of the epithelium fills and thus closes the perforation in 5 to 9 days, after which the epithelium becomes thinner so that it rapidly resumes a more normal appearance. The fibrovascular repair occurs secondary to this and bears no relation to the direction of the epithelial response. Young fibrous tissue takes 9 days to fill in a small perforation, by which time the epithelium over the site has almost returned to normal. There was a striking lack of activity in the mucosal layer on the inner side of the tympanic membrane during the healing of a perforation. The mucosa appeared to contribute little to the process.

In dogs, the healing of experimentally perforated tympanic membranes—in which most of the pars tensa was absent after perforation—was complete by 21 to 35 days.[66] In a dog in which loss of the tympanic membrane was associated with inflammation of the middle ear, the membrane was almost completely healed in 3 weeks after treatment was started. Healing progressed from below upwards and a red zone around the perforation indicated the continuing activity of the healing process (Figure 1.27 a, b).

Nonhealing of the tympanic membrane can usually be attributed to persistent infection and inflammation in the middle ear or the external ear canal. White fibrous tissue around the defect indicates that healing has halted (Figure 1.19). It will not be resumed until this fibrous tissue is removed and the middle ear is flushed until clean.

1.7.5 Trauma to the temporal bone

Bleeding from the external ear canal after a dog or cat has been struck by an automobile may be caused by fracture of the temporal bone, in which case the blood is escaping through a ruptured tympanic membrane, or it may be caused

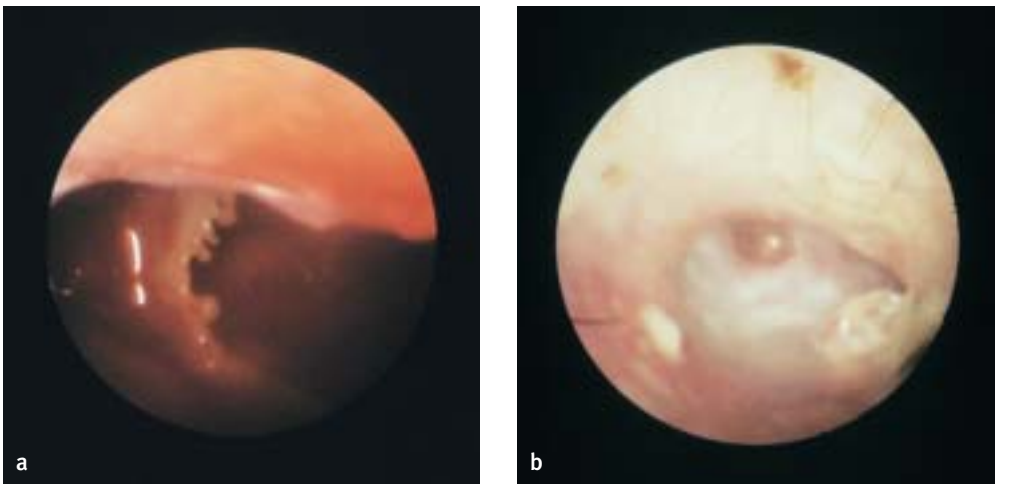


Figure 1.27 a, b: (a) The tympanic membrane is missing in this dog as a result of inflammation of the middle ear, indicated by the red mucosa. (b) Three weeks after treatment was started, the tympanic membrane was almost completely healed. Healing began at the bottom and progressed upward. The red zone around the perforation indicates continuing healing activity.

by laceration of the external ear canal. An important rule is that the ear canal should not be flushed in such cases, for fracture of the temporal bone can open the way from the external ear canal to the brain. The patient should first be given a thorough clinical examination for signs of shock, such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilation of the pupils, hypothermia, muscle weakness, restlessness, and depression or even coma.[71] Then a search should be made for other fractures or wounds. Temporal bone fractures do not per se require immediate attention unless brain damage is suspected. There may be sensorineural hearing loss and facial paralysis, depending on the fracture lines. The prognosis for spontaneous recovery of these functions is reserved.

1.8 Ototoxicity

Ototoxicity is defined as the toxic effect of a drug or other chemical on the inner ear, causing vestibular and cochlear damage. The effects depend on the concentration of the agent and the duration of its contact with the sensory hair cells. In dogs and cats the clinical signs are usually unilateral vestibular dysfunction and hearing loss (ipsilateral). The history in cases of acute ototoxicity in dogs and cats usually reveals that severe vestibular dysfunction began after local treatment of the ear with a disinfectant or antiparasitic drug, or sometimes after other otic drops were used following mechanical cleaning of the external ear canal.

Clinical signs of ototoxicity. The signs of vestibular dysfunction in dogs and cats caused by acute unilateral ototoxicity are rotation of the head and cranial portion of the body, with the treated ear down; deviation of the eyes toward the treated side; eye and sometimes head nystagmus with the fast component toward the untreated side; and if the animal is able to stand, there is a tendency to circle toward the treated side. If both labyrinths are affected, the animal is unable to stand, is severely disoriented, and does not respond to auditory stimulation. The vestibular system reacts immediately with its central compensatory mechanisms—a process of neuroplasticity, sensory substitution (vision and proprioception), and learning processes. Within 3 days the nystagmus disappears, within a week the dog can stand with some support, and within 3 weeks it can walk. The head rotation is usually permanent, but can be masked by compensation to the extent that it is only observed when the animal's interest is absorbed by some event. The clinical signs and dysfunctions that are still present at 3 months will be permanent. Although the clinical signs may vary in their initial severity and according to the progress of compensation, any vestibular dysfunction is permanent. Prevention of ototoxicity is often possible and will be most effective if its cause is fully understood.

Effects of ototoxic drugs. The ototoxic drug or chemical diffuses into the perilymph of the inner ear and enters the hair cells of the cochlea and the vestibular organ. The affected hair cells undergo ultrastructural changes and degenerate subsequently. The interval during which an ototoxic agent can affect the hair cells was measured experimentally following the instillation of chlorhexidine in the middle ear of an anesthetized guinea pig. Hair cell activity in the cochlea was measured by recording the cochlear microphonics (hair cell potentials). Within 10 minutes the threshold of hearing increased from 30 dB SPL to around 50 dB SPL, and within 80 minutes to 70 dB SPL.[22] Although the rate may be slightly different in dogs and cats, these findings illustrate how rapidly the hair cells can be destroyed.

Pathways of ototoxic drugs. There are two main pathways for the ototoxic agent to enter the

perilymph: via the bloodstream and locally via the middle ear, with passage through the semipermeable membrane of the round window and the oval window. Most cases of ototoxicity in dogs and cats occur via the local route and the clinical signs usually indicate unilateral involvement. Many drugs and chemicals have been tested for their ototoxicity, usually as a sequel to clinical suspicion that ototoxicity has been the cause of specific cases of vestibular dysfunction and hearing loss.[3, 4, 5, 6, 22, 24, 46] Agents that are locally ototoxic include antibiotics (aminoglycosides, polypeptides, chloramphenicol, erythromycin, tetracycline, and oxytetracycline), disinfectants, local anesthetics, vehicles used in otic drops and ointments (propylene glycol, glycerol, and phenols), and exotoxins produced by *Pseudomonas*, *Staphylococcus,* and *Streptococcus*. These agents do not pass the intact tympanic membrane but once they gain entrance to the middle ear they do pass through the membrane of the round window. Its permeability in cats was demonstrated in experimental studies of most of the above-mentioned agents carried out in cats with normal middle ears and in cats with otitis media.[25] There is evidence that in early stages of otitis media in cats the permeability of the round window membrane to macromolecules is increased, and thus also the permeability to smaller molecules such as toxins and enzymes.[23, 25] Staphylococcal pyrogenic exotoxin instilled into the middle ear of cats with otitis media passed through the membrane of the round window to appear in the perilymph within 25 minutes and was also found there 12 hours after exposure.[24] This may explain the occurrence of vestibular dysfunction in cats that have otitis media but an intact tympanic membrane. The studies of the permeability of the membrane of the round window also indicate that in the interest of preventing ototoxicity, drops should not be used in the treatment of otitis media when there is a possibility that the tympanic membrane is ruptured, nor should germicidal agents be used prior to otological surgical procedures.

Ototoxic drugs. The ototoxicity of certain systemically administered drugs is well documented. Aminoglycoside antibiotics in ototoxic doses tend to cause selective destruction of the outer hair cells in the basal turn of the cochlea, with progression toward the apex as the dose and the duration of treatment are increased.[58] The effects of systemically administered neomycin on the wave form of auditory-evoked brain stem potentials were studied in dogs. All treated dogs eventually showed a loss of auditory evoked brain stem potentials during a period of 22 to 50 days of continuous treatment with neomycin.[45] Antineoplastic drugs such as cisplatin and carboplatin are known for their ototoxicity. Studies of the temporal bones of human patients with cisplatin-induced hearing loss have demonstrated degeneration of outer hair cells at the basal turn of the cochlea, spiral ganglion cells, and cochlear neurons, although the vestibular neurons appeared unaffected.[69] The ototoxic effects vary with dose of the drug, the duration of treatment, and the age and comorbidities of the patient. Carboplatin has been thought to be less ototoxic than cisplatin. There is continuing research aimed at finding medications to prevent the ototoxic effect of these antineoplastic drugs because of their great importance in cancer therapy. Given the increased use of cytostatic drugs in dogs and cats, one should be aware of these ototoxic effects.

1.9 Hearing in dogs and cats

The mechanisms of hearing are considered in Chapter 1.1. The clinical consequences of hearing and hearing dysfunction will be discussed separately for dogs and cats.

1.9.1 Hearing and hearing loss in dogs

For dogs living in a social community the loss of hearing can be debilitating, for the recognition of patterns and habits are important to dogs. Most communication with dogs occurs via sounds, especially that with the humans with whom the dog lives or upon whom it is dependent. Conversation with a dog occurs at the same frequencies and intensity levels as does conversation between persons. But what does the dog hear? Most dog owners have the perception that their dogs hear more than they hear themselves. However, Brain stem Auditory Evoked Responses (Chapter 1.3) to click stimuli of varying intensity revealed that hearing thresholds in adult dogs were not different from those in humans.[73, 77] They differed only in the discrimination of frequencies, for dogs around 6 years of age can hear sounds at frequencies up to 32 kHz, while their optimal hearing is for tone bursts of 12 to 16 kHz.[73] (Figure 1.28). Young humans can hear sounds at frequencies between about 20 Hz and 20 kHz, while adult humans are limited to upper frequencies of about 15 kHz to 17 kHz.[52]

Hearing loss should be differentiated as conductive, sensorineural, or combined conductive and sensorineural hearing loss. Conductive hearing loss is caused by disorders in the conductive part of the auditory system: the external ear canal, the tympanic membrane, the ossicles, and the middle ear. Sensorineural hearing loss is caused by disorders in the sensorineural part of the auditory system: the cochlea, the cochlear nerve, and the central auditory system in the brain. In combined conductive and sensorineural hearing loss both parts of the auditory system are affected.

The most common causes of conductive hearing loss in dogs are cerumen plugs and severe tissue proliferation in the external ear canal, either of which can prevent the vibrations in air (sound) from reaching the tympanic membrane.[91] Otitis media and tumors involving the external ear canal and middle ear can also cause conductive hearing loss. When hearing loss is suspected in a dog, otoscopic examination of the external ear canal and tympanic membrane is the first diagnostic procedure (this always precedes even a hearing test). If otoscopy reveals no abnormality sufficient to explain the hearing loss, the loss is presumed to be sensorial and the appropriate diagnostic procedures are a hearing test and brain stem evoked response audiometry (BERA). The causes of sensorineural hearing loss include congenital deafness, presbyacusis (hearing loss associated with ageing), labyrinthitis, brain tumors, and hydrocephalus.[67]

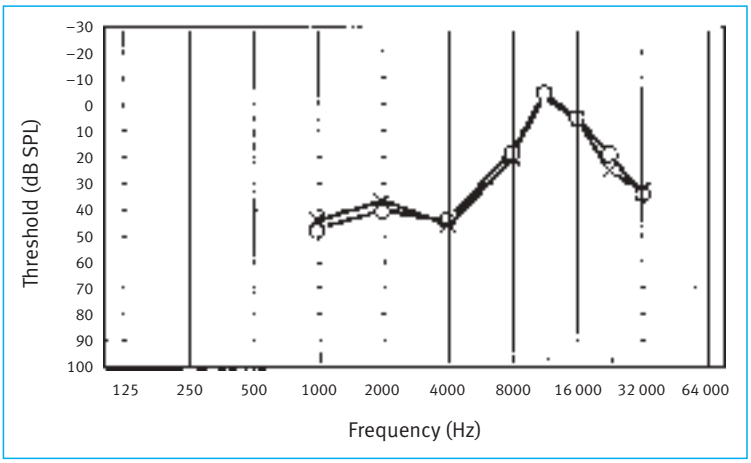


Figure 1.28: Threshold audiogram, showing mean threshold values in 10 normal dogs with a mean age of 6 years; x = left ear, o = right ear. The standard deviation of mean threshold values ranged between 6.7 and 24.6 dB. (From: Ter Haar G, Venker-van Haagen AJ, de Groot HN, van den Brom WE. Click and low-, middle-, and high-frequency toneburst stimulation of the canine cochlea. J Vet Intern Med 2002; 16(3): 274–280, Fig. 4).

1.9.2 Brain stem auditory evoked responses in dogs

Brain stem evoked responses to click and low-, middle-, and high-frequency tonebursts were recorded in 10 dogs, 3.5 to 7 years of age and weighing 12.5 to 21.3 kg .[73] Details of the materials and methods are given in Chapter 1.3.

The brain stem evoked response to click stimulation at 80 dB SPL resulted in the characteristic pattern of 5 to 7 positive waves. Following the conventional labeling with Roman numerals, wave I is the first identifiable wave more than 1.2 ms after stimulation (to bypass

stimulus artifacts) and wave V is that preceding the deepest negative trough more than 3.5 ms after stimulation (Figure 1.29). The most readily identifiable peaks were usually I, II, III, V, and VI. Wave IV was not readily identifiable in all recordings and wave VII was identified infrequently and was therefore excluded from further analysis. All toneburst stimulations at 80 dB SPL yielded the same characteristic pattern of at least 5 identifiable waves, except for the 1 kHz toneburst stimulation, which elicited a frequency-following response overlapped by biphasic potentials (Figure 1.29), but even in these tracings waves I and V could be identified. As the intensity of the stimulus decreased, the evoked response decreased in amplitude while peak latency increased until it reached a threshold.

At 80 dB SPL, peak latencies were significantly greater after toneburst than after click stimulation (Figure 1.30). Because the increase in peak I latency after toneburst stimulation accounts for most of the increase in latencies of all subsequent peaks, the peak I latencies of all toneburst stimulations were analyzed further. Peak I latency after 12 and 16 kHz toneburst stimulation was significantly shorter than that after 1, 2, 4, 8, 24, and 32 kHz toneburst stimulation. Peak I latency after 32 kHz toneburst stimulation was significantly longer than that at all other frequencies. The increase in individual peak latencies after toneburst stimulation was not caused by an increase in peak I latency alone, because there also were significant differences in interpeak latencies.

The interpeak latencies for peaks I to V were significantly longer after 4, 8, 12, 16, 24, and 32 kHz toneburst stimulation than after click stimulation, but after 2 kHz toneburst stimulation they were significantly shorter, and after 1 kHz toneburst stimulation they did not differ significantly from those after click stimulation. Significant differences in interpeak latencies also were found after all toneburst stimulations. The interpeak latencies I to V after 16 kHz toneburst stimulation was significantly longer than that at all other toneburst stimulation frequencies. From 2 kHz to 8 kHz, interpeak latencies I to V lengthened progressively and the differences between successive frequencies were significant.

Interpeak latency for peaks I to III after toneburst stimulation was not significantly different from that after click stimulation, except for that after 24 kHz toneburst stimulation, which was significantly longer. Interpeak latency I to III could not be determined after 1 kHz toneburst stimulation because peak III could not be identified. Interpeak latency I to III after toneburst stimulations was significantly longer at 24 and 32 kHz than at 2, 4, and 8 kHz.

The mean amplitude of wave I was significantly lower after all toneburst stimulations than after click stimulations. The amplitude decreased progressively with increasing frequency for toneburst stimulations from 2 kHz to 16 kHz, but increased again for tonebursts of 24 and 32 kHz. Wave I amplitude for 8, 12, and

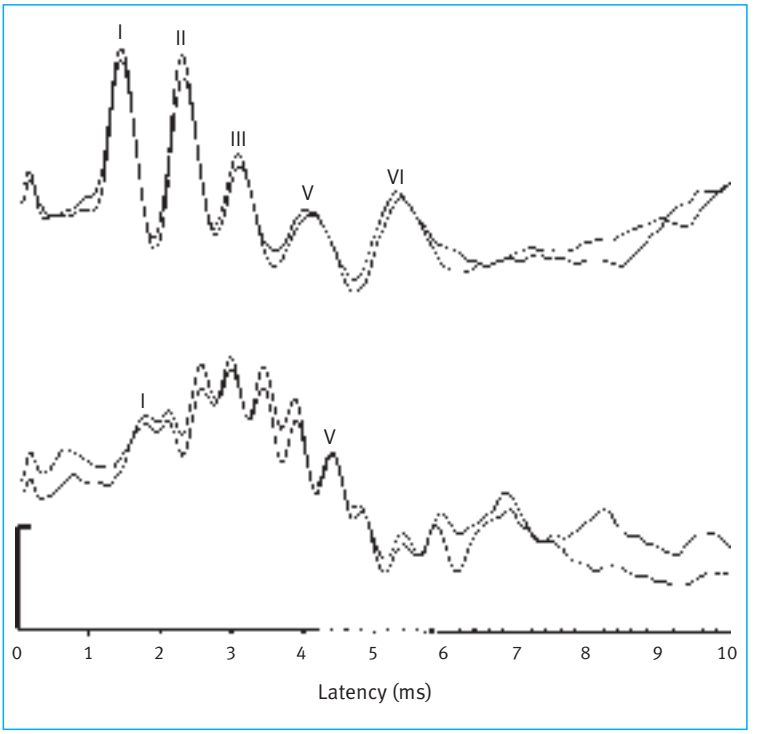


Figure 1.29: Representative sample (duplicate recordings) of brainstem evoked responses following 80 dB SPL click stimulation (upper tracing) and 80 dB SPL 1 kHz toneburst stimulation (lower tracing) in the left ear of one dog. The positive peaks are labeled with Roman numerals. Vertical bar = 1 µV. (From: Ter Haar G, Venker-van Haagen AJ, de Groot HN, van den Brom WE. Click and low-, middle-, and high-frequency toneburst stimulation of the canine cochlea. J Vet Intern Med 2002; 16(3):274–280, Fig. 2).

16 kHz toneburst stimulation was significantly lower than that for other frequencies. The differences in mean wave I amplitude among these 3 frequencies were also significant.

The mean amplitude of wave V was significantly lower after 4, 8, 12, and 16 kHz toneburst stimulation than after the click stimulation, whereas that after 32 kHz toneburst stimulation was significantly higher and that after 2 and 24 kHz toneburst stimulations did not differ significantly from that after click stimulation. The mean amplitude of wave V after the 4, 8, 12, and 16 kHz toneburst stimulations was significantly lower than after other toneburst stimulations, being lowest after 8 kHz. Compared with the amplitude ratio after click stimulations, the ratios after toneburst stimulations were significantly higher at 2, 16, 24, and 32 kHz, significantly lower at 8 kHz and 12 kHz, and not significantly different at 4 kHz. A trend could be recognized in amplitude ratios for toneburst stimulations: the ratio decreased progressively from 2 kHz to 8 kHz and then increased progressively from 8 to 32 kHz; the difference at each succeeding step being significant. The responses to 1 kHz toneburst stimulations were excluded because the potentials were biphasic.

There were marked differences in the threshold for different stimulations, the lowest being for click stimulations and for 12 kHz and 16 kHz toneburst stimulations; the differences among these 3 were not significant. The thresholds for all other toneburst stimulations were significantly higher than for click stimulations. The 2 kHz threshold was significantly lower than the 1 kHz threshold, but not significantly different from the 4 kHz threshold. The 8 kHz threshold was significantly lower than the 4 kHz threshold and higher than the 12 kHz threshold. The 24 kHz threshold was significant lower than the 32 kHz threshold. A threshold audiogram for toneburst stimulations was constructed from these values.[73] (Figure 1.28).

Other laboratories have published similar results for brain stem evoked response audiometry in dogs using click stimulation and reference values can be compared, but with certain limitations. Most laboratories have employed toneburst stimuli up to only 8 or 12 kHz, which,

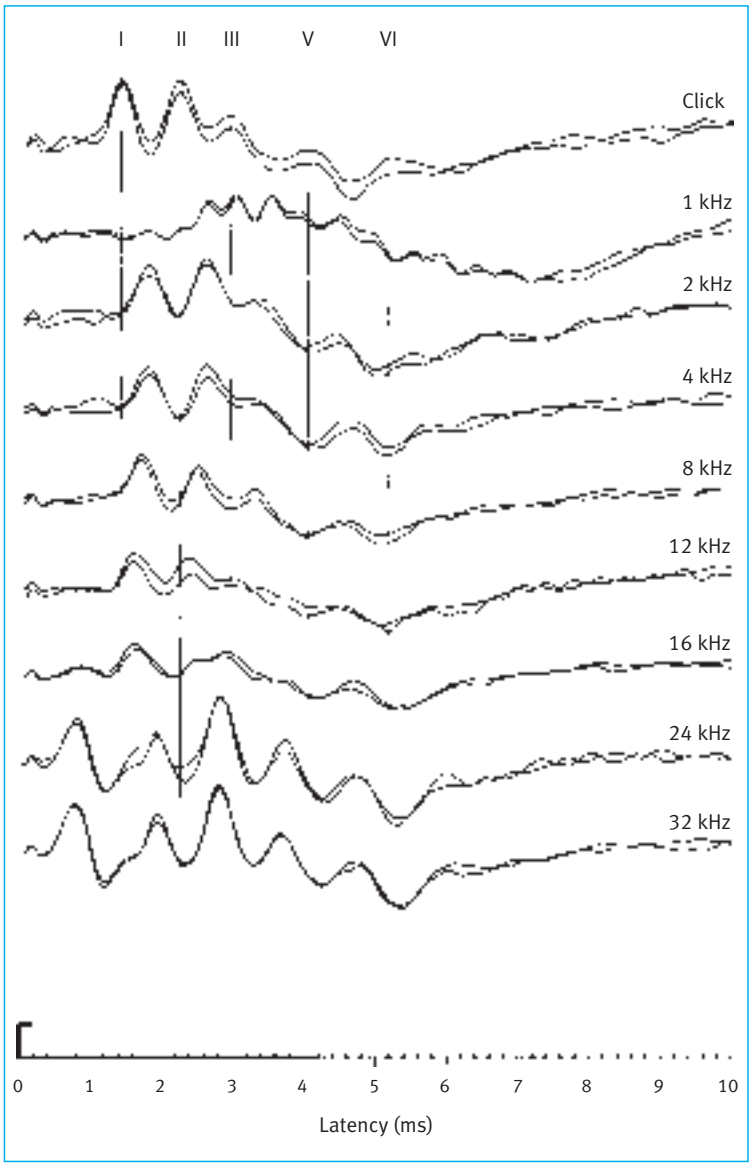


Figure 1.30:
Comparison of latencies of the evoked responses after 80 dB SPL click stimulation and after 80 dB SPL toneburst stimulation ranging from 1 to 32 kHz. Duplicate recordings were made at each frequency of stimulation of the left ear in one dog. The positive peaks are labeled with Roman numerals. Vertical bar = 1 µV. (From: Ter Haar G, Venker-van Haagen AJ, de Groot HN, van den Brom WE. Click and low-, middle-, and high-frequency toneburst stimulation of the canine cochlea. J Vet Intern Med 2002; 16(3): 274–280, Fig. 3).

being the standard for humans, is all that is possible with readily available testing equipment.

The relationship between latency of brain stem auditory-evoked potentials and head size in dogs was studied using click stimuli. The results showed that for every 1 cm increase in cranial distance an allowance should be made for an increase in latency of 0.006 ms for wave I, 0.03 ms for wave III, 0.05 ms for wave V, and 0.05 ms for the I–V interpeak interval.[43]

Sensorineural hearing can be assessed independent of a loss of conductive hearing. In place of sounds transmitted in air, vibrations of similar intensities are conducted to the bone of the skull. While the cochleas receive stimuli from a vibrator placed on the skull, the electrodes for recording brain stem auditory evoked potentials are positioned subcutaneously in the same way as when using air-conducted stimuli. The resulting evoked potentials are a summation of the activities of both cochleas. Studied in young dogs, the threshold for brain stem evoked potentials was about 50 dB higher for bone conduction than for air conduction of stimuli.[91]

1.9.3 Hearing and hearing loss in cats

For cats living in a social community, hearing loss can be a hindrance, but if the cat roams free it can also be a life-threatening disability. Cats react to the same sounds perceived by humans. The thresholds of hearing have been identified (see below) but the cochlear sensitivity to high tonebursts has not been reported.

As in dogs, hearing loss should be differentiated as conductive, sensorineural, or combined. Conductive hearing loss is caused by disorders in the conductive part of the auditory system: the external ear canal, the tympanic membrane, the ossicles, and the middle ear. Sensorineural hearing loss is caused by disorders in the sensorineural part of the auditory system: the cochlea, the cochlear nerve, and the central auditory system in the brain. In combined conductive and sensorineural hearing loss both parts of the auditory system are affected.

The most common causes of conductive hearing loss in cats are middle ear polyps that block the ear canal after rupturing the tympanic membrane, and severe tissue proliferation in the external ear canal. Other causes are otitis media and tumors arising in the external ear canal or the middle ear. When hearing loss is suspected in a cat, otoscopic examination of the external ear canal and tympanic membrane is the first diagnostic procedure (this always precedes even a hearing test). If otoscopy reveals no abnormality sufficient to explain the hearing loss, the loss is presumed to be sensorineural and the appropriate diagnostic procedures are a hearing test and Brain stem Evoked Response Audiometry (BERA). The causes of sensorineural hearing loss include congenital deafness, presbyacusis (hearing loss associated with ageing), labyrinthitis, and brain tumors.[67] There have been many reports of congenital deafness, especially in white cats, but hearing tests have not often been used in other types of hearing loss in cats.

1.9.4 Brain stem auditory evoked responses in cats

Hearing can in principle be tested in cats in the same way that it can in dogs, but there have been few reported studies of the sensitivity and frequency specificity of the cochlea in normal cats. One study of the frequency specificity of hearing in cats using auditory brain stem responses concluded that at near-threshold intensity, both the 2 kHz and 4 kHz tonebursts are frequency specific, while click and noise-burst BAER contain no contributions below 2 kHz.[40] The mean response threshold was 15 dB SPL for the click stimulus, 15 dB SPL for the

noise burst, 10 dB SPL for the 4 kHz toneburst, 15 dB SPL for the 2 kHz toneburst, 25 dB SPL for the 1 kHz toneburst, and 50 dB SPL for the 0.5 kHz toneburst. It was concluded that click and noise burst stimuli used in brain stem evoked response audiometry do not contain sufficient low-frequency energy at near-threshold levels to stimulate the apical region of the cochlea. The 2 and 4 kHz tonebursts are frequency-specific stimuli. Both the 0.5 and 1 kHz tonebursts are broadband stimuli and not frequency specific. The latter results may be applicable to clinical brain stem response audiometry when transposed down one octave.[18, 40]

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2 The Nose and Nasal Sinuses

2.1 Functional considerations

Nose. The nose represents the facial portion of the respiratory system. It comprises the external nose, with its the bony case and movable cartilaginous parts, and the nasal cavity. The bony external nose is formed laterally by the incisive bones and the maxillae, and dorsally by the paired nasal bones. The cartilages, which form the most rostral part of the external nose, consist of the paired dorsolateral and a ventrolateral cartilages, the paired accessory cartilages, and the unpaired septal cartilage which separates most of the nasal cavity into right and left halves. The nasal cavity is the respiratory passageway. It extends from the nasal openings in the nasal plane, which is the apical portion of the nose, to the choanae, which are the right and left openings into the nasal portion of the pharynx. The lateral and dorsal boundaries of the nasal cavity are as described for the external nose and the ventral boundary is formed by the incisive bone, the maxillae, and the palatine bone. The nasal conchae are cartilaginous or slightly ossified scrolls filling the nasal cavities.[14]

The nose is not only the entry and passageway that enables air to reach the alveoli, where gaseous exchange occurs, but it also modifies or regulates the flow of the air and facilitates water and heat exchange, thereby conditioning the air before it reaches the lungs. Olfaction is a very important sensory function in dogs and cats. It is facilitated by passage of the inspired air over the olfactory epithelium, a sheet of neurons and supporting cells that lines the nasal cavities.

Paranasal sinuses. They are well developed in dolichocephalic dogs and less so in brachycephalic dogs and in cats. They are diverticula of the nasal cavity formed by excavation between the inner and outer tables or plates of the skull bones. The sinuses develop mostly after birth and reach maximum size in the adult animal. They retain their connections with the nasal cavity, but the openings are generally narrow, allowing only a slow exchange of air.[11] In dogs and cats there is no communication between the left and right frontal or maxillary sinuses. The frontal sinus consists of a lateral, a medial, and a rostral part. In dogs, the lateral part occupies the entire truncated enlargement of the frontal bone that forms the supraorbital process. It may be partly divided by osseous septae. The rostral part is deficient medially, resulting in the nasofrontal opening into the nasal fossa, through which ectoturbinate 3 extends. The entire surface of the ectoturbinates and the frontal sinuses is covered with mucosa. The medial part of the frontal sinus is variable in size and contains ectoturbinates 1 and 2. The maxillary sinus or maxillary recess is a lateral diverticulum of the nasal cavity. It has a restricted opening to the nasal cavity and the nasal mucosa continues into it. It is formed by the ethmoid, maxillary, palatine, and lachrymal bones.[15]

There is much speculation about the functions of the nasal sinuses. It seems plausible that the frontal sinuses protect the rostral part of the brain against forceful frontal trauma, and give some thermal protection.

2.1.1 Regulation and conditioning of the inspiratory and expiratory airflow

The respiratory airflow through the nasal cavity is regulated by the ventilatory control systems. In the nasal cavity stimulation of the sympathetic nervous system decreases nasal secretion and causes vasoconstriction which widens the nasal passageway and decreases respiratory resistance. Stimulation of the parasympathetic system has the opposite effect, increasing nasal

secretion and causing vasodilatation which narrows the nasal passageway and increases respiratory resistance. The nose represents an important part of the resistance of the airway and thereby influences gas exchange in the alveoli. The resistance has to be overcome by greater negative pressure in the thorax during inspiration, which leads to better expansion and filling of the alveoli by the inspired air and a greater venous blood flow in the lungs. In humans, a prolonged increase in nasal resistance can lead to cor pulmonale, cardiomegaly, and pulmonary edema.[3] In dogs, pulmonary edema is known to develop in laryngeal obstruction. Pulmonary edema may occur by a similar mechanism when there is severe obstruction of nasal airflow. In humans and dogs the most common response to increased nasal resistance is, however, breathing through the mouth.[3, 48]

The inspired air is warmed (or cooled) as it passes through the nose by radiation from (or absorption of heat by) the mucosal blood vessels. The flow of blood is from posterior to anterior, opposite to the flow of the inspired air. Humidification occurs by evaporation from the blanket of mucus covering the mucosa and the serous fluid from the nasal glands. The nasal blood flow and the activity of the nasal glands are regulated by the autonomic nervous system. The autonomic innervation of the nose consists of parasympathetic and sympathetic nerve fibers, joined together in the vidian nerve. The conditioning of the inspired air by the nose is a very important function for protection of the alveoli and it occurs even under extremely dry and cold conditions, even then providing the bronchi with air at body temperature and close to 100 % humidity.[3]

2.1.2 Mucosal cleaning

The pseudostratified respiratory mucosa in the nose consists of ciliated, intermediate, basal, and goblet cells. They rest on a well-defined basement membrane supported by a deep, loose lamina propria containing small blood vessels, venous plexus, ducts of mucous and serous glands, sensory nerves, and blood cells. The tall ciliated cell is the predominant type and it extends from the basement membrane to the luminal surface, where there are cilia admixed with microvilli.[3] The cilia actively move the overlying blanket of mucus by a to-and-fro movement called the ciliary beat. There is a forceful forward movement and a less forceful recovery. In the recovery stroke the shaft of the cilium curls back on it self so that it does not reach the layer of mucus. The forward movement transports the mucus blanket toward the laryngopharynx and the esophagus. The bilayered mucous blanket, produced mostly by the serous and goblet cells, is sticky, tenacious, and adhesive. The outer layer is more viscid than the deeper, periciliary layer. The blanket functions as a lubricant, protects against desiccation, and traps insoluble particles and soluble gases.[3] Allergens and bacteria caught on the outer layer are thus carried to the esophagus. Soluble material reaches the periciliary layer and is removed with it. The motion of the cilium is caused by the sliding of one axonemal tubule against an adjacent one, creating a shearing force that induces bending and contact with the mucus, and may initiate the coordinated metachronous movement. The energy for this work is derived from the adenosine triphosphate found in the dynein arms of the axonemal tubules.[3]

Sneezing, a sudden, violent, involuntary expulsion of air through the nose, is a radical reflex for clearing the nasal cavity. It starts with a rapid inspiration that is followed by an audible expulsion of air through the nose. The reflex occurs after stimulation of sensory receptors in the nasal mucosa.

2.1.3 Olfaction

Three sensory systems are dedicated to the detection of chemicals in the environment: olfaction, taste, and the trigeminal chemosensory system.[40] In dogs and cats, the sense of smell is a very important sensory system. Olfactory information can influence feeding behavior, social interaction, and reproduction. A dog's sense of smell, together with its personality and intelligence, enables it to function as a nose for humans in many circumstances.

The transduction of olfactory information occurs in the nasal olfactory epithelium, a sheet of neurons and supporting cells that lines the caudolateral wall, the ethmoidal conchae, and the dorsal part of the nasal septum.[12, 16] The olfactory receptor neuron is a bipolar neuron that gives rise on its basal surface to an unmyelinated axon that carries the olfactory information to the brain. At its apex the receptor neuron gives rise to a single process that expands into a knob-like protrusion from which several microvilli, or olfactory cilia, extend into the thick layer of mucus that lines the nasal cavity and controls the ionic milieu of the olfactory cilia (Figure 2.1). Generation of receptor potentials in response to odors takes place in the cilia of receptor neurons. The axons of the olfactory receptor cells form the olfactory nerves that pass through the cribriform plate directly to the olfactory bulb, on the anteroventral aspect of the ipsilateral forebrain. Olfactory information is passed to the amygdala and primary olfactory cortex. Further pathways for processing olfactory information include the thalamus, hypothalamus, entorhinal cortex, and hippocampus.[40, 48]

2.1.4 Specific functional systems

Vomeronasal organ. The paired vomeronasal organ is located in the rostral base of the nasal septum as a tubular pocket of olfactory epithelium, partially enclosed by a scroll of cartilage. It plays a role in sexual behavior and in recognition of kin, via pheromones, some of which have been identified chemically. The neural pathways transmitting stimuli from the vomeronasal mucosa to the brain are distinct from those from normal olfactory mucosa.[14]

Nasolacrimal duct. This conducts the lacrimal secretion from the eye into the nasal vestibule via an orifice located at the external end of the attached margin of the alar fold. Obstruction of this duct, such as by a nasal tumor, causes tears to spill onto the face on the same side.[36]

2.2 History and clinical signs

2.2.1 History

The medical history in nasal disease usually reveals specific problems such as nasal discharge, sneezing, pain, or nasal obstruction. A loss of the sense of smell is rarely encountered

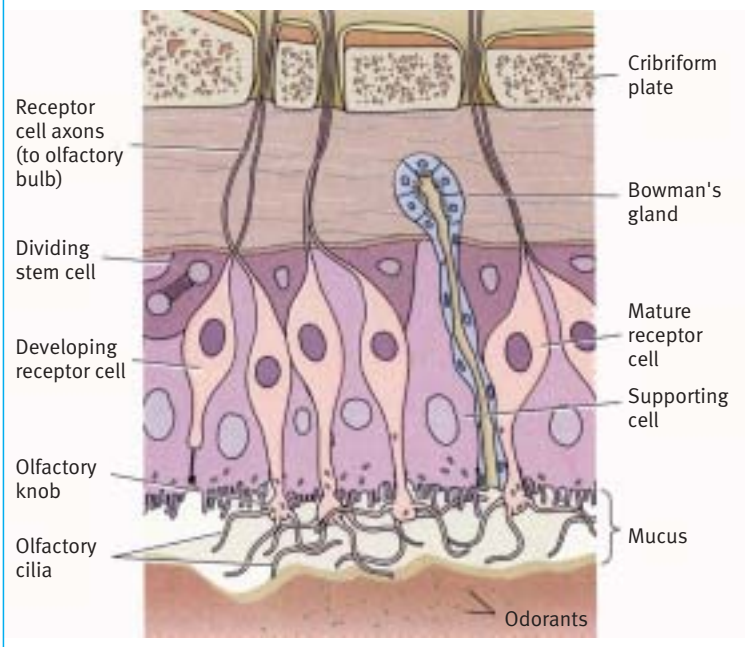


Figure 2.1: Diagram of the olfactory epithelium, showing the major cell types and projection of the olfactory receptor neurons to the olfactory bulb. Bowman's glands produce mucus and the supporting cells help to detoxify chemicals that come in contact with the epithelium. (From: Purves D, Augustine GJ, Fitzpatrick D, Katz LC, LaManta A-S, McNamara JO. The chemical senses. In: Purves D, Augustine GJ, Fitzpatrick D, Katz LC, LaManta A-S, McNamara JO, editors. Neuroscience. Sunderland: Sinauer Associates, Inc. 1997: 263–287, Fig. 14.5 A).

as a single sign in dogs or cats. Additional questions are asked about the animal's general condition, appetite, drinking, activity, and endurance, and about changes in its habits. This information is needed because abnormalities of the nasal mucosa not only result from nasal disease but sometimes indicate a systemic condition. The answers to these questions together with the results of a general physical examination will point the way to diagnosis.

2.2.2 Clinical signs

Nasal discharge can profoundly affect the status of the dog or cat in the household. Owners find the discharge of large amounts of serous fluid or purulent or bloody mucus to be extremely unpleasant, and neither the dog nor the cat is able to clean its nose sufficiently to avoid this attention. Bilateral nasal discharge usually indicates a more generalized disorder. Unilateral mucopurulent nasal discharge indicates unilateral rhinitis, which may be caused by local inflammation, a foreign body, an obstruction, a deformity, or unilateral mycotic infection. In both dogs and cats, bilateral mucopurulent nasal discharge is almost always a sign of viral disease and further examination will reveal more general signs of the disease.

The character of a nasal discharge is seldom pathognomonic for a specific disease. *Aspergillus* infection can be recognized, but not diagnosed, by a continuous stream of mucopurulent exudate containing some blood. In addition, toxin produced by the fungus causes depigmentation of the skin that is in contact with the discharge (Figure 2.2). Mucopurulent nasal discharge associated with recurrent nasal bleeding is aspecific and although it can occur in various nasal diseases, it indicates lesions in the nasal mucosa and is therefore more characteristic of locally invasive nasal disorders.

Figure 2.2: Golden retriever with Aspergillus infection in the left nasal cavity. The mucopurulent discharge containing some blood and the depigmentation of the skin that it contacts are indicative of Aspergillus infection. (From: Venker-van Haagen AJ. Diseases of the nose and nasal sinuses. In: Ettinger SJ, Feldman EC, editors. Textbook of Veterinary Internal Medicine. Elsevier Saunders; Fig. 210-4. Copyright 2005, with permission from Elsevier).

Sneezing, like coughing, is a reflex for protection of the respiratory mucosa. It is evoked by stimulation of the nasal mucosa. The afferent limb of the reflex is in the trigeminal system and the efferents are motor nerves for the respiratory system, including the larynx. The reflex is involuntary and starts with a deep inspiration that is followed by an explosive expiration through the nose. This reflex is essentially similar in humans, cats, and dogs and is therefore recognized and named by the owner as sneezing. It may be caused by mechanical stimuli, such as particles in the inhaled air or a foreign body, or inflammation, or either local drying resulting from abnormal turbulence in the nasal cavity or an extremely dry environment. Continuous sneezing can itself cause irritation of the mucosa, leading to more sneezing and sometimes epistaxis. The history will differentiate between sneezing that leads to epistaxis and that which occurs during the course of epistaxis.

Pain in the nose. Acute sneezing and rubbing of the nose may indicate acute pain in dogs and cats. It is usually not accompanied by abnormal nasal discharge but there may be a watery discharge. Further investigation is necessary to find the cause. Chronic pain in the nose may be

shown by an adverse reaction to the owner's customary petting of the animal's head, and a dog may object to having a collar or the loop of the leash drawn over its head, even though this is usually associated with the pleasure of a walk. Questions to obtain information about nasal pain should be adapted to the living conditions of the animal being examined.

Dyspnea in nasal obstruction. Dyspnea is the dominant sign in nasal obstruction. It is recognized by a nasal stridor or snoring or continuous open-mouth breathing. A nasal stridor is a soft, rustling or sniffing sound that is synchronous with inspiration or expiration or both. The sound is caused by narrowing of the passageway, which increases the velocity of the airflow.

When there is a unilateral nasal obstruction, the contralateral passageway is usually sufficient to sustain closed-mouth breathing at rest, but rapid opening and closing of the mouth can be observed during inspiration. Cats may simply slip air in via the openings at the caudal limits of the lips. In dogs dyspnea is not observed during a walk, since open-mouth breathing and panting are quite usual at that time. When there is bilateral nasal obstruction the dyspnea is severe and the spontaneous intake of oxygen is so low that if the animal does not breathe through its mouth, a tracheostoma may be needed to raise oxygen saturation to normal values. Both dogs and cats tend to avoid mouth breathing, even when obstruction of the nasal cavity is severe, almost to the point of suffocation. Apparently, avoiding the consequent bypassing of the nasal function of air cleansing and conditioning has a high priority.

Loss of the sense of smell. When there is severe obstruction of the nasal cavity, the dog or cat will obviously lose at least part of its sense of smell. However, this is rarely mentioned as a sign by the owner, the dyspnea and eventual nasal discharge being seen as dominating the clinical symptoms. Sometimes a dog's loss of the sense of smell is of direct consequence to the owner, and may be the sole problem. This usually concerns dogs used for their sense of smell and trained to work with their attendant to hunt, to detect drugs, to recognize human odors, etc. Physical examination and special diagnostic procedures may elucidate the diagnosis or at least exclude a number of causes.

2.2.3 Physical examination

After inspecting the overall shape of the animal's nose, the examiner listens to its respiration for the occurrence of nasal stridor. This should be done under quiet conditions, close to the dog's, and especially the cat's, nose, while gently closing its mouth. If there is a stridor caused by too narrow nostrils, its tone will be changed by moving the nasal alae laterally. The symmetry of the air stream is examined by watching the movement of a small fluff of cotton held before each nostril. At the same time, the odor of the expired air can be noted. The area around the nostrils is inspected for nasal discharge and crusts. The nasal plane is inspected for epithelial crusts, which could be caused by pathological dryness, and for epithelial lesions and depigmentation. The ventral wall of the nasal passages, which also forms the roof of the mouth, should be inspected through the opened mouth. The teeth, especially the canine teeth, should be inspected at the same time, because dental abnormalities can cause disorders of the nose.[10, 48] More in-depth inspections belong to special diagnostic procedures and require anesthesia.

2.3 Special diagnostic techniques

Special diagnostic procedures are important in the diagnosis of nasal disease and diseases of the paranasal sinuses. Radiographs give information about the extent and the character of

the disorder. Especially in trauma, obstructive nasal disease, and mycotic rhinitis and sinusitis, the changes may be sufficiently specific for a final diagnosis. When surgical treatment is planned, CT and MRI provide the necessary additional details of the structures involved in the disease. Rhinoscopy provides information about the deformities caused by the disorder and affords the opportunity for biopsies under visual guidance.

2.3.1 Diagnostic imaging

The standard radiographic examination of the skull consists of a lateral and a dorsoventral projection. These radiographs may provide all the information that is needed or they may serve as a primary inventory examination. These standard projections are usually of limited value in examination of the nose and nasal sinuses, and special projections, such as rostrocaudal radiographs, open-mouth projections, or radiographs with intraoral film, are required.

Radiography of the skull demands the most stringent and careful radiographic technique. Any unintended obliquity in positioning will hinder evaluation of the radiographs and thus deep sedation or general anesthesia is mandatory. When the diameter of the skull exceeds 10 to 12 cm, a grid must be used to diminish the unfavorable effect of scattered radiation on radiographic quality. A grid is not used when part of the skull is radiographed in open-mouth projections or when radiographs are made with intraoral film. Both nonscreen film in a lightproof envelope for intraoral application and screen film in a cassette with intensifying screens should be available. Nonscreen films require a much longer exposure time but produce radiographs with greater detail.

Radiographs provide a complete overview of all structures of the part of the skull being examined, with high spatial resolution. However, superimposition of structures on the film may make it difficult and sometimes impossible to distinguish a particular detail, especially when structures differ only slightly in density.[48]

Computed tomography (CT) is based on differences in attenuation of an x-ray beam in different parts of the body. With the x-ray beam collimated to a narrow fan shape, the x-ray tube revolves around the object during the exposure and the beam is altered as it penetrates the object. An array of sensitive detectors on the opposite side of the object quantitates the x-rays passing through, thereby determining the x-ray attenuation in different parts of the object, in all projections. Computer analysis of this collection of attenuation measurements results in a cross-sectional image of the object, which is displayed on a monitor with high spatial resolution and higher contrast resolution than provided by conventional radiographs. An intravenously administered radiographic contrast medium will increase the contrast between normal and abnormal tissue, and facilitate the recognition of blood vessels. Since the images represent slices of the object, they do not suffer from superimposition, but they also do not provide a survey view.

Positioning of the patient is no less important in CT than in conventional radiography. The position of the object within the gantry will determine the scan plane. The patient support is perpendicular to the opening of the gantry, and thus perpendicular to the scan plane. CT of the body is thus always axial, perpendicular to the long axis of the body, with some adjustment possible through angulation of the gantry relative to the patient support. Other scan planes can be achieved if the object can be positioned differently relative to the gantry opening. This is important, because interfaces between organs or structures can only be imaged satisfactorily when they are perpendicular to the scan plane. The head in particular can easily be examined

in different scan planes. Transverse (coronal) scans of the head are made with the animal in prone or supine position and the head extended. Dorsal (axial) scans are made with the animal in supine position and the nose pointing upward. Sagittal scans are made with the animal in lateral recumbency and the head raised on a support.[48]

Magnetic resonance imaging (MRI) is based on the magnetic properties of atomic nuclei which have an odd number of protons. Since protons are in a continuous state of rotation, called the nuclear spin, and have an electrical charge, they may be thought of as tiny magnets. Because the hydrogen nucleus consists of a single proton and hydrogen is abundant in living tissues, it is eminently suitable for magnetic resonance imaging.

In the body, the magnetic moments of the protons point in all directions, thus canceling each other. When the body is placed in a strong, homogeneous magnetic field (in clinical imaging, usually between 0.15 and 1.5 Tesla, 1 Tesla being 10,000 times the strength of the earth's magnetic field), the protons are forced into positions parallel to the axis of this magnetic field, not only spinning around their own axes, but also precessing around the axis of the magnetic field, like a child's spinning top. The frequency of precession is called the Larmour frequency and it depends on the strength of the external magnetic field.[48]

Using radio waves of the same frequency as the precession frequency, the protons can be made to resonate: they will precess around the axis of the external magnetic field at a larger angle and all protons will be in phase. When the radio frequency wave is switched off, pulse relaxation occurs through two phenomena: the protons realign in the magnetic field (T1-relaxation) and they go out of phase (T2-relaxation). During the process of relaxation the protons emit weak radio signals and it is these signals that are used to create the images. The images may be dominated by the concentration of protons (proton density), by T1-relaxation (T1-weighted images), or by T2-relaxation (T2-weighted images).

By using gradient coils to create small variations in the x, y, or z direction of the external magnetic field, certain scan planes and slices can be selected in which the precession of protons has exactly the right frequency to be susceptible to the radio wave pulses, and images can be made in different scan planes without having to reposition the patient. These images are influenced by the concentration of hydrogen nuclei in the part of the body being examined, by the chemical nature of the environment around the nuclei, and by the interactions between nuclei. MRI provides detailed anatomical images with soft tissue contrast that is superior to that of CT. The soft tissue contrast may be further enhanced by the intravenous administration of a contrast medium which is a paramagnetic substance (usually gadolinium). Its predominant effect is to shorten T1 and thus the regions that take it up are bright on T1-weighted images.[48]

2.3.2 Rhinoscopy

Rhinoscopy is performed following radiography of the nose and nasal sinuses, its purpose being to explore the nasal cavities for the abnormalities indicated by the radiographs. A lesion can be confirmed, a foreign body can be removed, and biopsies and material for cultures can be taken under visual guidance.

Procedure. For rhinoscopy the dog or cat must be anesthetized. After premedication with medetomidine, anesthesia is induced and maintained with propofol intravenously to effect. The anesthesia should be appropriate for surgery and an endotracheal tube should be in place. Monitoring of the ECG and the capnogram is recommended. Pain and vagal stimula-

tion are to be expected and may complicate the procedure. Monitoring of the depth of anesthesia is necessary. The animal is placed in sternal recumbency with its head supported by a pad high enough to extend the neck and position the nose in a horizontal plane. The table is elevated such that when the endoscopist sits facing the animal the view through the rhinoscope is level with the animal's nasal openings. The procedure begins, however, with a thorough inspection of the mouth and oropharynx. The mouth is inspected for any abnormality that could be associated with the nasal disease, such as dental root infection or a deformity or lesion in the roof of the mouth, which is the floor of the nasal cavity. In the oropharynx the soft palate is inspected for length and for flexibility in the region of the nasopharynx. The soft palate should be flexible enough to be moved upward into the nasopharyngeal cavity.

Equipment. A simple otoscope can be used for rhinoscopy. It should be of good quality in that a strong light source and dark specula are needed to illuminate at least the very rostral part of the nasal cavity. The nasal opening is approached from the lateral side at an angle of 45° to the vertical part of the nasal plane. The tip of the speculum is placed near the lateral part of the opening and the speculum is then introduced into the nasal opening by slowly turning the line of vision of the otoscope into a position parallel to the nasal cavity. This movement is necessary to push the nasal ala, which obstructs the opening, into a lateral position. The first view through the otoscope is often disappointing, for nasal disease usually causes discharge and the tip of the speculum is thus immediately blocked by mucus. The lens of the otoscope can be slid to one side, creating an opening for a small suction cannula, while still allowing vision through the lens. Under vision the mucus can be removed and the rostral part of the nasal cavity can then be examined. The use of an otoscope is often preferable for detection and removal of foreign bodies which enter the nasal cavity via the nasal opening. A foreign body forceps developed for use through an otoscope under visual guidance can be helpful.

A telescope is needed for complete examination of the nasal cavity. The basic equipment consists of a light source, a flexible fiberoptic cable, and a suitable telescope, for which a 25°-vision rigid telescope with a diameter of 2.7 mm and a length of 15 cm can be recommended. The best instruments have a wide-angle lens, which is important for orientation and as well as aiding the examination. This telescope is satisfactory for most dogs and cats, but for very small cats a 1.2 mm-diameter telescope is often needed. In most dogs and cats a suction cannula (size 6) or a foreign body or biopsy forceps can be introduced alongside the 2.7 mm diameter telescope.

A single-outlet light source can be used for rhinoscopy but a combined light source and electronic flash generator is needed to obtain photographic images. The camera should be adapted to the telescope and the timing of the flash. For teaching purposes a video recorder with a chip camera is a great asset.

Further equipment for rhinoscopy consists of several suction cannulas (size 6 and smaller), a vacuum suction device, a selection of biopsy forceps, and a small dropper bottle of 0.1 % adrenalin solution to stop profuse bleeding (do not use more than 1 or 2 drops at a time).

Examination. The inspection of the nasal cavity is guided by anatomical borders. These are the nasal opening rostrally, the nasal septum medially, the dorsal roof of the nasal cavity, and ventrally the floor of the nasal cavity. The cribriform plate is part of the caudal boundary, the other part being the ventrally positioned opening to the nasal pharynx, called the choanae. The endoscopic procedure aims at bringing the greater part of the nasal cavity into vision. The procedure is limited by the conchae, if not by the pathological process. Careful maneuvering

and patient repositioning of the telescope will result in a reliable impression of the normal and pathological structures in the nasal cavity.

The normal nasal cavity in dogs and cats contains the conchae, which are covered with mucosa. Viewed through the rhinoscope they are immediately in vision rostrally. On the bottom of the nasal cavity medially, a narrow space will be found to lead to the nasal pharynx. For further examination the telescope follows the narrow spaces between the conchae. In the caudal part, the mucosa is normally more red and blood vessels are abundant and clearly visible in the mucosa. The divisions of the conchae are multiple and irregular. There is no place in the normal nasal cavity where conchae are not in view.

Abnormalities in morphology of the nasal cavity may come to attention when a young dog or cat has obstructive nasal disease or chronic rhinitis. The radiographs will reveal an abnormal configuration and rhinoscopy excludes other abnormalities. If there is obstruction, surgical intervention is indicated.

Foreign bodies usually cause unilateral nasal discharge, often of sudden onset. Radiographs can only distinguish radiodense material decisively, whereas plant material is the most common foreign material in the nasal cavity. During rhinoscopy, mucopurulent exudate will at first conceal the foreign material. Thorough aspiration will reveal focal inflammation and the foreign body, more or less embedded in the inflammatory tissue. Removal is not difficult once the view is clear. Foreign material inhaled and accumulated in the nose may be more difficult to remove completely. Alternately flushing and aspirating sometimes helps.

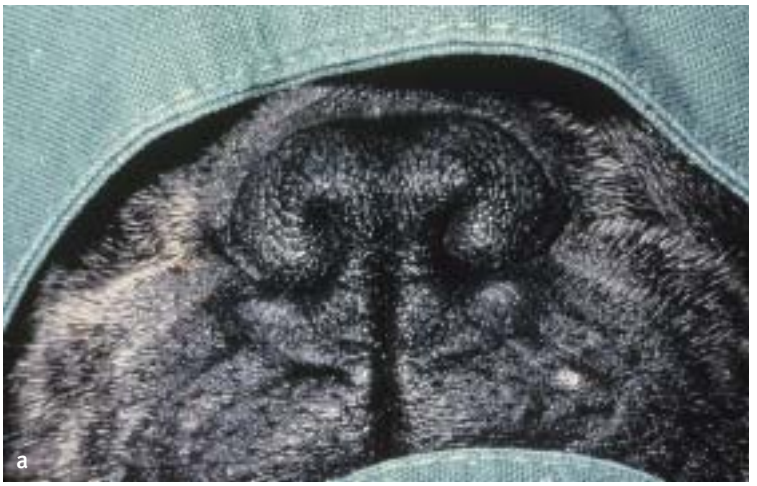
A tumor in the nasal cavity will usually be diagnosed by radiographs, which will show increased density together with loss of structures unilaterally or bilaterally. When radiographs suggest tumor and confirmation is wanted, rhinoscopy will reveal obstructive tissue between the normal conchae, often of a different color, and a biopsy for histological examination can be obtained to confirm the diagnosis.

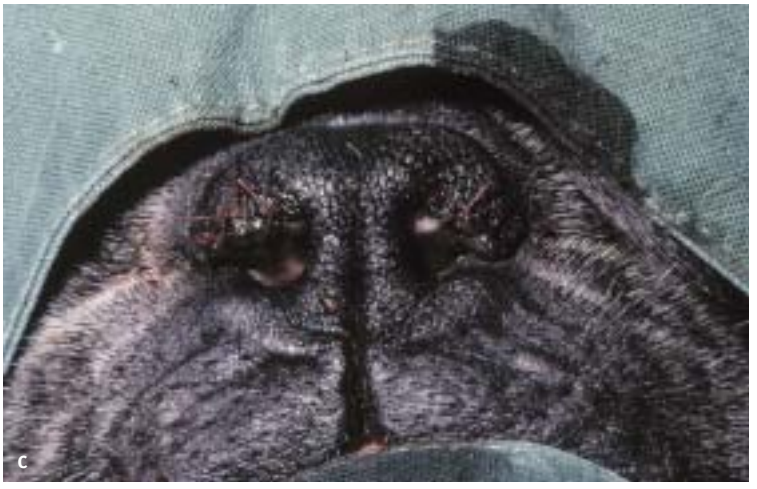
Proliferative rhinitis and polyps are most often seen in cats but may also occur in dogs. Biopsy of the obstructive tissue reveals that it is inflammatory tissue. Surgical exposure to remove the polyps involves removal of most of the conchae as well. Rhinoscopic removal is not advised, for it is often incomplete and bleeding cannot be controlled.

Atrophy and hypertrophy usually involve both the mucosa and the cartilaginous structures of the conchae. The abnormal appearance is clear, but the underlying cause of the disease remains unclear. Bacterial culture may indicate a specific infection, e.g., *Bordetella* in cases of severe atrophy. A very special opportunity for rhinoscopic diagnosis is presented by *Aspergillus* »plaques« in the caudal part of the nasal cavity. Because of the marked atrophy of the conchae in the area around the fungal growth, a real cavity is formed and the fungus is easily recognized. When it is important to know the exact species of the fungus, a biopsy of the plaque can be taken for culture.

2.3.3 Olfactory tests

There is no simple method to study olfaction in dogs and cats. The sense of smell should be tested by activation of the olfactory receptor neurons and determination of the brain response by electroencephalographic olfactometry analysis. Some authors have reported results in dogs presumed to have normal olfactory function and in others which have lost the sense of smell.[20, 31, 32]





2.4 Congenital diseases of the nose and nasal sinuses

2.4.1 Congenital malformation of the nasal plane

Congenital malformation of the nasal plane is a common finding in brachycephalic breeds. The cartilage supporting the nasal plane is soft and thus the alae collapse, narrowing the nostrils. This also occurs in Persian cats but is most often seen in brachycephalic dogs. Not all owners recognize that the narrowness of the openings of the nostrils is a severe handicap for the dog, because these dogs have additional congenital abnormalities causing upper airway obstruction, and owners are told that snoring characterizes the breed. The narrowed nostrils do not, however, cause snoring but a softer, rustling or sniffing sound that is synchronous with inspiration or expiration or both. The alae of soft cartilage are drawn inward toward the nasal septum during inspiration, which virtually closes the nostrils. During expiration the obstruction also causes a soft stridor, often with the escape of bubbles of mucus. In longstanding obstruction of the nose the dog becomes emaciated, because mouth breathing is not readily exploited and eating, during which the dog needs repeated rapid inspirations, takes more time and effort. The increased respiratory activity also uses more energy, especially when inspiration is obstructed. Another effect of the nasal obstruction is that the dog's sense of smell is diminished. This be-

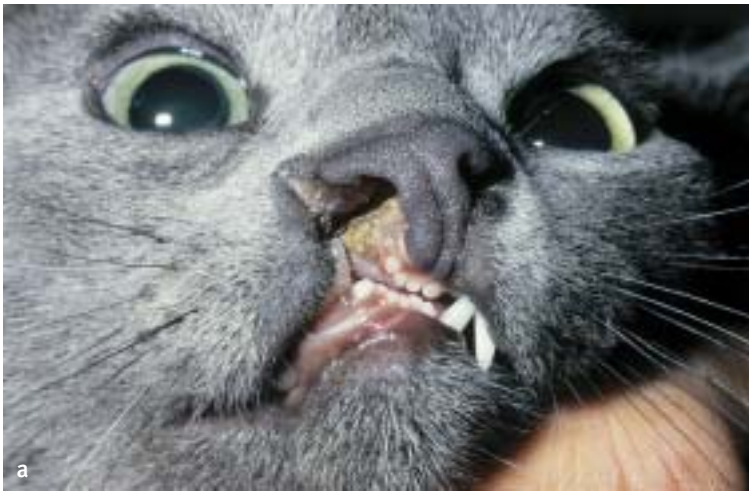
Figure 2.3 a–c:
Stenotic nares in a French bulldog (a). Using a Beaver no. 65 pointed scalpel, a cone-shaped wedge is excised from the right ala. The first suture is placed where it will be most effective in opening the naris (b). The wound is then closed with simple interrupted sutures and the procedure is repeated on the left ala (c). (From: Venker-van Haagen AJ. Resection of stenotic nares. In: van Sluijs FJ, editor. Atlas of small animal surgery. Wetenschappelijke uitgeverij Bunge, Utrecht, 1992, Fig. I-1, I-2, with permission from Reed Elsevier).

comes apparent when, after surgical widening of the nostrils, the dog's interest in the odors of its environment is quite obviously increased. Dogs are macrosomatic and olfactory information is very important in their awareness.

Clinical examination. During the clinical examination, moving the alae laterally slightly with the thumb will change the sound of the stridor, indicating its cause. The clinical examination should be extended to include all other possible sites of upper airway obstruction as well as the lungs. It is not always necessary to shorten the soft palate in these dogs, and widening the nostrils may decrease the dyspnea to a sufficient and acceptable degree. When radiographic examination reveals no unacceptable risks for anesthesia, surgery can proceed. During the induction of anesthesia, the relative length of the soft palate, the width of the pharynx, and the size of the larynx can be recorded for later reference. After endotracheal intubation the dog is placed in sternal recumbency with its head resting on a pad high enough to raise the nostrils to a convenient level. The aim of surgery is to greatly widen the nostrils. The ala on one side is moved laterally with the thumb and the part to be removed is incised from the upper medial border to the lowest medial border and then along the ventral border to the lateral limit. The point of the scalpel (Beaver, 65) is then inserted at the beginning of the incision and a cone of tissue is excised, the base of which has just been outlined by the incision. Blood is removed but coagulation is not used. The ventral edge of the wound is sutured to the dorsolateral edge, using interrupted sutures of absorbable material, such that the nostril is widened substantially (Figure 2.3 a–c). The bleeding stops after the first 2 or 3 sutures have been tied. The contralateral nostril is then widened in the same manner. Aftercare consists of rest, analgesics, and broad-spectrum antibiotic treatment for 5 days. Immediate relief of the dyspnea caused by the nasal obstruction can be expected. If also required, the surgical procedure to shorten a too-long soft palate is described in Chapter 3.4.3.

Various congenital malformations of the nasal plane and more extensive clefts can be repaired surgically. The success of surgery is largely dependant on the available tissue around the cleft. A cleft in the nasal plane usually results in not only a remarkable facial deformity but also drying of the exposed mucosa, causing crusts, irritation, and rhinitis (Figure 2.4 a). Surgical repair should aim at remodeling of the nasal plane and protection of the mucosa, while adequately maintaining the air passage through the nostrils (Figure 2.4 b).

Figure 2.4 a, b: Congenital malformation of the nasal plane in a cat. The lesion causes not only a remarkable facial deformity but also drying of the exposed mucosa, resulting in crusts, irritation, and rhinitis (a). After surgical remodeling of the nasal plane, air passage through the nostrils was adequate (b).



2.4.2 Nasal dermoid sinus cysts

Nasal dermoid sinus cysts are not uncommon in dogs.[1] The cyst is presented as a small fistula in the midline of the bridge of the nose, intermittently producing discharge. Exploration by circumferential dissection of the fistula usually reveals that a larger area is involved subcutaneously, with epidermal and adnexal tissue and especially hair, continuing within the nasal septum. The nasal dermoid sinus cyst should be removed completely. The nasal dermoid sinus is a congenital neural tube defect. In humans these cysts may have intracranial extensions and thus CT and MRI are performed routinely before surgery.[25]

2.4.3 Congenital cerebrospinal fluid fistula

A congenital cerebrospinal fluid fistula has been reported in a cat.[28] At intervals of 6 weeks since the age of 5 months there had been episodes of bilateral clear nasal discharge together with the development of a lump, representing a cyst, on the forehead. The cyst would rupture spontaneously, releasing clear liquid, and then the nasal discharge would cease. The cat was examined at the age of 15 months and radiographs and a fistulogram were made. The dye entered the right cerebrospinal cavity of the olfactory bulb and the lateral ventricles of the brain, filling the ventricular system and creating a myelogram. The resulting diagnosis was olfactory bulb cavity nasal-cutaneous fistula with cerebrospinal rhinorrhea. The fistula was closed successfully.[28]

2.4.4 Congenital malformation of the frontal sinuses

The frontal sinuses vary in relative size, being larger in dolichocephalic breeds and smaller in brachycephalic breeds. Sometimes both frontal sinuses are abnormally small or even absent. Their absence is not associated with clinical signs but is usually found when radiographs of the skull are made for another reason.

2.4.5 Congenital ciliary dysfunction

Congenital ciliary dysfunction has been documented in dogs of various breeds.[13] Primary ciliary dyskinesia is a disorder in which ciliary function is ineffective and uncoordinated. Cilia are complex structures lining various organs, including the upper and lower respiratory tracts, auditory tubes, ventricles of the brain, spinal canal, oviducts, and efferent ducts of the testis. The combination of ciliary dysfunction (sinusitis, bronchiectasis) and situs inversus is known as Kartagener's syndrome. Cilia are thin, longitudinal extensions from the free surface of the cell, encased by the cell membrane.[13] During an arc-like effective stroke, they move in an extended planar fashion, contacting, propelling, and then dropping below the overlying mucus layer into the periciliary fluid. The recovery stroke occurs within the periciliary fluid in a side-arm fashion. Coordinated beating of adjacent cilia moves the mucus blanket in the respiratory tract and auditory tubes toward the pharynx. Ultrastructural lesions in the respiratory cilia and sperm flagella in persons with Kartagener's syndrome were described as defects in the dynein arms. The diagnosis was based on electron micrographs of transverse sections of respiratory cilia. The diagnostic significance of these lesions has been contested on the basis of the difficulties inherent in electron microscopy of cilia, the presence of ciliary lesions in clinically normal persons, and the production of ciliary lesions by other disorders such as bacterial or viral infections, smoking, and asthma. Despite the controversy, several distinct ultrastructural abnormalities, when present in sufficient numbers of cilia, are believed to be diagnostic for primary ciliary dyskinesia.[13]

Mucociliary clearance in the dog's nasal cavity can be measured by placing a drop of ^{99m}Tc macroaggregated albumin deep in the cavity via a catheter, beyond the nonciliated rostral half. The velocity of mucus clearance ranges from 7 to 20 mm/min.[13] The test is not affected by anesthesia. Not all normal dogs have a »normal« clearance rate and inflammation can change the velocity of the ciliary beat. To avoid spurious values, the test should be repeated and performed bilaterally.

Chronic rhinitis is present in most if not all dogs with primary ciliary dyskinesia. The rhinitis begins at the age of a few days to 5 weeks, but some dogs have remained asymptomatic for months. Complications are caused by colonization of the mucosa and the conchae by *Pasteurella multocida* and *Bordetella bronchiseptica*, which can cause atrophy of conchae via bone resorption. Disease of the lower airways of dogs with primary dyskinesia varies from mild bronchitis and bronchiolitis to severe bronchopneumonia with bronchiectasis and ventral lung lobe consolidation.[13] The prognosis is reserved. Affected dogs that develop severe recurrent pneumonia eventually die of this. Continuous treatment with broad-spectrum antibiotics will prolong survival. Cultures should be repeated to change the antibiotics on the basis of sensitivities of the bacteria involved.

2.5 Rhinitis and sinusitis

2.5.1 Infectious rhinitis and sinusitis

Viral upper respiratory disease in cats

Rhinitis and sinusitis are symptoms of viral upper respiratory disease, a prominent disease in cats. Feline herpesvirus-1 and feline calicivirus are the most prevalent and virulent respiratory pathogens of cats and account for at least 80 to 90 % of infectious upper respiratory disease in cats.[8, 17] The introduction of modified live virus vaccine against these two viruses has substantially decreased mortality and morbidity but has not eliminated the diseases. It is important to know that immunization against these viruses protects the cat from development of severe disease, but not from infection.[2] Because these viruses can spread rapidly among kittens and the prevalence of chronically infected virus carriers is high, elimination of the disease is not feasible. It is estimated that 80 % of cats recovering from acute infection become chronic carriers. The predominant route of infection is by direct cat-to-cat contact.[17] The chronic carrier state may develop subsequent to infection with either feline herpesvirus or calicivirus, and also occurs in vaccinated cats. Although cats carrying feline herpesvirus do not necessarily shed virulent virus continuously, they should be considered infectious when they are sneezing and have nasal discharge. Calicivirus carriers shed virulent virus continuously from the oropharynx. They may have no clinical signs or mild nasal discharge, gingival ulceration, and periodontitis.

Feline viral upper respiratory disease is most common in cat colonies and occurs most often in kittens under 10 weeks of age.[17] The disease is only occasionally fatal in kittens younger than 10 weeks of age, but the morbidity is high in an affected colony.

Clinical signs and etiology. The initial clinical signs are paroxysmal sneezing, conjunctivitis, and serous ocular and nasal discharge. About 5 days after the onset of sneezing, the nasal discharge becomes mucopurulent and there may be ocular complications, such as conjunctivitis, chemosis, blepharospasm, keratitis, and symblepharon. The disease persists for 2 to 3 weeks, but many recovered cats become chronic carriers.

Feline herpesvirus produces a latent infection that lasts several months to even years. Viral replication and shedding is intermittent. Stressful episodes and glucocorticoid adminis-

tration can induce shedding of virulent virus in carrier cats. Adult cats shedding feline herpesvirus may develop rhinitis and sneezing in this period. In contrast, chronic calicivirus carriers shed virulent virus continuously from the oropharynx and are therefore a continuous threat to susceptible kittens. Most carrier cats do not show clinical signs, but when they do, gingival ulcerations, periodontitis, and nasal discharge are predominant.

In clinical practice there is probably little value in distinguishing calicivirus infection from herpesvirus infection. Clinical diagnosis of feline viral upper airway disease is made on the basis of the history and physical examination. Identification of carriers may be important when there are repeated outbreaks of the disease in colonies. For this purpose, a swab from the oropharynx can be used to inoculate appropriate transport media for submission to a specified laboratory for viral culture.[17]

Treatment in acute feline viral upper respiratory disease consists of supportive therapy designed to meet nutritional and fluid requirements. Treatment of chronic carriers is limited to palliative therapy intended to relieve clinical signs. Short-term broad-spectrum antibiotic treatment (e.g., for 5 days) will relieve the signs of rhinitis to the extent that they are caused by secondary bacterial infection. This therapy can be repeated when the rhinitis recurs some months later and is also appropriate for intermittent rhinosinusitis.

Figure 2.5: A 5-month-old Samoyed with bilateral mucopurulent nasal discharge due to canine distemper. Viral rhinitis is a prominent clinical sign of canine distemper. (From: Venker-van Haagen AJ. Diseases of the nose and nasal sinuses. In: Ettinger SJ, Feldman EC, editors. Textbook of Veterinary Internal Medicine. Elsevier Saunders, Fig. 210-3. Copyright 2005, with permission from Elsevier).

Viral rhinitis

Viral rhinitis is a prominent clinical sign of canine distemper (Figure 2.5). In countries where stray dogs are limited and veterinary care is adequate, vaccination has reduced the occurrence of the disease to sporadic cases. Herpes infection in newborn puppies is characterized by profuse mucopurulent nasal discharge. The diagnosis is usually made at necropsy.

Primary bacterial rhinitis

In dogs a specific primary bacterial infection occurs within the nostril (usually unilaterally) and the adjacent rostral part of the nasal cavity. It is recognized as a painful infection with mucopurulent discharge and swelling, together with crust formation on the nasal plane around the nostril. Rhinoscopy reveals inflammation in and directly caudal to the nostril, while the mucosa of the conchae and nasal cavity caudal to the lesions is normal. Culture of the discharge usually reveals *Staphylococcus aureus*. Treatment consists of systemic broad-spectrum antibiotics and vitamin A ophthalmic ointment applied several times daily in and around the nostril to prevent the formation of painful crusts.

Primary bacterial rhinitis is uncommon in both dogs and cats, but bacterial rhinitis can develop secondary to viral rhinitis in cats, or foreign bodies in dogs or cats, or many other disorders in which there is disruption of normal mucociliary mucosal integrity.

Mycotic rhinitis

Mycotic diseases involving the nasal cavity, the frontal sinuses, and the nasal plane occur in both dogs and cats. In dogs the most prevalent mycosis in the nasal cavity and frontal sinuses is that caused by *Aspergillus*. This fungus is also found rarely in the nasal cavity in cats.

Cryptococcus neoformans is a more frequent cause of nasal disease in cats, and in certain areas it is even quite common; it also causes nasal infections in dogs. *Alternaria* may infest the nasal plane in cats, causing proliferation of the skin and thereby dyspnea.[48]

Aspergillosis. Members of the genus *Aspergillus* are considered to be opportunistic, producing infections in man and animals especially when resistance to infection is reduced or when large numbers of spores are present. Spores of *Aspergillus fumigatus* are present on household plants, on furniture made of plant material, around bird cages, and simply in house dust.[46]

In dogs *Aspergillus* plaques are usually found in the caudal part of the nasal cavity or in the frontal sinus. They are presumed to represent primary infections. The toxins produced by the fungus cause atrophy of the conchae around the plaques and severe destruction of the mucosa and underlying structures in the entire nasal cavity and frontal sinus. There may be periostitis and resorption of the frontal bones and involvement of the internal surface of the frontal bone may open the way to the brain. The disease may spread bilaterally, destroying all internal and external bony structures, as well as the orbit, the nasal septum, and the nasal plane. *A. fumigatus* was identified in 25 of 27 of our cases involving the sinus and related structures.[46] Disseminated aspergillosis caused by *A. terreus*, not originating from the airways, has also been reported.[9, 49] Aspergillus can be associated with longstanding traumatic changes in the mucosa, caused by persisting foreign bodies in the nasal cavity or by oronasal fistulas. These infections are presumed to be secondary to the trauma.[48]

Clinical signs. The clinical signs of aspergillosis in the nose and frontal sinus are dominated by profuse mucopurulent nasal discharge and nasal pain. Depigmentation of the nasal plane below the nostril from which there is discharge is a characteristic sign (Figure 2.2). In addition to intermittent hemorrhagic discharge, profuse nasal bleeding is not exceptional. When only the frontal sinus is involved, hemorrhagic discharge or profuse bleeding from the nose may be the only sign. The nasal infection is often unilateral at first, becoming bilateral later. Aspergillosis is often not suspected in its initial stage and histories of nasal discharge that has been present for months are not uncommon. There is no apparent correlation between the duration of the initial nasal discharge and the severity or progression of the signs by the time of diagnosis. Other factors such as the number of infecting spores and the resistance of the host may play a role. Depression is a prominent sign when the frontal sinus is infected.

Aspergillosis as the cause of rhinitis is diagnosed by the finding of fungus plaques and culturing of the fungus. Recognition of the fungus plaque is facilitated by the atrophy of the conchae around it, which reduces the normal obstruction to rhinoscopic vision in the caudal part of the nasal cavity (Figure 2.6). The extent of the destruction in the nasal cavities and the frontal sinuses is well demonstrated by radiography, CT, or MRI, but the diagnosis depends on finding the fungus (Figure 2.7 a–c). When the fungus is in the frontal sinus alone, radio-

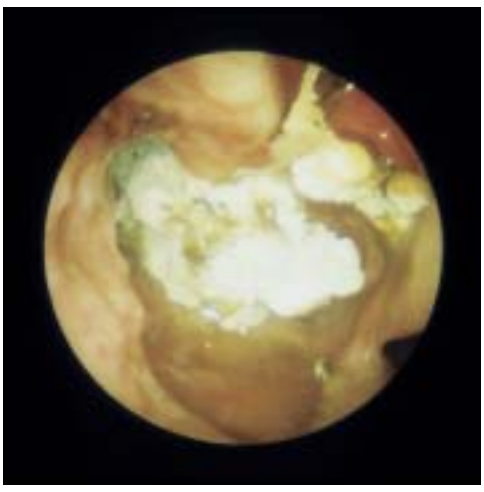
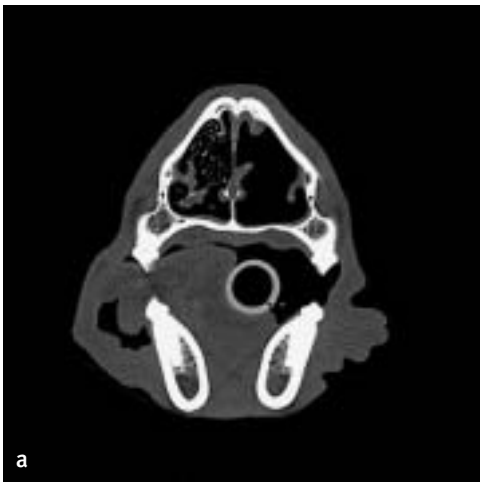
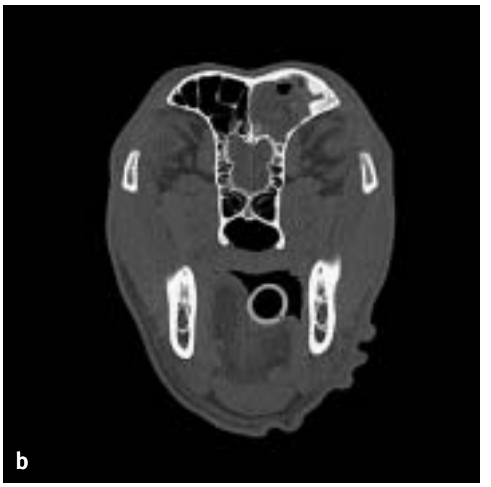


Figure 2.6: Rhinoscopic view of a fungal plaque. Its recognition is facilitated by the atrophy of the conchae around it, which reduces the normal obstruction to rhinoscopic vision of the caudal part of the nasal cavity.





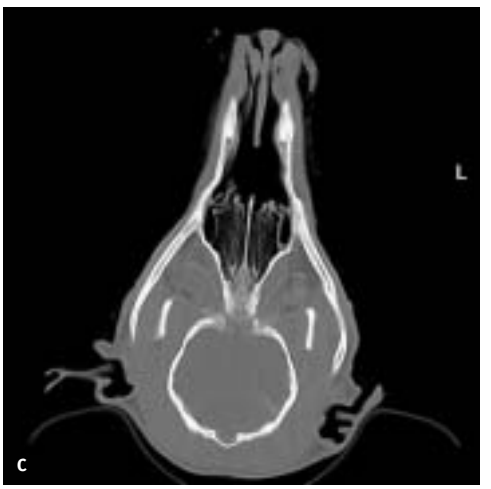


Figure 2.7 a–c: CT showing destruction in the nasal cavity and the frontal sinus in a dog with Aspergillus infection. (a) The conchae have disappeared from the left nasal cavity. (b) In the left frontal sinus the bony structures have been replaced by a soft tissue mass. (c) The horizontal projection shows destruction of the nasal septum but an intact cribriform plate. (Courtesy of Dr. G. Voorhout, Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University, and Dr. G. ter Haar, Division of Surgery, ENT, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University).

graphs reveal irregular soft tissue densities in the frontal sinus, thickening of the wall of the sinus, and sometimes reaction of the periosteum. CT scans then reveal the severity of further bony involvement. A 3 to 4 mm diameter trephined opening in the frontal bone on the side of the affected sinus can expose the fungus as thick, grayish-yellow material, sometimes with greenish lumps. This material should be removed and cultured. Cultures from nasal discharge are often negative because the fungus is usually located in the caudal part of the nasal cavity and is not shed in the nasal discharge. A serological test is available for detecting antibodies to *Aspergillus* in serum.[48]

Therapy. The treatment of sinonasal aspergillosis is topical or systemic. Topical treatment is usually with enilconazole or clotrimazole. The choice between them is arbitrary, since there has been no satisfactory clinical trial to compare them. The ongoing discussion is how to apply a suspension of the drug and how long the treatment should be continued. In order to irrigate the frontal sinus and the nasal cavity, for example, tubes can be introduced through trephine holes (Figure 2.8). Experience in about 120 cases (an average of 10 per year) indicates that administration of 10 cc of a 10 % suspension of enilconazole per tube twice daily for 14 days is sufficient. It is unpleasant for the dog because enilconazole has a bitter taste which most dogs dislike intensely. A retrospective study of these cases treated at the Utrecht University Clinic for Companion Animals revealed success in about 95 % in over 100 cases.[21] Hence 90 % success can be expected in treatment of a smaller number of cases. A less unpleasant systemic treatment would be much preferred, but the success rates and observed toxicities of other methods are not yet encouraging.[48]

Clotrimazole therapy is reported to have been successful in 52 of 60 dogs.[29] After premedication with a combination of neuroleptics,

anesthesia was induced with a short-acting injectable agent and then maintained with isoflurane and oxygen via an endotracheal tube. The dog was placed in sternal recumbency if infusion catheters were to be inserted into the frontal sinuses via an opening in each frontal bone, and in lateral recumbency when this was omitted. The nasal cavities were closed off by inflating one Foley catheter in the nasal pharynx, introduced via the oropharynx, and one in each nasal cavity, introduced via the nostril. Clotrimazole was administered as a 1 % solution, divided equally between 2 catheters when only the nasal cavities were treated and among 4 catheters when the frontal sinuses were also treated. During the 1-hour infusion the dog's head was repositioned to allow the clotrimazole to contact all mucosal surfaces. Nasal discharge ceased 2 weeks after treatment in most of the dogs. Two dogs were euthanized because of serious neurological signs which developed soon after the treatment.[29]

The systemic treatment of nasal aspergillosis has been reported but the results were less satisfactory than those of topical treatment. Of the systemically used azoles, fluconazole and itraconazole, the former seems to be less hepatotoxic in dogs. If there are lesions of the cribriform plate or the boundaries of the frontal sinus, so that there is a risk that topically applied antimycotic suspension may reach the orbit or the brain, systemic treatment is preferred to local treatment and can be used successfully.

Cryptococcosis is found as a cause of obstructive rhinitis with mucopurulent discharge in dogs and cats. Cats may also have crusts on the nasal plane and the bridge of the nose, and some develop mucopurulent conjunctivitis. In fresh material from the nose placed on a slide and stained with India ink, *Cryptococcus* organisms are recognized as thick, encapsulated, round to oval yeasts. They can be cultured on Sabouraud's agar. Ketoconazole, itraconazole, or fluconazole can be used for therapy, which should be continued for 8 weeks.

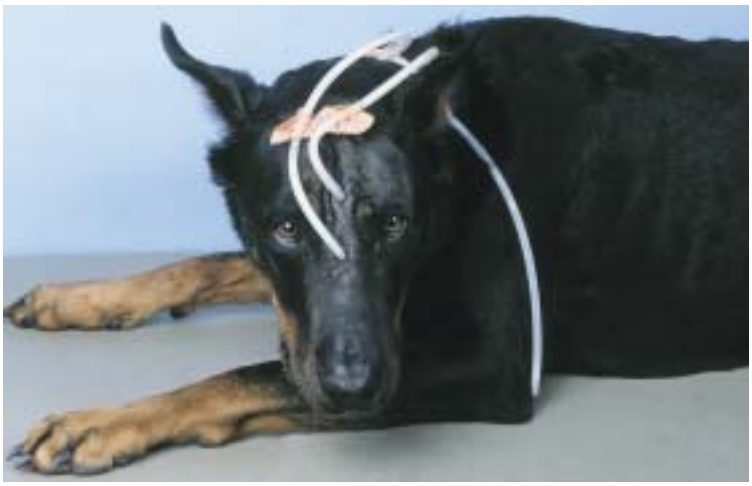


Figure 2.8:
Doberman pincher under treatment for nasal aspergillosis. Tubes have been introduced through trephine holes for irrigation of the left frontal sinus and the left nasal cavity.

Alternaria is found to cause granulomatous infections with crusts on the nasal plane in cats (Figure 2.9). Antimycotic treatment may be disappointing and removal of the nasal plane, as for squamous cell carcinoma, can be a satisfactory solution.[30]

Rhinosporidiosis has been reported to occur in dogs.[8] It is a noncontagious disease of humans and animals, caused by *Rhinosporidium seeberi*. The clinical signs in animals are similar to those of polypoid rhinitis, characterized by sneezing and unilateral nasal discharge which can be seropurulent or hemopurulent.

Figure 2.9:
Alternaria infection of the nasal plane and the bridge of the nose of a cat. (Courtesy of Dr. G. ter Haar, Division of Surgery, ENT, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University).

Biopsy material reveals the fungus. *Rhinosporidium seeberi* has been found in biopsy material from a cat with nasal polyps.[33]

Parasitic rhinitis

Pneumonyssoides caninum. This nasal mite is found quite frequently in Sweden and Norway. In a prospective study in 474 dogs, 145 cats, and 66 wild red foxes were submitted for necropsy to 2 institutes in Upsala, Sweden; 95 (20 %) of the dogs but none of the cats were infected. The median number of mites per infected dog was 13 (range 1 to 250).[19] In a prospective study in 250 dogs submitted for necropsy during 1 year in Norway, 18 (7 %) were infected with *Pneumonyssoides caninum*. It was concluded that this nasal mite infection is common in Norwegian dogs.[5] It has also been reported to occur in dogs in the United States, Australia, Iran, and Greece, so worldwide distribution is suspected.[38]

The clinical signs of *Pneumonyssoides caninum* infection are nasal discharge, sneezing, and loss of the sense of smell. The mites are found by rhinoscopy. Treatment with ivermectin[38] or milbemycin[4] is effective.

Capillaria aerophilia, a parasite sometimes found in the trachea and bronchi of humans and animals, was found to be the cause of infection in the nasal cavity of a dog, resulting in recurrent epistaxis.[23]

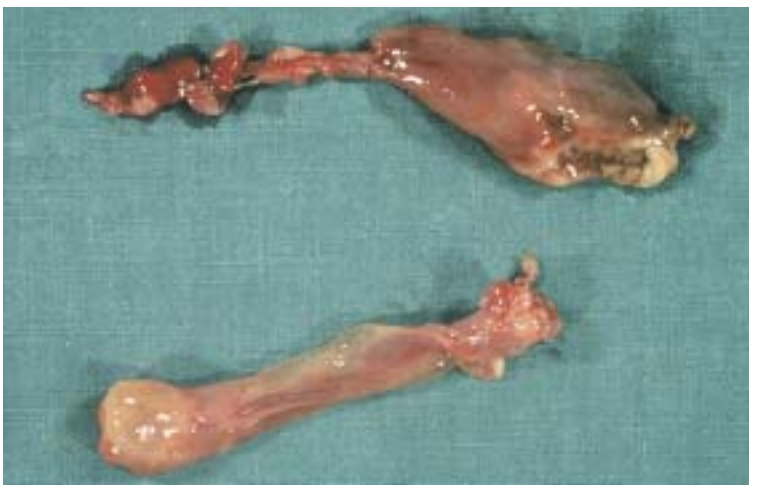


Figure 2.10: Two polyps removed from one nasal cavity of a cat.

2.5.2 Noninfectious rhinitis and sinusitis

Rhinitis and nasal polyps

Nasal polyps are focal proliferations of the mucosa which are not neoplastic but inflammatory, consisting of a rounded extremity attached to the mucosa by a stalk (Figure 2.10). They are most common in the nasal cavity of cats and are rare in dogs. They rarely arise in the sinuses, although extreme cases of polyps in the nasal cavity and frontal sinuses in cats have been described and illustrated.[34] Nasal polyps consist of focal accumulations of edema fluid and proliferation of the submucosal connective tissue, with a variable inflammatory infiltrate consisting of eosinophils, plasma cells, and lymphocytes.[41] They usually originate in the caudal part of the nasal cavity and consequently are covered by ciliated respiratory epithelium. The occurrence of nasal polyps is usually associated with chronic rhinitis. Recent studies of inflammatory nasal polyps in humans suggest an important role for proinflammatory cytokines, chemokines, and chemotactic factors in their pathogenesis, along with a variety of environmental, genetic, and biochemical factors.[24]

Clinical signs. The clinical signs of nasal polyps are those of obstructive rhinitis, with dyspnea and loss of olfaction, and nasal discharge during sneezing. Bilateral obstruction may be caused by bilateral polyps or by a unilateral polyp that extends into the nasopharynx. Radiographs reveal a unilateral or bilateral density with minimal loss of conchae. The diagnosis is made by rhinoscopy. The polyp is seen as a red mass in the nasal cavity and biopsy reveals it to be inflammatory tissue.

Surgical removal of the polyp. Treatment consists of surgical removal of the polyp, but locat-

ing its origin and removing it under vision can be very difficult. The alternative is to remove the polyp together with all of the conchae in the nasal cavity, which can be done via a small opening in the nasal bone. Especially in the cat, the removal of olfactory epithelium results in loss of the ability to recognize food. Hence when polyps are bilateral it is advisable to remove them in two stages. After one nasal cavity has been freed of polyps the cat regains its sense of smell with the contribution of the intact side. If necessary, surgery can also be performed on that side after the cat has recovered fully and eats without hesitation.

Dogs sometimes have a single nasal polyp but they may also have multiple proliferations in the caudal nasal cavity that close the opening to the nasopharynx. Treatment consists of surgical removal of the abnormal tissues via an opening in the nasal bone. The polyps usually originate on one side but sometimes, as in cats, they extend into the nasopharynx and may obstruct the caudal nasal openings bilaterally. Using suction through the caudal nasal opening, the polyp can be drawn out of the nasopharynx into the caudal nasal cavity, from which it can be removed. Dogs seldom have problems in recognizing food after surgery. In both dogs and cats the excised tissue should be examined histologically for possible neoplasia, regardless of the histological findings in biopsies obtained before surgery. The polypous proliferation in dogs may recur after a year or two, but single polyps in cats and dogs seldom recur.

Neurogenic rhinitis

Clinical signs. The clinical signs of neurogenic rhinitis in dogs are dryness of the nasal plane on one or both sides, sometimes with crusts, and slight mucopurulent nasal discharge, often together with sneezing. The most frequent clinical presentation is dryness of one side of the nasal plane together with ipsilateral keratoconjunctivitis sicca and mucopurulent conjunctivitis. The cause of the disorder is loss of parasympathetic innervation, and when keratitis sicca is present, this dysfunction also involves the lacrimal, palatine, and nasal glands.[44] In dogs with this disorder, ipsilateral otitis media was recognized by the reddening of the tympanic membrane. It was concluded that the lesion in the parasympathetic nerves was probably caused by otitis media, for the parasympathetic nerves are carried in the chorda tympani and pass freely through the middle ear. In all cases, treatment of the otitis media with broad-spectrum antibiotics resolved all problems.[48]

The diagnosis of neurogenic rhinitis is more difficult when it is bilateral. The clinical signs are persistent bilateral mucopurulent discharge, sometimes with sneezing and dryness of the nasal plane. The Schirmer tear test should be helpful to evaluate the production of mucus in the nasal mucosa, but no values for this have been reported. Clinical examination and exclusion of other causes may lead to the presumption of a possible neurogenic cause. Massaging nonperfumed moistening cream into the nasal plane 8 times daily and administering 4 drops of artificial tears into the nasal cavity 4 times daily will relieve the clinical signs if the presumptive diagnosis is correct. This treatment will be required lifelong, since there is no known specific treatment for the parasympathetic nerve dysfunction.[48]

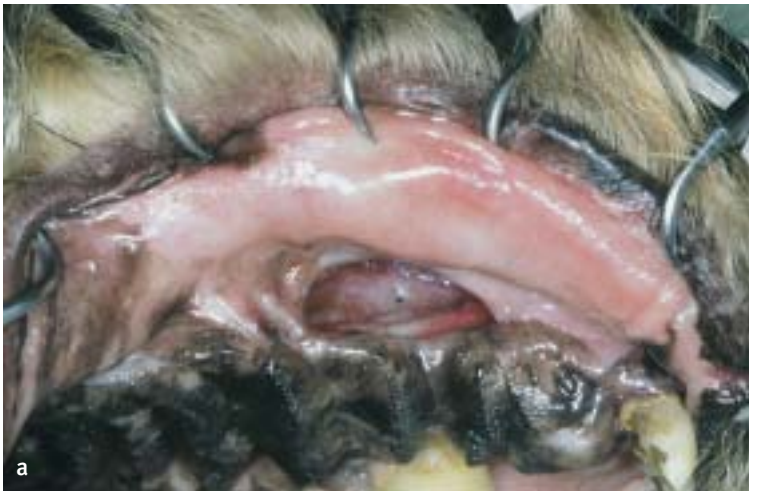
Rhinitis caused by dental disease

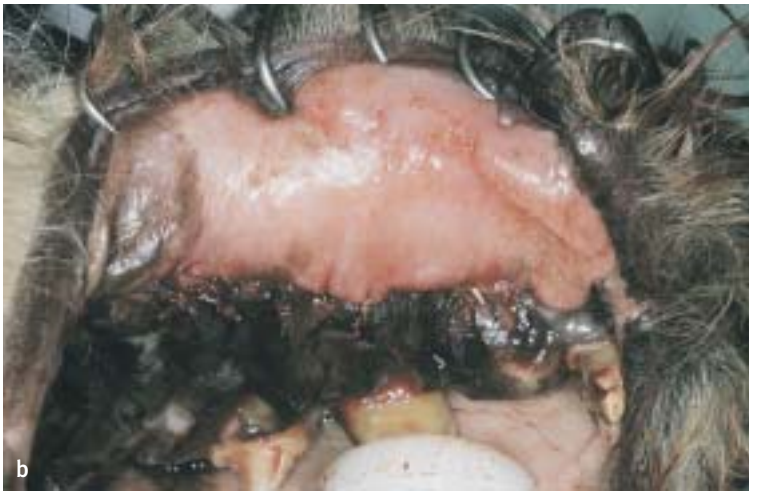
Severe periodontitis can lead to bone resorption and when this occurs in the maxilla, fistulae may allow passage of infectious material from the alveolar cavity into the nasal cavity, resulting in chronic rhinitis. In dogs and cats the maxillary canine tooth is best known as the origin of this complication. The clinical signs are usually unilateral nasal discharge, sometimes with blood, and oral inspection reveals inflammation of the gingiva around the canine tooth. Radiographs of the nasal cavities that include the maxillary canine teeth will show a soft tis-

sue process around the root of the infected canine tooth. Rhinoscopy often reveals proliferation of tissue around the root of the canine tooth, sometimes resulting in narrowing of the nasal passage. A veterinary dentist should be consulted for treatment. Since both periodontitis and bone resorption are considered to be irreversible, the treatment is often removal of the tooth and specific care of the fistula.[18]

An oronasal fistula may result from extraction of a maxillary canine tooth. The passage of food and debris through the fistula, together with local drying of the nasal mucosa, may result in rhinitis in that nasal cavity. The fistula should be closed surgically, making a thick rotational buccomucosal flap of substantial size. After débridement of tissue around the fistula opening in the nasal cavity, the site for the buccal gingival flap is prepared by removing the buccal mucosa and periosteum lateral to the fistula. The buccal flap is enlarged by extending the incisions in the mucosa and separating a thick layer of mucosa supported by submucosa and fascia from the inner side of the cheek opposite the fistula, leaving the flap attached at its proximal border. The flap should easily cover the fistula opening and the sutures should lay on the maxillary bone around the opening[37] (Figure 2.11 a, b).

Figure 2.11 a, b: Oronasal fistula in a dog resulting from extraction of the canine tooth (a). (b) A buccal mucosal flap now covers the fistula opening, with the sutures lying on the maxillary bone caudal, ventral, and rostral to the opening.





Rhinitis caused by foreign bodies

Nasal foreign bodies such as grass and other kinds of plant material are common in young dogs and cats, causing a unilateral mucopurulent nasal discharge. The history may reveal when the material entered the nasal cavity, for the incident is usually marked by a short period of unusual behavior including rubbing of the nose and ferocious sneezing. Since most nasal foreign bodies are plant material, which has about the same density as soft tissue, they are not revealed by radiography. Rhinoscopy in search of such material needs patience and the use of suction under visual control, to remove the mucopurulent exudate so that the foreign material is exposed and can be removed. Grass can enter the nasal cavity while a cat is chewing on it, as cats often do, for out of a ball of mucus and grass in the pharynx, a sprig of grass can enter the nasal cavity via the nasopharynx. It can be found by rhinoscopy and removed under visual control via the nostril.

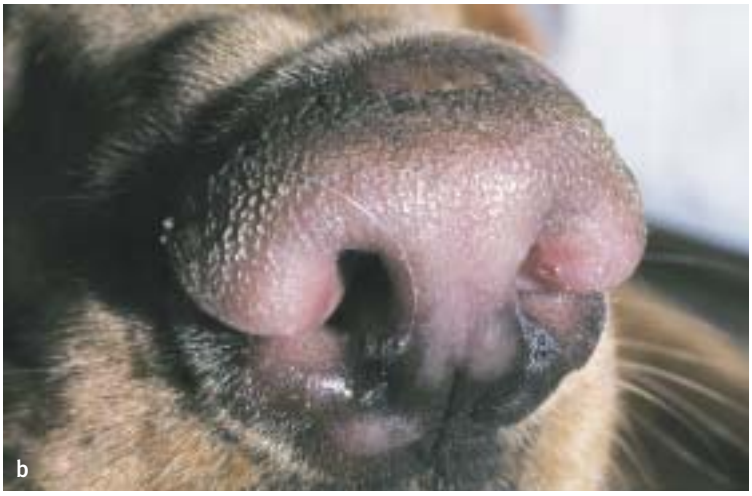
Nasal sarcoidosis

Sarcoidosis is a systemic disease of unknown cause, characterized by noncaseated epithelioid granulomas. In humans it occurs rarely as an isolated lesion in the upper respiratory tract, but nasal sarcoidosis is usually associated with lower respiratory involvement.[26]

The first dog in which nasal sarcoidosis was diagnosed was a 1.5-year-old mixed-breed male. The clinical signs were depigmentation and swelling of the nasal plane (Figure 2.12 a, b), mucous nasal discharge, and nasal obstruction which caused dyspnea and open-mouth breathing. Nodules were found on the bridge of the nose and on the pinnae (Figure 2.12 c). Radiographs of the lungs revealed no abnormalities. In subsequent cases nodules have been found not only on the nasal plane, the bridge of the nose, and the pinnae, but also in the mucosa of the nasal cavity when rhinoscopy was added to the diagnostic procedure because of rhinitis. Obstruction of the nasal airway caused dyspnea in these cases. In none of the dogs were the lungs involved in the disease.

The histological diagnosis based on biopsies from dogs with similar clinical conditions has varied over the years from sarcoid to sarcoidal granulomas to histiocytosis (as in Bernese mountain dogs). These histological diagnoses are similar in that the granulomas have »naked« epithelioid cells, unaccompanied by surrounding inflammation and fibrosis.[43]

In the first dog the disease was resolved by treatment with oxytetracycline given orally, and the other dogs responded, sometimes dramatically, to treatment with glucocorticoids administered systemically and intranasally. In one dog the disease was refractory and continuous nasal dyspnea resulted in euthanasia. In humans the treatment for nasal sarcoidosis consists of topical glucocorticoids together with systemic glucocorticoids and cytotoxic agents.[26]



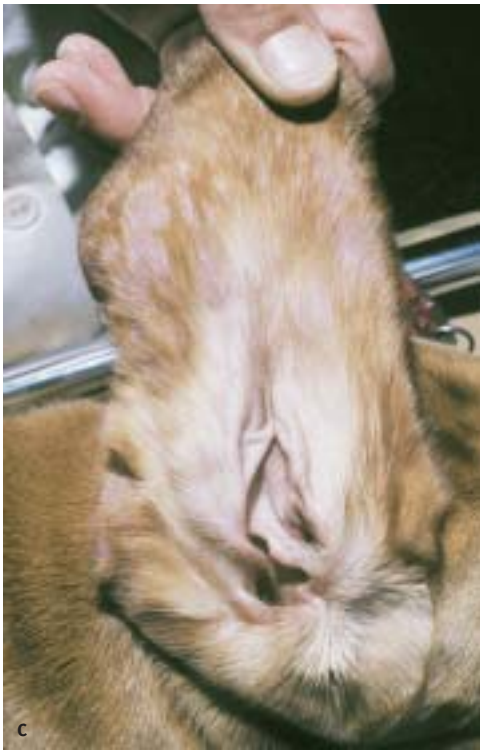


Figure 2.12 a–c: Nasal sarcoidosis in a dog. There are nodules on the bridge of the nose (a), depigmentation and swelling of the nasal plane (a, b), and nodules on the auricles (c).

Allergic rhinitis

Allergic rhinitis is presumed to occur in dogs and cats, but confirmation of the diagnosis has not yet been convincing, partly because IgE-based rhinitis, as occurs in humans, has yet to be demonstrated.

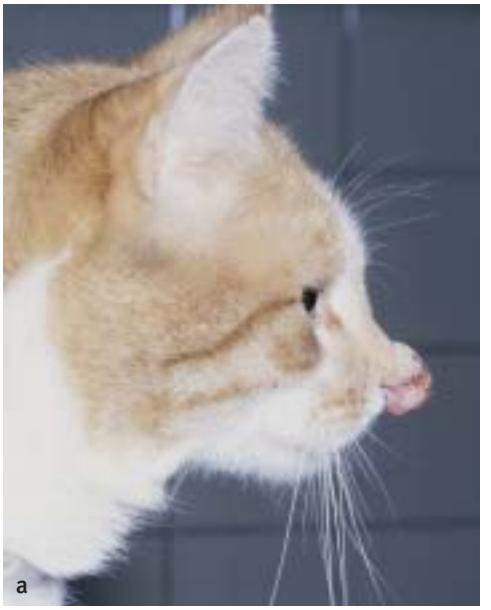


Figure 2.13 a, b: Fibrosarcoma of the nasal plane in a cat (a). The tumor was removed surgically without too much cosmetic damage to the nostrils (b).

Nonspecific chronic rhinitis

Nonspecific chronic rhinitis occurs in dogs and is characterized by long-term mucous or mucopurulent nasal discharge. The radiographic findings are a slight increase in density in the nasal cavities without destruction of bony structures. Rhinoscopy reveals only hypervascularization and thickening of the mucosa. The rhinitis is refractory to symptomatic treatment. The possible role of immunological mechanisms in the disease was examined by comparing immunoglobulin concentrations in nasal lavage fluids from 33 dogs with nonspecific chronic rhinitis and 19 healthy control dogs.

The immunoglobulin IgA was detected in nasal lavage fluids from both groups of dogs and the differences between them in relative levels of IgA were not significant. IgM, IgG(a,b), and IgG(d) were detected more frequently in dogs with nonspecific chronic rhinitis ($p < 0.05$), and the relative levels were higher. The presence of IgG(a,b) may indicate the chronic character of the rhinitis and the presence of IgG(d) may indicate an allergic component in the pathophysiology. The role of IgM is not completely clear. It was concluded that the allergen-specific IgE content of nasal secretions may be a means of differentiating dogs with allergic rhinitis.[51]

2.6 Tumors of the nasal plane, the nasal cavity, and the frontal sinus

2.6.1 Tumors of the nasal plane

Tumors of the nasal plane occur in both dogs and cats. Fibromas and fibrosarcomas grow beneath the epithelium initially, causing a nodular enlargement of the nasal plane (Figure 2.13 a). The diagnosis is made by fine needle aspiration biopsy. Surgical removal at an early stage is possible without too much cosmetic damage or damage to the nostrils (Figure 2.13 b). Squamous cell carcinoma of the nasal

plane also occurs in dogs and cats. The predominant clinical sign is a lesion characterized by excoriation and bleeding (Figure 2.14 a). Since the nasal plane is not always swollen, the lesion is not always recognized as tumor. Metastasis is possible but rarely occurs. Regional lymph nodes should be palpated and biopsied if enlarged. Radiographs of the thorax are recommended in advanced cases. The diagnosis is made by biopsy of the lesion, taking care not to damage more of the nasal plane than necessary, but the histological report may be misleading when inflammation and not tumor is found. If the process increases and tissue destruction continues, surgical removal of the nasal plane is justified, even when repeated biopsies are negative for tumor. The excised tissue should also be submitted for histological examination. In the differential diagnosis of processes causing proliferation of the tissue of the nasal plane with crusts and destruction, infection by *Alternaria* and eosinophilic granuloma should be included. Both are easily identified in biopsy material.

Surgical removal of the nasal plane has been described for dogs and cats.[50] The nasal plane is removed with one firm incision, beginning just rostral to the nasal bone and ending at the rostral margin of the lip on the midline. One firm incision that includes part of the deeper cartilages of the conchae will allow histological confirmation of a tumor-free cut surface. Hemorrhage is controlled by applying gentle pressure with gauze sponges for several minutes. Electrocoagulation is used for persistent focal bleeding. The bleeding is easier to control in cats than in dogs. A single-layer purse-string suture of polyamide or polydioxanon is then placed through the skin near the cut edge. The suture is gently tightened until the constriction leaves a free margin of a millimeter or so between the nasal openings and the skin. The suture is removed 3 weeks later. The cosmetic results are satisfactory in cats (Figures 2.14 b–d) but in dogs they are sometimes more difficult to accept. In both, the best results are obtained when the tumor does not extend around the nasal plane.

Figure 2.14 a–d: Squamous cell carcinoma of the nasal plane in a cat (a). Six weeks after surgical removal of the nasal plane, the wound was still slightly swollen but the nasal openings were satisfactorily functional (b). Three months after surgery, the wound was healed and the nasal openings were functional, but the tumor was recurring below the left nasal opening (c). The recurrence was removed by a wide incision, necessitating a skin flap from the left (d). Further recovery was uncomplicated.

2.6.2 Tumors in the nasal cavity

Tumors occur in the nasal cavity of dogs and cats of all ages, but most often from the age of 5 years onwards. Almost all are malignant. They invade the surrounding tissue but rarely metastasize before the animal is euthanized. Animals living in the company of people are rarely allowed to die spontaneously from the disease. The most frequent tumors are squamous cell carcinoma and adenocarcinoma; less frequent are chondrosarcoma, osteosarcoma, lymphosarcoma,[48] and primary venereal tumors.[39] In a review of nasal and paranasal tumors in 123 cats, malignant lymphoma was the most common.[35]

Clinical signs. The clinical signs include sneezing, hemorrhagic discharge, and mucopurulent discharge. In most cases unilateral obstruction of the nasal cavity is recognized because of nasal stridor. No evidence of pain is observed and the dog or cat becomes dyspneic only when its mouth is closed, which means when sleeping. As long as the tumor is unilateral the dyspnea is moderate. When the tumor obstructs both nasal cavities, dyspnea during sleep becomes a serious hindrance, for it causes the animal to awaken repeatedly during the night and to be very depressed in the morning. There is still no evidence of pain.

Diagnostic radiographs should be made under anesthesia. Tumor is suspected when increased density is found in one or both nasal cavities, with loss of normal maxillary and ethmoidal conchae.[42] The extension of the tumor is considered in offering a rough estimate of the animal's life expectancy, but this is usually decided by the owner's interpretation of the quality of the animal's life. In all cases in which the radiographic diagnosis is uncertain, rhinoscopy is the next diagnostic procedure. Under rhinoscopic visualization the tumors vary greatly in shape and firmness, and their color ranges from gray to deep red. Biopsies are always taken for histological confirmation of the diagnosis. If no therapy is planned, neither CT nor MRI is indicated.

In dogs and cats tumors in the nasal cavity are often presented with extensive bony involvement. Without treatment, life expectancy depends on the animal's and the owner's tolerance of the clinical signs, but the usual interval between diagnosis and euthanasia is 3 to 5 months. When, with good reason, no therapy is offered beyond permanent tracheostomy, which is rarely accepted by owners, recurrent nasal bleeding and dyspnea are the usual reasons for euthanasia in both dogs and cats. Cats, however, often stop eating, which can provide a humane end point and a reason for euthanasia.

Radiation therapy could be considered and details concerning it have been described.[8] It is the only form of treatment that improves the quality and duration of survival. Cytoreductive therapy alone does not increase survival time or the disease-free interval in dogs or cats. The poor response to surgery is attributed to the local invasiveness of most tumors in the nasal cavity. Chemotherapy, immunotherapy, and cryosurgery have not been shown to improve survival of patients with these tumors.[8]

2.6.3 Tumors in the frontal sinus

Tumors in the frontal sinus develop within a bony case. Since the frontal sinus does not have a specific function, development of a tumor is noticed only when there is recurrent unilateral nasal bleeding, or when the enlarging tumor causes pressure atrophy of the frontal bone and becomes apparent as a swelling arising in the frontal sinus.

In the diagnostic investigation of recurrent nasal bleeding, radiographs will be made of the nasal cavity and the frontal sinus and a radiographic density in the frontal sinus will be noticed. The differential diagnosis of such a

density in combination with a radiographically normal ipsilateral nasal cavity includes aspergillosis, tumor, and accumulation of mucus due to obstruction of the nasofrontal duct. Since the frontal sinus is only separated from the brain by a thin layer of bone, additional diagnostic imaging by CT or MRI is wanted. When a swelling arising in the frontal sinus is recognized by physical examination, the same procedure of taking radiographs followed by CT or MRI is indicated. Not only the extension of the lesion, but also differentiation of the contents of the frontal sinus can be resolved. Tumor in the frontal sinus can be recognized by CT or MRI with an intravenously administered contrast medium. The contrast medium will enhance vital tissue but not debris. It must then be determined by CT or MRI whether the bony case of the frontal sinus is intact, especially whether the orbit or the brain is invaded, and whether the contralateral frontal sinus is included in the process.

When these procedures indicate that the tumor can be removed, a surgical approach via the frontal bone is indicated. At the end of the surgical procedure it is important to examine the patency of the nasofrontal duct and to relieve any obstruction. Like tumors in the nasal cavity, most tumors in the frontal sinus are malignant and complete surgical removal is unlikely. Biopsy of the tumor through a small trephined opening in the frontal bone should be considered as an intermediate step.[48]

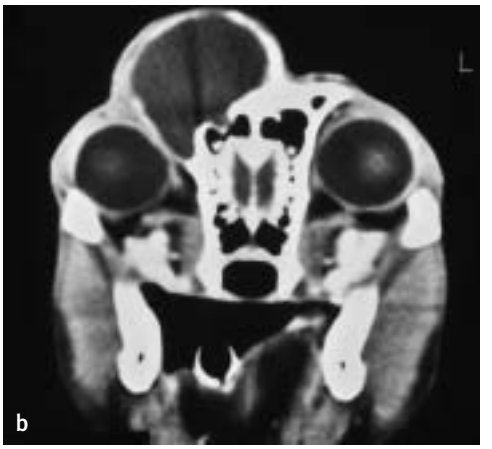
2.7 Trauma to the frontal sinus and the nose

2.7.1 Trauma to the frontal sinus

Traumatic injury to the frontal sinus can be caused by blunt or sharp objects. The frontal bone in dogs and cats is relatively thick and provides good protection, so a fracture of it implies a very heavy blow to the head. The patient should therefore be given a thorough clinical examination for (1) signs of shock such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilation of the pupils, hypothermia, muscle weakness, restlessness, and depression or even coma,[45] and (2) other fractures or wounds. Frontal bone fractures do not require immediate attention unless brain damage is suspected. In the absence of signs of brain damage, and when other traumatic injuries have been attended to, the nature and extent of the frontal bone fracture should be examined by radiography and/or CT. Prolonged anesthesia is needed and thus these procedures are usually delayed for 24 hours or more.

When bone fragments are seen to be present in the frontal sinus they should be removed, for like all small bone fragments they are likely to become sequestered. Surgery should be performed with full attention to aseptic procedures. Before attempting reconstruction of the frontal bone, it is important to examine the patency of the nasofrontal duct and to relieve any obstruction. Airtight suturing of the subcutis, including periosteum, followed by routine skin closure prevents the development of subcutaneous emphysema. Administration of broad-spectrum antibiotics for 3 weeks and strict limitation of activity during this period (keeping a cat confined to the house) will prevent complications.[48]

A complication of frontal bone trauma was demonstrated in a case report.[47] A dog was presented with a painful, fluctuating swelling over the right frontal sinus (Figure 2.15 a). The history revealed that the dog had been struck by a car 8 months earlier and that it had become painful after repeated aspiration of brownish-red fluid from the swelling. CT scans revealed deformation of the right frontal sinus. At soft tissue settings, the volume of the sinus was increased, its content was dense material rather than air, and the density of the frontal bone was decreased. The right orbit was not involved, nor was the left frontal sinus (Figures



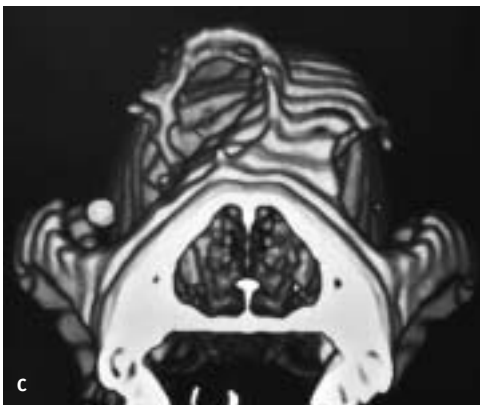


Figure 2.15 a–c: A Cairn terrier was presented with a painful fluctuating swelling over the right frontal sinus, 8 months after the dog had been stuck by a car (a). CT scans revealed deformation of the right frontal sinus. At normal settings (b) the volume of the sinus was seen to be increased, its content was dense material rather then air, and the density of the frontal bone was decreased. The right orbit was not involved, nor was the left frontal sinus (c). Surgical findings and treatment are described in the text. (CT courtesy of Dr. G. Voorhout, Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University).

2.15 b, c). At bone settings, the structure of the right frontal bone indicated advanced atrophy. Surgery was undertaken to examine the right frontal sinus and the associated nasofrontal opening, obstruction of which was presumed to be preventing drainage of the mucus normally produced in the sinus. The opening was restored by removing the loose bone fragments and granulomatous mucosa which were obstructing it. The problems were thereby resolved and did not recur.

2.7.2 Trauma to the nose

Trauma to the nose is often characterized by massive bleeding, which adds to the other effects of the impact in promoting shock. A thorough examination for signs of shock is indicated, as described for trauma to the frontal sinus. Fractures and wounds should be noted but priority must usually be given to the treatment of hypovolemic shock. When the patient is sufficiently stable, the larger lacerated vessels should be ligated and skin sutures should be placed as needed. Skin sutures, sometimes supported by subcutaneous sutures, may be sufficient to remodel the outer form of the nose. Fractures of the choanae are best left alone, for they are unlikely to ever result in obstruction. In almost all severe traumatic damage to the nose there is temporary obstruction, making tracheostomy necessary. Adequate oxygenation aids in preventing general malaise and loss of appetite. Liquid or soft food facilitates eating. In dogs the tracheal cannula is often left in place for 10 days or longer. In cats in which there are difficulties with long-term tracheostomy, a small intranasal catheter may be placed and connected to the oxygen supply. This method, however, depends very much on the pathway through the wounded nose. In our experience the nose is functionally adequate in 2 to 3 weeks. If needed, more corrective surgery could be attempted after 6 weeks.[48]

2.8 Epistaxis

The nose has an extremely good blood supply from both the external and the internal carotid arteries, with bilateral anastomoses between the internal carotid artery and the maxillary artery (the main branch of the external carotid artery). The maxillary artery is the main supply to the nasal cavity. Chronic unilateral occlusion of the carotid artery in dogs caused numerous collateral connections to develop between corresponding vessels on the two sides.[6, 7] Many conditions, both local and general, can cause nasal bleeding, but most nasal bleeding is incidental, caused by transient local disorders, and stops spontaneously or after simple medical intervention. However, any nasal bleeding—occurring most often in dogs—is frightening to the owner and quickly causes an incredible mess in the house. A strategy for handling epistaxis as an emergency is therefore helpful.

2.8.1 Management of acute epistaxis

The history may aid in the differential diagnosis, for trauma can obviously explain acute nasal bleeding and a history of nasal discharge points to a local cause. In addition to drying blood on the face and front legs, there is often still fresh blood running from one of the nostrils. Pale pink or white mucosa may reflect severe blood loss, but shock must be taken into account. The amount of blood that has been lost cannot be estimated by the visible loss from the nostril, because a considerable amount will have been swallowed and will pass through the gastrointestinal tract, resulting in melena.

Clinical signs of shock, due to severe blood loss and/or trauma, include tachycardia, prolonged capillary refill time, weak pulse, rapid respiration, and hypothermia. With or without shock, intravenous fluid therapy can be started directly, during the clinical examination. In most cases the dog is restless and sneezing, and walking around increases both its and the owner's agitation. Administration of a sedative calms the dog and usually decreases the epistaxis. Sedation with phenobarbital intravenously has the advantage that it can be administered to effect and does not lower the blood pressure. The hematocrit can be measured but it will not be stabilized soon after the blood loss and thus the measurement must be repeated. If the nasal cavity is obstructed by clotted blood or other masses, endotracheal intubation should be considered. In apparently uncomplicated blood loss, the dog can be sedated for 12 to 24 hours, by which time the epistaxis usually stops and a diagnostic plan can be made. If the epistaxis continues, a blood transfusion should be given, always preceded by collection of a sample for routine hematology and coagulation studies before the values are changed by the transfusion. Only in life-threatening continuous blood loss is ligation of the ipsilateral external carotid artery indicated.

2.8.2 Causes of epistaxis

Most causes of epistaxis are local, the most common being trauma, inflammation, infectious diseases, and tumors, but some cases are idiopathic. Trauma may be external, usually caused by accidents, or internal, as may be incurred during rhinoscopy, biopsy of intranasal tissue, or nasal surgery. Foreign bodies seldom cause epistaxis, the predominant signs they cause being instead sneezing and unilateral rhinitis. Local inflammation may be caused by polyps in the nasal cavity and dental root infections, especially from the canine teeth. In other cases the focus of inflammation is found but the cause cannot be determined. An infectious disease known for causing epistaxis is aspergillosis in the nasal cavity. The *Aspergillus* plaques can cause nasal bleeding but the invasive rhinitis caused by the toxins from the fungus is also

characterized by lesions in the mucosa. The epistaxis is intermittent but can be severe. *Linguatula serrata* is an arthropod that parasitizes the nasal cavity of dogs. It sometimes causes severe rhinitis with sneezing and epistaxis.[27] Tumors arising in the nasal cavity often cause intermittent epistaxis, which may be severe, but there may also be nasal discharge containing some blood.

Epistaxis can occur in systemic diseases that affect coagulation. Coagulation disorders may also be congenital, such as hemophilia A and B in cats and von Willebrand's disease in dogs. Nasal bleeding may be one of the signs or even the first sign of these diseases. Acquired coagulopathies due to deficiency of clotting factors can occur in liver failure and renal failure. In dogs, intoxication by coumarin, a common rat poison, is a well-known cause of epistaxis. Myelosuppressive drugs may cause epistaxis, as does estrogen, in both cases due to decreased production of platelets. Epistaxis can be one of the clinical signs of systemic infectious diseases that cause ulcerations in the nasal mucosa, leishmaniasis being an example.[22] Hemopoietic diseases, such as aplastic anemia, lymphoma, and widespread metastasis of hematopoietic neoplasms, form another group of diseases causing epistaxis.

2.8.3 The diagnostic plan

When the nasal bleeding is largely under control, the diagnostic plan can begin with the history and clinical examination. The medical history will usually include the time of onset of the nasal bleeding and the circumstances under which it occurred, and whether this is the first episode or whether there have been one or more previous episodes. Concurrent nasal discharge in the past may indicate a longstanding nasal disease now causing epistaxis. Further questions are asked about the animal's general condition, appetite, drinking, activity, endurance, and dyspnea. The general clinical examination follows, it being of special importance because of the variety of causes of epistaxis. The color of the mucosa provides a rough estimation of the blood loss. The nose is inspected and the passage through the nose is tested with a fluff of cotton held in front of each nostril. The findings of the clinical examination may clearly point to either a local or a systemic disorder. If local disease is suspected, routine hematological studies are appropriate but if a systemic disease is suspected, coagulation studies should be added.

If local disease is suspected, radiographs of the nose and nasal sinuses should be obtained when the condition of the animal allows sedation or anesthesia. If there is severe dyspnea an endotracheal tube should be inserted. The radiographic technique is described in Chapter 2.3. Radiographs of the nasal cavity shortly after nasal bleeding may reveal densities erroneously suggesting tumor growth, but thorough study of the fine trabecular pattern will aid in determining whether there is loss of nasal structures.[42] The radiographic findings will indicate the next step in diagnosis. Following trauma, a radiographic density in the frontal sinus or other findings requiring surgical intervention may indicate the need for CT or MRI. If the radiographic findings are less pertinent to determining the cause of the epistaxis, rhinoscopy is the next step. While rhinoscopy sometimes clearly reveals the site of the bleeding, in dogs and cats this is often difficult to reach for coagulation because of the conchal structures. More often the exact origin of the bleeding is not found by rhinoscopy. However, the finding of either *Aspergillus* plaques or tumor can be assumed to explain the epistaxis.

2.8.4 Management of intermittent epistaxis of unknown origin

If the cause of the nasal bleeding has not been determined by the forgoing examinations, laboratory studies are undertaken to diagnose or rule out the many systemic disorders that can cause epistaxis. When this also fails to reveal the cause of the epistaxis, surgical intervention is indicated. The nasal cavity on the side of the epistaxis is approached via an opening in the nasal bone and all structures within it are removed.

For this procedure, the animal is anesthetized and an endotracheal tube is inserted and its cuff is inflated. It is helpful to begin the intravenous administration of whole blood or plasma, or if unavailable, a blood replacement product. After surgical preparation of the bridge of the nose and the surrounding area, the affected nasal cavity is approached by making a median rostral-to-caudal skin incision over the bridge of the nose and removing the skin, subcutis, and periosteum at the planned site of trepanation. An osteotome is used to make a small square or rectangular opening in the nasal bone on the side of the epistaxis, just large enough to allow a curette to be used to remove the structures in the nasal cavity. The tissues that are removed are preserved for histological examination. There may be substantial bleeding and thus both homeostasis and the rate of transfusion should be carefully monitored. Once the conchae and the mucosa are completely removed, the bleeding will stop, although repeated temporary packing with cotton or gelatin sponges, and considerable patience, may be necessary. Packing material should preferably be removed before the wound is closed, for it can lead to severe sneezing which may induce renewed bleeding. If thought to be absolutely necessary, gelatin sponge should be left in place rather than cotton. The wound is closed by a continuous suture in the subcutis, including the periosteum. The subcutis over the opening in the nasal bone must be securely apposed to make the closure airtight. The skin is closed routinely. Aftercare consists of administering analgesics and monitoring the hematocrit. Slight blood loss via the nostril is to be expected during the first 48 hours. Continuing blood loss via the nasopharynx and the gastrointestinal tract may be signaled by melena. In most cases of intermittent epistaxis of unknown origin this surgical treatment solves the epistaxis.

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3 The Pharynx

3.1 Functional considerations

The pharynx is a fibromuscular tube that serves as a conduit for both the respiratory and the digestive tract. It is divided into three anatomical segments: the nasopharynx, the oropharynx, and the laryngopharynx. The nasopharynx is the respiratory portion above the hard and the soft palate, extending from the choanae of the nasal cavity to the intrapharyngeal opening of the pharynx, formerly called the pharyngeal isthmus. The oropharynx is the region between the soft palate and the base of the tongue, bounded laterally by the palatoglossal and palatopharyngeal arches, and caudally by the tip of the epiglottis. The laryngopharynx is the portion of the pharynx dorsal to the larynx, extending from the rostral border of the epiglottis to the caudal border of the cricoid cartilage.

Intrapharyngeal opening. The intrapharyngeal opening is the opening of the nasopharynx over the edge of the soft palate into the oral and laryngeal parts of the pharynx. During swallowing the circular closure of the intrapharyngeal opening by the coordinated action of several paired muscles separates the nasopharynx from the oropharynx. The palatopharyngeal muscle constricts the pharynx and draws it forward and upward while the elevator muscle of the soft palate raises the caudal part. The tensor muscle of the soft palate also contributes to the closure of the intrapharyngeal opening.[11, 23] Failure to separate the nasopharynx from the oropharynx during swallowing, called velopharyngeal insufficiency,[63] may result in reflux of fluid into the nasal airway. Inappropriate closure of the intrapharyngeal opening causes »reverse sneezing«.

3.1.1 Auditory tube serves to equalize atmospheric pressure

The auditory tube or eustachian tube opens on the lateral wall of the nasopharynx, above the middle of the soft palate. It connects the cavity of the middle ear with the cavity of the nasopharynx and serves to equalize the atmospheric pressure on opposite sides of the tympanic membrane.

Palatine tonsil. The palatine tonsil is a thin lymph node located bilaterally in the lateral wall of the oral part of the pharynx, just caudal to the palatoglossal arch. In dogs it is hidden from casual observation by being located in the tonsillar crypt or fossa and covered by the tonsillar fold, which arises from the ventral surface of the lateral portion of the soft palate.[16] In cats the palatine tonsil is very small and not covered by a mucosal fold. The palatine tonsil has no afferent lymphatics and its efferent vessels drain into the retropharyngeal lymph node.

Although the laryngopharynx has both a respiratory and an alimentary function, its primary importance is in deglutition. A bolus of food is conveyed by the base of the tongue into the laryngopharynx, where six pairs of extrinsic muscles control the shape and size of the nasal and laryngeal parts of the pharynx and thereby further shape the bolus before its propulsion into the esophagus.

3.1.2 Swallowing

Swallowing is a physiological phenomenon that occurs many times daily. Although it may be initiated consciously as a voluntary act during eating, most swallows occur subconsciously between meals, without apparent cerebral participation. Swallowing irrespective of eating occurs about once a minute in awake individuals and is driven by salivation, which stimulates the sensory receptors in the mouth and phar-

ynx.[9] Studies in humans have shown that salivation and swallowing virtually cease during sleep.

Swallowing is a complex and complicated function that can be divided into four overlapping stages: oral preparatory, oral, pharyngeal, and esophageal. The first two are voluntary and the last two are involuntary. In the oral preparatory stage the food is chewed and prepared for the oral stage. In the oral stage the food bolus is moved from the front of the oral cavity to the faucial arch, where swallowing is initiated.

The pharyngeal stage has four defined components: palatopharyngeal closure, peristalsis of the pharyngeal constrictor muscles, airway protection, and cricopharyngeal relaxation. The larynx is elevated and closed, protecting the airway. These reflex movements occur in sequence and overlap one another. The reflexes are mediated by the reticular formation in the brain stem and follow a fixed pattern. The interneuronal network involved in this pattern is referred to as the central pattern generator (CPG) for swallowing. There is coordination with the respiratory center.

In the esophageal stage, the bolus is moved through the esophagus by the peristaltic contractions of the esophageal musculature.

Triggering of the swallowing action. The act of swallowing is triggered by the contact of a liquid or solid with the soft palate, the dorsal or the pharyngeal surface of the tongue, or the laryngopharynx.[35] The receptors in the oropharynx correspond to the glossopharyngeal nerve, those in the laryngopharynx correspond to the cranial laryngeal nerve, and those on the dorsal surface of the tongue correspond to the lingual nerve, a branch of the trigeminal nerve. Not only does triggering of the swallowing action rely on these receptors, but sensory feedback also assists tongue movement in the oral and pharyngeal stages in the formation of the food bolus. Reaching the stimulus threshold to elicit repeated swallows is enhanced by continuous feedback from the soft palate, the pillars of the fauces, the tonsils, the base of the tongue, and the pharynx.[35]

The sensory nerve fibers conveyed by the cranial laryngeal nerve and the glossopharyngeal nerve terminate in the solitary tract and nucleus (NTS) in the brain stem. The fibers of the trigeminal nerve terminate in the spinal tract of the trigeminal nerve and the solitary nucleus.[64] Within these two systems the solitary tract and nucleus constitute the main afferent system involved in swallowing, particularly the caudal part of the solitary system which receives the afferents of the cranial laryngeal nerve, or superior laryngeal nerve (SLN) in biomedical literature. Stimuli via afferent fibers in the SLN appear to be the most potent in triggering swallowing.[27] Swallowing can also be initiated by stimulation of a limited area of the frontal cortex. The corticofugal swallowing pathway terminates within the solitary system, the NTS. The solitary system is truly the major afferent system for swallowing.[27]

Central pattern generator for swallowing. The major afferents for swallowing, the SLN fibers running within the solitary tract, are not connected directly to the cranial motor nuclei involved in swallowing—V, VII, IX, X, and XII—in the brain stem. The successive excitation of motor neurons which is responsible for the entire sequence of swallowing depends on an interneuronal network which organizes this activity through excitatory and inhibitory connections between neurons. This organizing system, which is obviously placed between the afferents and the motor neurons, represents the major component of the central pattern generator (CPG) for swallowing, the so-called »swallowing center«. There are actually two CPGs, one in each side of the brain stem, for bilateral organization of motor activity in the bilateral swallowing muscles. The activity of the two CPGs during swallowing is coordinated via interconnections between them,[27] (Figure 3.1).

The interneurons are located in two distinct regions of the medulla: a dorsal region that includes the NTS and the adjacent reticular formation, and a ventral region corresponding to the lateral reticular formation around the nucleus ambiguus. Microelectrode recordings have shown that the neurons situated in the dorsal region exhibit a premotor activity pattern, that is, sequential firing similar to the sequential motor pattern in buccopharyngeal swallowing. These neurons are thought to be the »master« neurons of the CPG, the neurons that generate the swallowing pattern and thus play a vital role in pharyngeal swallowing. It has also been suggested that the neurons in the dorsal region which are triggered into action can develop this timing without sensory feedback and that they represent activity patterns of different swallowing muscle groups.[26, 36] In the ventral region of the CPG around the nucleus ambiguus, there are translateral connections between the left and right NTS and further synaptic connections between bilateral regions of the brain stem involved in swallowing.[26, 36]

Once activated, the CPG can trigger and organize the sequential swallowing pattern without feedback phenomena. However, its activity can also be regulated by peripheral afferent inputs that can control the entire motor sequence of swallowing. Inputs from the cortical swallowing area can also control CPG activity, at least for the buccopharyngeal phase.[27]

Motor innervation for swallowing. The motor neurons of cranial nerves V, VII, IX, X, and XII are located bilaterally in the brain stem. Motor neurons of the trigeminal nerve innervate the mylohyoid, rostral digastric, and pterygoid muscles and the tensor muscle of the soft palate; motor neurons of the facial nerves innervate the caudal digastric and stylohyoid muscles; and motor neurons of the nucleus ambiguus innervate the muscles of the pharynx and larynx and the striated muscles of the esophagus. In the nucleus ambiguus the rostral-to-caudal sequence of the motor nuclei is: esophagus, pharynx, larynx. The nucleus ambiguus may be considered to be the main motor nucleus for swallowing, together with the hypoglossal nucleus for the tongue.[10, 27, 34] The smooth muscles of the esophagus of the cat are innervated by the preganglionic neurons located within the dorsal nucleus of the vagus.

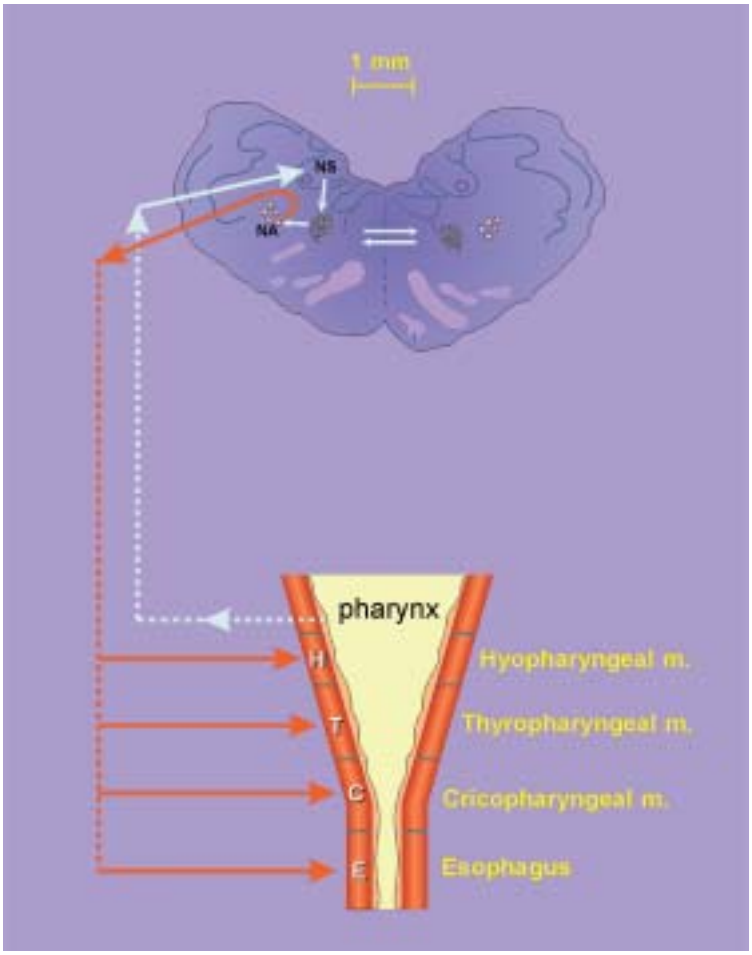


Figure 3.1:
Functional relations between the pharynx and the brain stem (shown in transverse section) during pharyngeal swallowing. Afferent fibers in the glossopharyngeal nerve, the pharyngeal branch of the vagus, and the cranial laryngeal nerves activate the solitary nucleus (NS). Via synapses with interneurons of the central pattern generator, sequential activation of the neurons in the nucleus ambiguus (NA) generates pharyngeal swallowing. Interconnections between the left and right central pattern generators coordinate their activity (From: Venker-van Haagen AJ. Diseases of the throat. In: Ettinger SJ, Feldman EC, editors. Textbook Veterinary Internal Medicine, Philadelphia: WB Saunders Company, copyright 2000: 1025–1031, Fig. 124-1; with permission from Elsevier).

The peripheral motor innervation of the larynx is described in Chapter 4.1. The motor supply to the pharyngeal muscles is distributed by the pharyngeal plexus. The glossopharyngeal nerve and the pharyngeal branch of the vagus nerve contribute to the plexus.[17] The contributions of the glossopharyngeal nerve and the pharyngeal branch of the vagus nerve to the swallowing process in dogs have been investigated.[65] The anatomy of the pharyngeal plexus was studied by dissection in 5 canine cadavers. Branches of the glossopharyngeal nerve (IX) joined the branches of the pharyngeal branch of the vagus nerve (Xph) in various ways, resulting in the combined innervation of the pharyngeal muscles by both nerves (Figure 3.2). In 10 anesthetized dogs the left and right parent trunks of the glossopharyngeal nerve and the pharyngeal branch of the vagus nerve were stimulated electrically and the evoked responses were recorded by electromyography (EMG) in the left and right stylopharyngeal, hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles. In all 10 dogs the left and right stylopharyngeal and hyopharyngeal muscles were innervated by the ipsilateral IX nerve but not always by the Xph nerve, and in all 10 dogs the left and right thyropharyngeal and cricopharyngeal muscles were innervated by the ipsilateral Xph nerve but not always by the IX nerve. After completion of the stimulation experiment, the right glossopharyngeal parent trunk was transected in 5 of the 10 dogs and the right pharyngeal branch of the vagus nerve was transected in the other 5 dogs. The nerve ends were sufficiently separated to eliminate the possibility of regeneration. Two to 4 weeks later the spontaneous EMG activity was recorded from the left and right hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles and the muscles of the soft palate and the tongue. Under general anesthesia, the bipolar needle electrode (Disa 18K80, similar to Danica 9013L060) was inserted into the muscles as shown in Figure 3.3. Unilateral transection of the parent trunks of the glossopharyngeal nerve or the pharyngeal branch of the vagus nerve resulted in denervation potentials in the ipsilateral pharyngeal muscles, but not in the contralateral muscles or in the tongue. Four weeks later the corresponding contralateral trunk of the IX nerve (5 dogs) or the Xph nerve (5 dogs) was transected. EMG recordings of spontaneous activity in the pharyngeal muscles revealed denervation potentials in the left and right hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles and the soft palate in all 5 dogs in which the Xph was transected bilaterally, but not in all muscles in the 5 dogs in which the IX nerve was transected bilaterally. Contrast videofluorography revealed more severe irregularities in the swallowing action after transection of the pha-

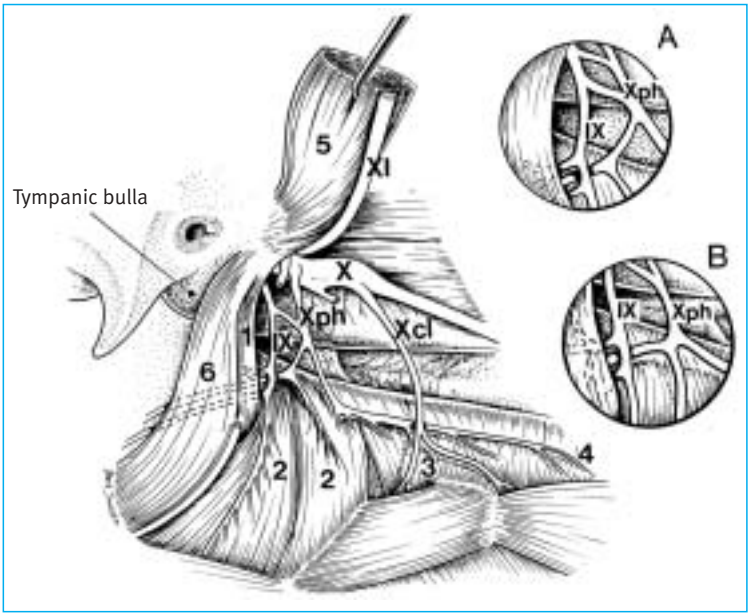


Figure 3.2:
Anatomy of the pharyngeal plexus, based on dissections in 5 canine cadavers. Branches of the glossopharyngeal nerve (IX) join branches of the pharyngeal branch of the vagus nerve (Xph) in various ways, resulting in the combined innervation of the pharyngeal muscles by both nerves. Inset A: Here one of two parallel branches joins the pharyngeal branch of the vagus nerve. Inset B: Here a tripartite division provides the branch supplying the hyopharyngeal muscle. 1 = stylopharyngeal muscle, 2 = hyopharyngeal muscle, 3 = thyropharyngeal muscle, 4 = cricopharyngeal muscle, 5 = sternocephalic muscle, 6 = styloglossal muscle. (From: Venker-van Haagen AJ, Hartman W, Wolvekamp WThC. Contributions of the glossopharyngeal nerve and the pharyngeal branch of the vagus nerve to the swallowing process in dogs. Am J Vet Res 1986; 46(6): 1300–1307, Fig. 2).

ryngeal branch of the vagus nerve than after transection of the glossopharyngeal nerve, and the difference was greater after transection of the corresponding contralateral nerve. No abnormalities in swallowing were observed in the dogs in which both glossopharyngeal nerves had been transected, but those in which the pharyngeal branch of the vagus nerve had been transected on both sides had various degrees of dysphagia. In summary, the pharyngeal plexus distributes the motor supply to the pharyngeal muscles, and of the two motor nerves supplying the innervation via this plexus, the pharyngeal branch of the vagus nerve is functionally more important than the glossopharyngeal nerve. The pharyngeal muscles are innervated unilaterally by both of the parent trunks of the pharyngeal plexus.[65]

Continuous EMG recordings were made from paired wire electrodes implanted in the left and right hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles of 5 normal beagles to determine the sequence of activity in each muscle and the combined muscle activity, both at rest and during swallowing of food. The data were digitized during 30-second periods and stored on diskette for further analysis. In all 5 dogs the pattern of muscle activity during swallowing was distinct, in a constant sequence (hyopharyngeal, thyropharyngeal, cricopharyngeal), and bilaterally synchronous. During eating, there were 5 to 12 short periods of synchronous activity in each muscle between swallowing actions, tentatively interpreted as bolus formation (Figure 3.4). During the resting period, there were longer periods of activity that were synchronous with respiration.

Peripheral feedback regulation in swallowing.

Control of the swallowing motor sequence by peripheral feedback is strongly suggested by the finding in humans that the amplitude and duration of electromyographic activity in the mylohyoid, geniohyoid, and genioglossus muscles depend in part on the consistency of the

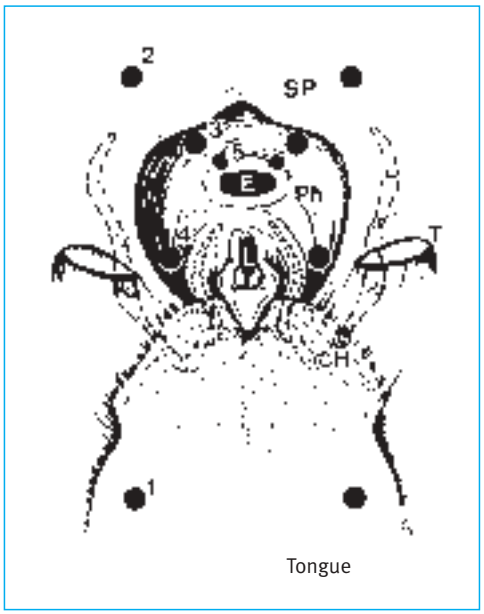


Figure 3.3:
Diagram of the oral view of the pharynx in the dog. The black dots indicate placements of the electrode in the tongue (1), the soft palate (2), thyropharyngeal muscle (3), hyopharyngeal muscle (4), and cricopharyngeal muscle (5). H = hyoid bone, T = tonsil, SP = soft palate, Ph = pharynx, E = esophageal inlet, L = laryngeal inlet. (From: Venker-van Haagen AJ, Hartman W, Wolvekamp WThC. Contributions of the glossopharyngeal nerve and the pharyngeal branch of the vagus nerve to the swallowing process in dogs. Am J Vet Res 1986; 46: 1300-1307, Fig. 1).

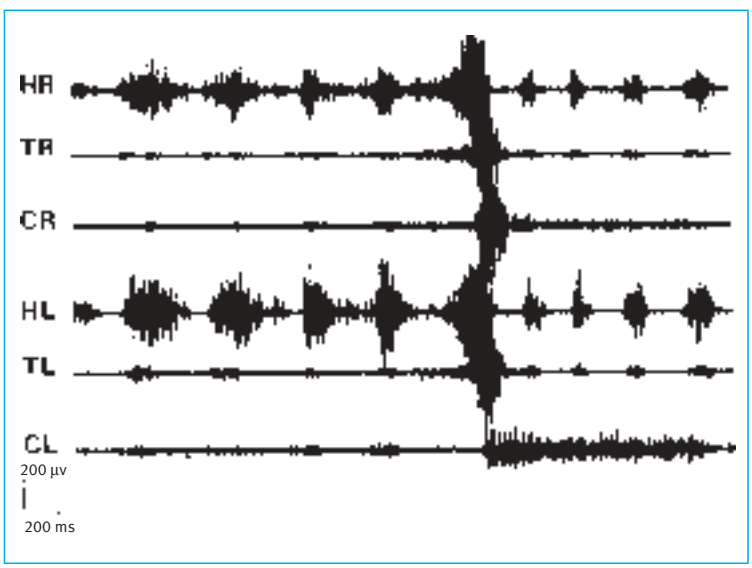


Figure 3.4:
Electromyographic recordings showing the sequence of activity in the right hyopharyngeal (HR), right thyropharyngeal (TR), right cricopharyngeal (CR), left hyopharyngeal (HL), left thyropharyngeal (TL), and left cricopharyngeal (CL) muscles during swallowing in a clinically normal dog. During eating, short synchronous periods of activity were visible in the pharyngeal muscles on both sides, tentatively interpreted as bolus formation. (From: Venker-van Haagen AJ, Hartman W, Van den Brom WE, Wolvekamp WThC. Am J Vet Res 1989; 50: 1725–1728, Fig. 1).

bolus.[24, 26] In dogs the regulation of swallowing was markedly disturbed by transection of peripheral components of the pharyngeal plexus,[66] while stimulation of the peripheral pathways influenced the contraction timing during swallowing.[69]

3.2 History and clinical signs

3.2.1 History

The medical history in diseases of the pharynx usually reveals specific problems caused either by dysfunction of the airway through the oropharynx or nasopharynx or by difficulty in swallowing (dysphagia). In some cases the appearance of the pharyngeal mucosa may suggest a systemic disorder, but in all cases additional questions are asked about any changes in the animal's general condition, appetite, eating, drinking, physical activity, and habits. The answers to these questions together with a general physical examination will provide an impression of the patient's condition.

3.2.2 Clinical signs

Dyspnea. Signs of dyspnea in pharyngeal disease are caused by obstruction of the nasopharynx or the oropharynx. Obstruction of the laryngopharynx primarily hinders the passage of air through the nasopharynx and hence it also results in signs of nasopharyngeal obstruction. Severe obstruction of the oropharynx or laryngopharynx hinders the passage of food as well as air and thus causes dysphagia as well as dyspnea. Large masses in the nasopharynx can also obstruct the oropharynx and thus also result in dysphagia.

When dyspnea is caused by nasopharyngeal obstruction the signs are those of more forceful inspiration, which usually produces a snoring stridor. In dogs these sounds may be obvious and more pronounced during sleep. In cats the sounds may be soft and sometimes difficult to distinguish from the wheezing stridor caused by nasal obstruction. The dyspnea may be mild or severe but in principle it is not life threatening, because opening the mouth allows air to pass through the oropharynx to the larynx. However, both cats and dogs tend to avoid mouth breathing even in this situation, and almost to the point of suffocation. The medical history and clinical signs are similar to those of obstructive nasal disease but differ in that nasal discharge is absent and the stridor is more snoring than wheezing. The sense of smell (olfaction) is normal as long as there is some air passing through the nasal cavity, but absent when there is total obstruction. Total obstruction of the nasopharynx is recognized in dogs by mouth breathing and frequent panting. In cats mouth breathing must also occur, but cats tend to be very calm and they can let air slip in and out of the mouth via an almost invisible parting of the lips near the commissures, with only now and then a quick opening and closing of the mouth during inspiration. In both cats and dogs the clinical signs of dyspnea caused by nasopharyngeal obstruction are obvious, but in cats the observation requires more time and ingenuity.

When dyspnea is caused by oropharyngeal obstruction the most likely finding will be a mass or edema, and the dyspnea will be accompanied by gargling sounds. If the nasopharynx is obstructed while the mouth is closed, both dogs and cats open and close the mouth during respiration and the gargling sounds occur during inspiration via the open mouth. The signs of dyspnea are usually accompanied by loss of appetite, salivation, a preference for sleeping with the neck extended, and fear of palpation of the pharyngeal area. A fetid breath is another sign of a mass in the oropharynx with lesions in the mucosa.

Dysphagia in pharyngeal disease. A history of difficulty in swallowing should be differentiated from a wide variety of signs such as coughing, vomiting, regurgitation, loss of appetite, and even nasal discharge.[46] Swallowing is the action in which food or liquid is passed from the oropharynx to the esophagus and thence to the stomach. The signs of dysphagia involving the pharyngeal phase are gagging, choking, and repeated swallowing of one bolus. The food may be regurgitated and will be seen to be covered with thick mucus, and the dog may eat it again. When there is severe dysphagia and part of the food or liquid passes into the larynx and trachea, there is immediate coughing and, after successful ejection of the misdirected particles of food into the pharynx and after signs of choking, swallowing is repeated. If the food or liquid is ejected into the nasopharynx, snoring and sneezing may follow. When given solid food, a dog with severe dysphagia may stop eating and walk away from the pan. When this dog drinks it drools much of the water and mucoid saliva into and around the water pan. Knowledge of these signs can be helpful in composing meaningful questions for the history and in recognizing dysphagia.

When swallowing is painful the animal may turn away from both food and water after attempting to eat. This can be a sign of severe inflammation or trauma. An obstruction in the oropharynx causes repeated attempts to swallow as long as the food has not passed the obstruction.

In cats dysphagia usually results in repeated attempts to swallow, but a cat is soon discouraged and stops eating. The history and clinical signs of dysphagia in cats are therefore very difficult to distinguish from not eating per se. Sometimes salivation is the only indication of difficulty in swallowing (even saliva) and of pharyngeal pain.

Dysphagia can be life-threatening because of the resulting emaciation and because aspiration pneumonia may also occur. Clinical examination and special diagnostic procedures will help to establish the diagnosis and to reveal signs of complications.

3.3 Special diagnostic techniques

Direct inspection of the pharynx with a laryngoscope is the most important diagnostic procedure in disorders of the oropharynx and laryngopharynx. Under anesthesia it is possible to inspect the soft palate, the base of the tongue, the palatine tonsils, and the hypopharynx including the laryngopharynx, with a minimum of instruments. The soft palate can be retracted with a forceps to facilitate inspection of the nasopharynx, in which any large masses can be recognized and biopsies can be taken. Complete inspection, including the rostral part of the nasopharynx, the caudal part of the choanae, and the openings of the auditory tubes, can be performed with a flexible endoscope capable of 180° retroflexion and having a working channel for the passage of biopsy forceps.

3.3.1 Pharyngoscopy

The animal must be anesthetized. The instruments needed are a laryngoscope fitted with a blade suitable for the size of the animal, a long tissue forceps, a Senn retractor with blunt prongs,[40] and several sizes of endotracheal tubes. After premedication with medetomidine, anesthesia is induced by intravenous administration of propofol to effect.

For inspection of the oropharynx and the hypopharynx, the animal is placed in the sphinx posture with its head supported by an assistant standing at its side. The assistant opens the animal's mouth and extends its neck, using one hand to raise the upper jaw and the other to depress the lower jaw and flatten the tongue. The laryngoscope is introduced over the tongue and the mouth, oropharynx, palatine tonsils,

soft palate, and base of the tongue are inspected. Then the hypopharynx is inspected by using long forceps or a Senn retractor to extend visibility caudal to and above the soft palate. It is advantageous to perform pharyngoscopy without an endotracheal tube in place, but if the animal is dyspneic endotracheal intubation precedes pharyngeal inspection, and before biopsies are taken the animal is always intubated and the cuff is inflated, to prevent leakage of blood into the trachea and bronchi.

For complete inspection of the nasopharynx, a flexible 5-mm diameter endoscope capable of 180° retroflexion is used. The animal is intubated and placed in lateral recumbency with the mouth open and fixed in that position. The nasopharynx is very sensitive and care should be taken that the level of anesthesia is deep enough to prevent pain and closure of the mouth. A wooden block or metal spreader placed between the upper and lower canine teeth prevents the animal from biting the endoscope. The endoscope is introduced with its tip straightened until it passes the caudal rim of the soft palate and then the tip is retroflexed 180° as it is introduced into the nasopharynx. Careful manipulation will bring the rostral part of the nasopharynx into view. Biopsies can be obtained with a biopsy forceps introduced through the working channel of the endoscope. This use of the flexible endoscope is especially helpful for diagnosis in the area of the choanae, an area often inadequately inspected by rhinoscopy or by retraction of the soft palate to allow inspection of the nasopharynx. The flexible endoscope facilitates recognition of both neoplasms and foreign bodies.[77]

3.3.2 Diagnostic imaging of the pharynx

Radiographic examination. Plain radiographs of the pharyngeal area are especially useful for recognition of structures obstructing the airway and the passage of food; for locating radiopaque foreign bodies such as stones, needles, and bones; and for inspection of the hyoid bone for fracture and for arthritis of its joints, either of which can cause dysphagia. Laterolateral radiographs usually provide sufficient information. When the exact location of a foreign body (especially a needle) is needed to enable an attempt to remove it, a ventrodorsal projection is also necessary. Laterolateral radiographs are useful for detecting processes in the nasopharynx. Although the two rami of the mandible overlie the rostral nasopharynx in this projection, experienced radiologists are able to resolve this problem. Radiographs do not provide information about pharyngitis, tonsillitis, or congenital hypoplasia of the soft palate. Laterolateral radiographs of the pharynx in brachycephalic dogs can be misleading, because a thick soft palate resembles a large obstructive mass, while in reality the palate is arched dorsally, leaving space for passage of air and food. The diagnosis of an overlong soft palate should therefore not be made on the basis of radiographs but by pharyngoscopy.

Computed tomography and MRI are indispensable in determining the location and extent of neoplasms in the pharyngeal area. The nasopharynx and the base of the skull are difficult to assess by radiography but can be imaged clearly by CT and MRI. Lymphoid tissue, both hypertrophied and normal, is commonly seen in the nasopharynx; it is usually symmetrical. A benign lymphoid tumor of the nasopharynx is limited to the mucosa and has a smooth outline; any extension into the deeper plane should be considered suspicious of invasion and thus malignancy. Nasopharyngeal carcinomas are also infiltrative.[32] The base of the skull is an anatomical region that can be evaluated best by CT. The unique ability of CT to image both bone and soft tissue structures with excellent tissue-contrast discrimination makes it ideal for studying the base of the skull. Both CT and MRI provide an accurate assessment of the

total extent of the disease process and precise identification of intraorbital or intracranial extension of a tumor (e.g., craniopharyngioma) or inflammatory process. CT is superior to MRI for details of a bony process and MRI is superior to CT for details of soft tissue process. In many instances they are complementary.[32]

Contrast videofluorography is almost indispensable for imaging the dynamics of the swallowing process in dogs. Recordings are made while the animal eats food (ground meat) mixed with barium. The procedure needs the cooperation of the patient and is extremely time consuming, even in a routine set-up. Most dogs are cooperative, but fasting for more then 1 day may be necessary before some will eat in the unfamiliar surroundings. These studies are virtually impossible in cats. All phases in the swallowing process—oral, pharyngeal, and esophageal—can be studied repeatedly once the video recordings have been made. The diagnosis is based on a detailed description of the process of formation of the bolus, the relaxation of the cricopharyngeal muscle, and the passage of the bolus through the cervical and thoracic esophagus and into the stomach. Leakage of liquid or food into the larynx and the trachea can be recognized with certainty on the video recordings and is a definite indication of dysfunction of the swallowing process.[46, 65, 66, 71]

3.3.3 Electromyography of the pharyngeal muscles

Electromyography (EMG) of the pharyngeal muscles is useful in dogs with signs of dysphagia when no abnormalities are found by pharyngoscopy. After premedication with medetomidine and induction of anesthesia with propofol, the dog is placed in the sphinx posture and the mouth is held open as described above for pharyngoscopy. The appropriate level of anesthesia is that at which there is just a low degree of normal electromyographic activity in the pharyngeal muscles, synchronous with respiration. The bipolar needle electrode (Danica, 9013L0601) is inserted into the muscles of both halves of the tongue and soft palate and into the bilateral thyropharyngeal, hyopharyngeal, and cricopharyngeal muscles, to record the spontaneous muscle action potentials (Figure 3.3). Fibrillation potentials were found to predominate in denervated pharyngeal muscles.[65] In dogs with histological evidence of muscular dystrophy in the pharyngeal muscles there were fibrillation potentials, positive sharp waves, and, most characteristic, abundant complex repetitive discharges.[45] Following electromyography of the pharyngeal muscles, recordings can be made from the cervical part of the esophagus with the aid of a long holder for the bipolar needle electrode.[45]

3.4 Congenital deformities and disorders of the pharynx

3.4.1 Hypoplasia of the soft palate

Cleft lip and palate are common congenital malformations in dogs and cats, and are characterized by midfacial soft tissue and skeletal fusion abnormalities. Congenital clefts can be associated with exposure to such drugs as glucocorticoids, metronidazole, or griseofulvin, or can be inherited as a multifactorial genetic trait.

The premaxilla and the alveolus form the primary palate. The palatal processes fuse to form the secondary palate together with the nasal septum, and the palate grows from rostral to caudal. Ossification of membrane-formed bone occurs in beagle embryos at 32 days.[15] Failure of ingrowth of mesodermal tissue at this point results in lack of cohesion of the palatal segments, resulting in a cleft.

Puppies and kittens with visible congenital abnormalities are often destroyed on the first day after birth. Many breeders check newborn

puppies or kittens for cleft palate and if a cleft is found they euthanize the affected animal. When a cleft is diagnosed on the first day of life, it is usually wide and the rostral hard palate is incomplete. If the puppy or kitten is allowed to live and is given extra care, such as additional feeding to maintain growth, it will survive. After weaning, oropharyngeal dysphagia and nasal discharge will become obvious, making the animal undesirable as a pet. An incomplete repair will have similar results, but by that time it will be difficult to advise euthanasia. The decision for or against euthanasia should be based on a fair evaluation of the possibility of successful repair of the cleft and the decision should be made during the very first days of life.

3.4.2 Congenital malformation of the soft palate

Congenital malformation of the soft palate usually involves incomplete closure between the nasopharynx and the oropharynx and the clinical signs of this hypoplasia depend upon the size of the opening. A small opening just involving the soft palate usually only leads to occasional misdirection of milk or food. Since the occurrence of this is irregular and the signs do not lead to continuous nasal discharge, the diagnosis is often not made until the animal is over 1 year of age. Hypoplasia of the soft palate may also be nearly complete, allowing milk or food to leak freely into the nasal cavities. This becomes evident when the animal no longer nurses, holding its nose slightly upward, but eats and drinks from a pan. During swallowing the nasopharynx should be separated from the oropharynx by circular closure of the intrapharyngeal opening, but in hypoplasia of the soft palate this structure is missing. Inspection of soft palate defects should preferably be done under anesthesia, the aim being to estimate whether enough tissue is available for successful functional repair.

Surgical repair of a cleft in the soft palate is most successful when soft palate muscle is available and the cleft is on the midline. The soft palate is a sheet of muscle having an important role in swallowing, especially in closing off the nasopharynx to prevent leakage of food and water into the nose. Its repair is successful if its muscular function is restored. Care should be taken to suture the nasopharyngeal mucosa and the oropharyngeal mucosa separately. The muscle layer is included in the closure of one or the other. It is advantageous to perform surgery when the dog or cat is over 3 months of age and the tissues are more firm.

When the soft palate is incomplete the choice of technique is based on analysis of the possibilities. Certain points are essential for successful surgery: a) transposition of tissue flaps rather than use of foreign material; b) adequate mobilization of the flaps; c) preservation of the main palatine vessels; d) closure of the soft palate in two layers, nasopharyngeal and oropharyngeal, including the muscle layer in one of them; and e) avoidance of using forceps to handle the edges to be sutured.[39]

If surgical repair is not possible, the animal can be kept alive with special care. Ideally, food and drink should be offered in such a way that the animal swallows with the nose pointed upward, to prevent reflux into the nasal cavity. Hand feeding of easily swallowed balls of food is most successful. Allowing the animal to drink from a stream of water above the level of its head also helps.

3.4.3 Hyperplasia of the soft palate

Hyperplasia of the soft palate is associated with brachycephaly and a relatively narrow pharynx. It is thought that the genetic defect responsible for shortening the nose does not affect the soft tissue, the result being too much tongue and soft palate in a narrow pharynx. The clinical signs of an overlong soft palate are snoring,

regurgitation, and dyspnea, usually increasing in severity during the second and third years of the dog's life. The pharyngeal disproportions are not the same in all brachycephalic dogs. In some the pharyngeal mucosa and soft palate are very thick and the musculature is insufficient, which results in snoring during closed-mouth breathing. Little can be done for these dogs when dyspnea eventually develops. This is in contrast to those with an overlong soft palate, which can be reduced in length so that it no longer covers the laryngeal inlet.[71]

The surgical technique to shorten an overlong soft palate is simple but the patient's recovery following surgery may be complicated by obstruction of the airway due to swelling of the mucosa of the remainder of the soft palate. A short-acting anesthetic should be used, for which a relatively safe method is premedication with medetomidine followed by induction with propofol to effect. After endotracheal intubation the dog is placed in the sphinx posture with the upper jaw fixed, the neck extended, and the lower jaw hanging down. To improve visibility, the tongue is unfolded and, without traction, is lightly bound to the endotracheal tube and the lower jaw with a wet bandage. The soft palate is grasped with tissue forceps and withdrawn until its free border is brought into view. The forceps is then reattached at the midpoint of the free border. A moist gauze pad is put in place to prevent the leakage of blood into the trachea and esophagus. By gently retracting the soft palate and positioning it over the gauze pad, the portion to be removed can be determined. The two limiting landmarks for removal of the caudal part of the soft palate are the palatine vessels laterally and the muscles of the soft palate rostrally. The function of the pharyngeal isthmus is critical for closing off the nasopharynx during swallowing, to prevent leakage of food and liquid into the nasal cavities. The muscles are not clearly visible but a good rule of thumb is that the relatively wrinkled part of the soft palate does not contain muscles. It is helpful to first incise the oropharyngeal mucosa, which is well within view, and then incise the nasopharyngeal mucosa together with the adipose tissue along the same line. The oropharyngeal mucosa is sutured to the nasopharyngeal mucosa with interrupted sutures, using absorbable suture material with good knot security. The moist gauze pad is removed and the result of the soft palate correction is examined. There should be no residual bleeding. The wrapping around the tongue, lower jaw, and endotracheal tube is removed and the dog is allowed to recover from anesthesia. The endotracheal tube should not be removed until the dog is awake and ready to walk. French bulldogs can have such a narrow pharynx that a tracheostomy is absolutely necessary during the first 4 days after surgery. The success of palate shortening in diminishing the dyspnea is much dependent on the narrowness of the abnormal upper airway passages.

3.4.4 Choanal atresia

Bilateral atresia of the choanae is the most frequently encountered congenital nasal anomaly in humans and is a common cause of neonatal respiratory distress. Failure of the breakdown of the buccopharyngeal membrane is considered to be the cause. The symptoms are respiratory distress from birth until the neonate begins to cry.[54] Most pups with bilateral choanal atresia probably die before the condition can be diagnosed, yet occasionally an affected pup survives and one such case has been reported.[6] In bilateral choanal atresia there is no passage of air through the nose and thus breathing through the mouth is essential for survival. In unilateral choanal atresia the passage of air may be sufficient and the diagnosis may be delayed until much later in life. The diagnosis of bilateral choanal atresia can be confirmed by the failure of a catheter to pass through the nose into the nasopharynx, by direct examination of the nasopharynx via the mouth with a

flexible endoscope capable of 180° retroflexion, and by a CT scan. Treatment was attempted in one case by applying firm pressure against the atretic bony plate via the nose, in order to create an opening. This only remained open for about two weeks and repeating the procedure did not produce a permanent opening. Although this dog could live by breathing through its mouth, it was later euthanized because of the almost continuous dyspnea. In a reported case in a 20-month-old Shih Tzu, a transpalatal approach was used and also in this case the obstruction recurred.[6] In human surgery it has been pointed out that the challenge, regardless of the approach, is to provide adequate mucosal lining of the new choanae to prevent the formation of granulation tissue which culminates in stenosis. This requires perforation of the atretic plate and stenting for 6 weeks, together with preservation of mucosal flaps.[54] In a review of choanal atresia in 78 children, surgery was eventually effective in establishing a patent airway in all of them, nearly all having undergone correction by the transnasal approach. On average, 2.7 procedures were required in children with unilateral atresia and 4.9 procedures were required in those with bilateral atresia.[55]

3.4.5 Craniopharyngioma (Rathke's pouch tumor)

The craniopharyngioma is derived from rests of epithelium of the primordial craniopharyngeal canal, or Rathke's pouch.[51] Rathke's pouch is a diverticulum arising from the embryonic buccal cavity, from which the anterior lobe of the pituitary gland is developed. In humans craniopharyngiomas are most commonly diagnosed in children and young adults and are usually benign, but occasionally give rise to malignancy. They can arise at any point along the craniopharyngeal canal and therefore some lie within the sella turcica and others are external to it.[51] In dogs the craniopharyngeal canal occasionally persists in the adult, particularly in bulldogs.[18]

Craniopharyngiomas are composed of well-differentiated epithelial elements including cysts and ameloblasts, as well as bone. This tumor is rarely encountered first in the nasopharynx and is most often located intracranially above the sella turcica. In humans it accounts for 10 to 15 % of all intracranial neoplasms in childhood and adolescence. In dogs craniopharyngiomas are rare and only a few reports have been published.[12, 13, 22, 38]

Clinical signs of craniopharyngioma. The clinical signs include anisocoria, strabismus, progressive depression, somnolence, seizures, extraocular motor paralysis, sudden blindness, and hypopituitarism causing growth hormone deficiency, secondary hypothyroidism, and secondary hypoadrenocorticism. The signs are related to the site of origin of the tumor, its proximity to the pituitary gland and optic chiasm, and its size. In humans craniopharyngiomas occasionally reach 8 to 10 cm in diameter.[51] The tumor is found on laterolateral radiographs and by CT. Therapy in humans consists of total or partial surgical removal together with radiation. The 10-year survival rate is 90 %. Despite major microsurgical advances, however, total removal of these tumors is associated with a high risk of death, endocrinological complications, and behavioral dysfunction. There have been no reports of surgery for craniopharyngioma in dogs and the diagnosis has been based on necropsy findings.[12, 22, 38] No reports of craniopharyngiomas in cats have been found.

The histological pattern is variable and there is a wide spectrum of cell types, recalling the cells of the enamel organ of the tooth. These tumors are thus also known as ademantinomas or ameloblastinomas.[51] Nests or cords of stratified squamous or columnar epithelial cells are embedded in a loose fibrous stroma. Multiloculated cysts often contain papillary projections

of epithelium. Calcification and metaplastic bone formation occur in the necrotic centers of the solid tumors, as well as in the cystic variety, and are of considerable radiological diagnostic importance.[51]

3.5 Pharyngitis

3.5.1 Nasopharyngitis

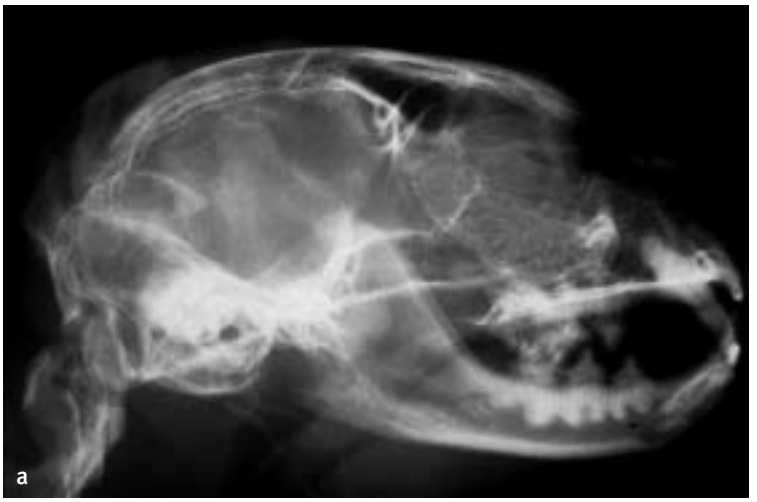
The anatomical position and functional features of the nasopharynx warrant separate consideration. Inflammation of the nasopharynx is often associated with rhinitis, but may occur separately. The clinical signs of nasopharyngitis are snoring, reverse sneezing, and dyspnea during closed-mouth breathing. Aerophagia may occur in severe dyspnea. The nasopharyngeal mucosa may be hypersensitive to physical contact during oral and pharyngeal inspection under sedation. Mucopurulent material may be found at the intrapharyngeal opening.

Viral nasopharyngitis is one of the signs in feline herpesvirus-1 and feline calicivirus infections. These virulent feline respiratory pathogens account for at least 80 to 90 % of infectious upper respiratory airway disease in cats.[7, 20] They affect the mucosa of the pharynx, nose, and nasopharynx. Pain in the nasopharyngeal area may be a prominent sign on the first day of the viral infection. The cat abruptly stops eating and if oral inspection is performed under sedation, light pressure on the soft palate will be found to cause an adverse reaction, although the oral surface of the palate does not appear to be especially inflamed. Salivation begins a day later and the disease becomes more easily recognized by its clinical signs, which persists for 2 to 3 weeks. As long as nasopharyngeal pain continues, intravenous fluid therapy is necessary to maintain homeostasis, and pharyngeal pain should be suppressed by analgesia. Forced feeding is contraindicated while there is pharyngeal pain. Prevention and other aspects of these viral diseases are discussed in Chapter 2.5. In dogs with viral rhinitis there are no obvious signs that nasopharyngitis plays an important role.

Primary bacterial nasopharyngitis is uncommon in both dogs and cats. Secondary bacterial nasopharyngitis may develop by extension from bacterial rhinitis, or be caused by a congenital or traumatic nasopharyngeal fistula, foreign bodies, polyps in the nasopharynx, or insufficient closure of the nasopharyngeal opening during swallowing. In all of these situations the bacterial infection is treated in the course of treating the primary disease.

Reverse sneezing. Inflammatory irritation of the nasopharyngeal mucosa may cause reverse sneezing. This symptom is caused by the untimely closing of the intrapharyngeal opening, independent of swallowing. It consists of 1 to 2 minute periods of severe inspiratory dyspnea characterized by repeated short snoring sounds, extension of the neck, bulging of the eyes, and abduction of the elbows. It is a familiar sign in dogs and although cats have a similar reflex, the sound and the manifestation are modest compared to those in dogs. Since the symptom is alarming and severe dyspnea is obvious, owners may quite understandably present the problem as an emergency. And since dogs do not demonstrate reverse sneezing on command, the diagnosis depends on the medical history. The symptom is difficult for owners to describe and since it does not occur in humans, it is very helpful if the person taking the history recognizes the signs and can mimic the sound. This can be done by closing the caudal nasopharynx by pressing the base of the tongue upward, while stretching the neck, turning the elbows outward, opening the eyes wide, and then trying to inhale through the nose. This evokes immediate recognition by owners. The treatment of reverse sneezing should consist of

curing the nasopharyngeal mucosal irritation, but once a dog has acquired the habit of reverse sneezing, such therapy usually fails to change the habit. However, symptomatic therapy is usually effective. It consists of activating the dog's swallowing reflex, which culminates in reopening of the intrapharyngeal opening and thus interruption of the reverse sneeze. Reverse sneezing will recur in more or less frequent periods, but this symptomatic therapy relieves the dog each time. It may even prevent further irritation of the nasopharyngeal mucosa. The owner should be given an explanation of the reflex and shown how to induce swallowing by massaging the dog's throat or briefly closing the nares.



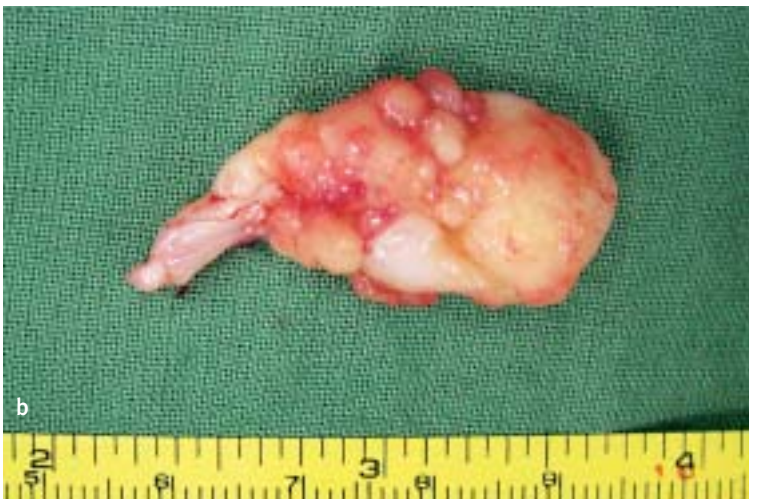


Figure 3.5 a, b: Laterolateral radiograph of a cat's head showing a density produced by a polyp in the nasopharyngeal area (a). The same polyp after removal (b).

Nasopharyngeal polyps are polyps found in the nasopharynx but not necessarily originating in the nasopharyngeal mucosa. The most common polyp in the nasopharynx of the cat originates in the middle ear and descends to the nasopharynx via the auditory tube. The mucosa of the middle ear in some cats is affected by polypous inflammatory disease. Bacterial infection causes focal hypertrophy of the mucosa, leading to polyps which are not true neoplasms.[52] They develop on a stalk and extend through the auditory tube to reach the nasopharynx, where they may grow substantially—a diameter of 3 cm is not uncommon—and thus form a nasopharyngeal polyp (Figures 3.5 a, b). A polyp that arises in the mucosa of the middle ear and grows through the tympanic membrane into the external ear canal is termed a middle ear polyp. It will be found to consist of focal accumulations of edema fluid accompanied by hyperplasia of the submucosal connective tissue and a variable inflammatory infiltrate of eosinophils, plasma cells, and lymphocytes.[52] It is covered by ciliated columnar epithelium and goblet cells.[42] Secondary bacterial infection and inflammation of the nasopharyngeal mucosa is to be expected.

The clinical signs of a nasopharyngeal polyp are due to obstruction of the nasopharynx, inspiratory dyspnea being the principal effect. Food intake is interrupted because of blockage of the passage of air through the nose, but there is no nasal discharge initially. After a long interval secondary inflammation in the nasal cavity may cause rhinitis. Diagnosis is based on the clinical signs and on a laterolateral radiograph of the pharyngeal area, which reveals a mass in the middle of the nasopharynx, depressing the soft palate. Oropharyngeal inspection under sedation reveals ventral depression of the soft palate, which sometimes partly blocks the view of the larynx and thus hinders endotracheal intubation (Figure 3.6). Treatment consists of removal of the polyp under anesthesia. The soft

palate is retracted rostrally or incised so that a curved mosquito forceps can be inserted between the dorsal wall of the nasopharynx and the polyp in order to clamp the polyp stalk. After rotating the forceps and polyp, to be certain that no nasopharyngeal mucosa is included, the polyp is removed by a sharp tug. Bleeding is controlled by pressing a gauze sponge into the nasopharynx at the location of the openings of the auditory tubes. If it has been necessary to incise the soft palate, the nasopharyngeal mucosa and the oropharyngeal mucosa are sutured separately, the muscle layer being included with one of them. An antibiotic is prescribed only if there is severe rhinitis and nasopharyngitis. In most cases the cat awakens breathing through the nose without dyspnea, and no further care is needed.

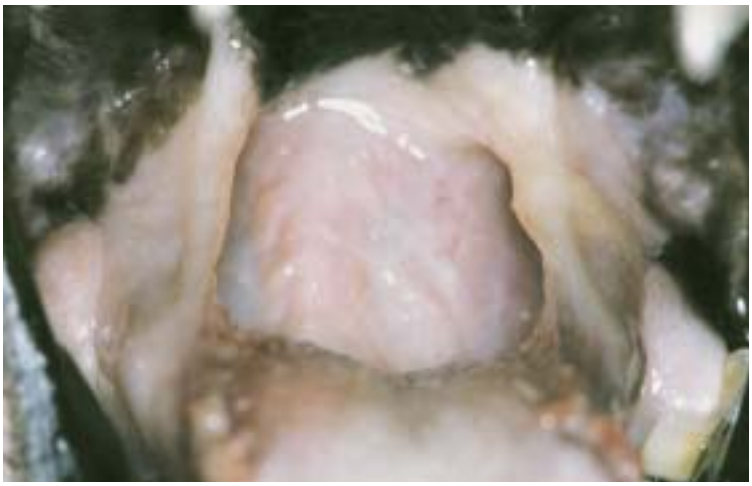


Figure 3.6: Depression of a cat's soft palate caused by a nasopharyngeal polyp.

A foreign body in the nasopharynx is accompanied by secondary bacterial infection. The usual route of entry is via the intrapharyngeal isthmus. When a cat chews on grass, a ball of grass and mucus may become lodged in the hypopharynx and a blade of grass may enter the nasopharynx during attempts to swallow the grass ball. The blade may remain behind when the grass ball finally passes the upper esophageal sphincter. Sometimes the blade of grass enters one of the nasal cavities and causes unilateral rhinitis, and sometimes snoring and repeated swallowing indicate that the blade of grass is also located in the nasopharynx and sometimes the laryngopharynx. Diagnosis is made by inspection of the oropharynx and nasopharynx under sedation, with rhinoscopy also if there is unilateral rhinitis. Small blades of grass are difficult to find and the 180°-retroflexion endoscope may be necessary. When small stones are thrown up in the air for dogs to catch, occasionally one passes right through the oral cavity and ends up in the esophagus or the nasopharynx (Figures 3.7 a, b). Its presence in the latter will be indicated by snoring, repeated swallowing, and sometimes reverse sneezing. Since the stone tends to become wedged into the rostral nasopharynx above the hard palate, its removal is complicated. A small midline incision is made through the rostral part of the soft palate, just large enough for the insertion of a curved mosquito forceps. The tip of the forceps is maneuvered rostral to the stone so that the stone can be drawn caudally into the nasopharynx over the soft palate, where it can be massaged into the laryngopharynx and be removed. Snoring, repeated swallowing, and sometimes reverse sneezing, together with a fetid breath may indicate decaying material in the nasopharynx. A piece of wood, plant, or meat may cause these signs. These materials are difficult to recognize on a laterolateral radiograph of the nasopharynx and may remain undetected for a long time. Especially more rostrally located foreign bodies can only be detected and removed by use of the 180°-retroflexion endoscope. Removal of a large foreign body may necessitate incision of the rostral part of the soft palate and even removal of a portion of the caudal part of the hard palate. Longstanding inflammation together with mucosal lesions may cause stenosis in the rostral nasopharynx.

Secondary nasopharyngeal stenosis due to chronic upper respiratory tract infection was

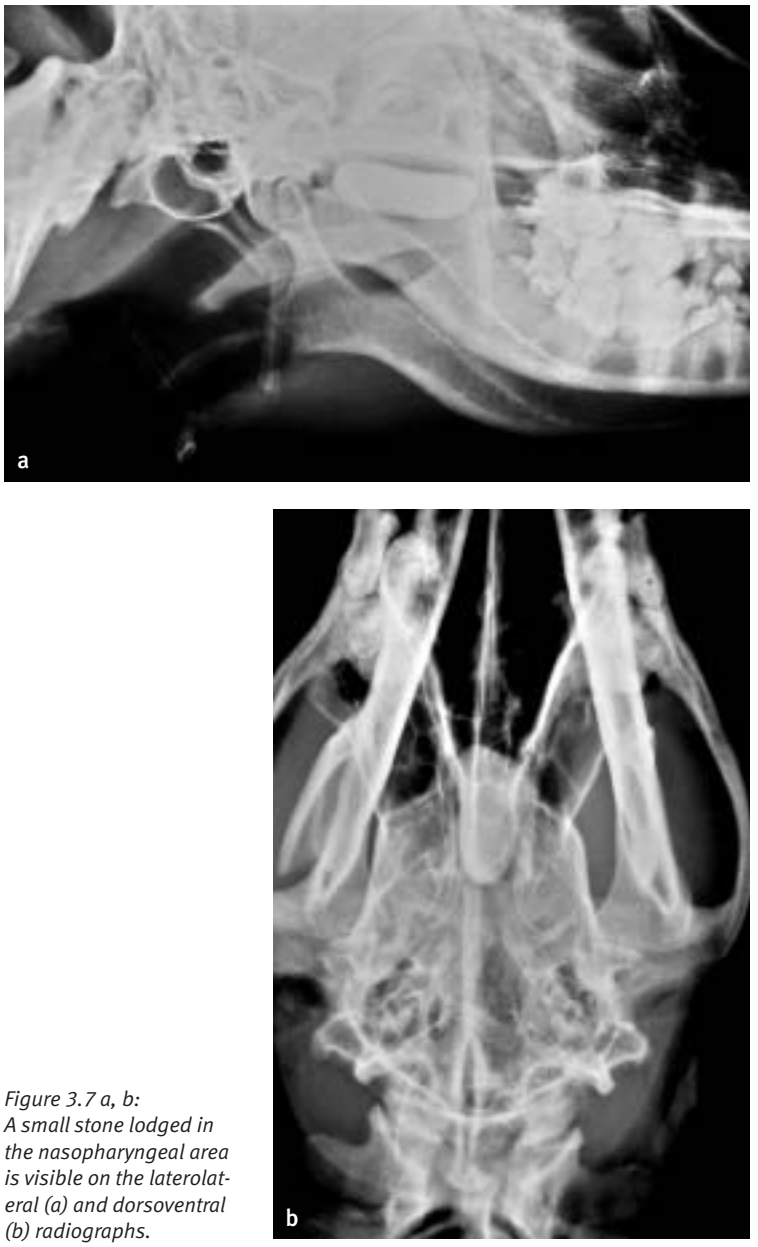


Figure 3.7 a, b:
A small stone lodged in the nasopharyngeal area is visible on the laterolateral (a) and dorsoventral (b) radiographs.

described in 4 cats.[37] The presenting signs in these cats were snuffling and partial nasal obstruction, but there was little nasal discharge. There were no remarkable findings by rostral rhinoscopy but attempts to pass a 3.5 French catheter encountered obstruction at the level of the choanae. Inspection with the aid of a dental mirror showed that the opening of the nasopharynx into the caudal nares—normally 6 mm dorsoventrally and 5 mm in width—was reduced to a pinhole. The obstruction was caused by a thin but tough membrane, which could be resected via an incision through the soft palate. Histological examination showed the membrane to consist of fibrous tissue with a mild infiltration with mast cells, eosinophils, and lymphocytes. The cat's breathing was normal after surgery and remained so for years.[37] The uncomplicated follow-up is encouraging, in contrast to the poor prognosis for surgery for congenital stenosis of the choanae (Chapter 3.4.4).

Regurgitation of food and liquid into the nasopharynx contributes to secondary bacterial infection and inflammation. Regurgitation may be caused by insufficient closure of the intrapharyngeal opening, associated with a congenital oropharyngeal cleft or hypoplasia of the soft palate (Chapter 3.4.1), or it may be the consequence of dysphagia (Chapter 3.8) The signs of nasopharyngeal regurgitation are sneezing or reverse sneezing while eating and drinking, and ceasing to eat or drink and walking away from the pan. These signs should be understood to represent an extremely unpleasant feeling arising from irritation of the very sensitive nasopharyngeal mucosa rostral to the intrapharyngeal opening. Signs of bacterial infection and inflammation are reverse sneezing, snoring, and repeated swallowing. Mucopurulent discharge may be found during nasopharyngeal inspection. Further diagnostic investigation of the cause of the dysfunction of intrapharyngeal opening includes nasopharyngeal inspection under sedation and contrast videofluorography. Treatment follows demonstration of the underlying cause (Chapter 3.8).

Fungal infection of the nasopharynx occurs in both dogs and cats. It can occur as an extension of fungal disease in the nasal cavity, as in aspergillosis in dogs, or as a granulomatous

process with concurrent fungal rhinitis, as in cryptococcosis in cats.

In dogs with nasal aspergillosis, *Aspergillus* may also be found in the nasopharynx, although its presence there causes no additional clinical signs and has no consequences for the therapeutic strategy. However, the mucosa of the nasopharynx may be found to be affected by toxins produced by *Aspergillus* in the nasal cavity.

Cryptococcus neoformans and *C. neoformans* var. *gattii* have been reported to cause granulomatous inflammation in the nasopharynx in cats.[5, 33] Infections by similar strains of *Cryptococcus* were reported to occur in the airways in horses.[50] All three reports originated from Australia. In dry environmental conditions, the organisms can become aerosolized.[30] An association has been found between *C. neoformans* var. *gattii* and exposure to *Eucalyptus camaldulensis* or the closely related *E. rudis* in horses.[50] The organisms usually colonize the upper respiratory tract, but occasionally become established in the lung or disseminated throughout the body. Cryptococcosis in cats is also reported from the United States, the infection there being associated with organically enriched soil and with immunosuppression in the cats by infection with feline leukemia virus.[30] The diagnosis of cryptococcosis in the nasopharynx is made by endoscopic examination using an endoscope with 180° retroflexion in order to obtain smears or biopsies of the mucosa, in which the round yeast is found in abundance. Staining fresh smears with India ink reveals the capsule of the yeast. Treatment consists of fluconazole orally for 2 to 4 months, sometimes extended to 6 months. No adverse reactions were observed in 28 cats cured in this way.[30]

3.5.2 Oropharyngitis and tonsillitis

In dogs and cats, acute pharyngitis is a typical sign of viral infections. It is characterized by pain, fever, and extreme discomfort. The dog or cat does not attempt to eat, often salivates, and makes ineffective swallowing movements. The neck is held extended and palpation of the pharyngeal area evokes reactions indicating discomfort.[71] The most common causes of these viral infections in cats are herpesvirus type 1 and calicivirus. In dogs, canine distemper, canine adenovirus-2, and canine parainfluenza virus may play a role. Primary bacterial infections causing pharyngitis are uncommon. Acute tonsillitis (of the palatine tonsils) is often associated with bacterial infections, beta-hemolytic streptococcus being the most prominent agent in dogs. The clinical signs are similar to those of acute pharyngitis, which usually accompanies acute tonsillitis. Since the palatine tonsils are usually enlarged in acute pharyngitis, it is not helpful to separate these two diseases in the acute stage. In cats, the palatine tonsils are also red and enlarged in herpesvirus type 1 and calicivirus infections, but the tonsillitis is related to the pharyngitis and specific bacterial tonsillitis has not been described.

Acute pharyngitis is often a symptom of systemic infection and the findings of a general examination and laboratory blood analysis will guide further steps in diagnosis and treatment. Symptomatic treatment consists of parenteral administration of broad-spectrum antibiotics to prevent additional bacterial infections even when a viral disease is suspected. Intravenous administration of fluids or parenteral/enteral nutrition may become life-saving. Analgesics are given during periods of extreme pain, usually the first 3 to 5 days. After 5 days liquid food may be given in small portions, several times a day, until the animal begins to exhibit interest in more usual foods. Pharyngeal pain during swallowing of food continues for 2 weeks.[71]

Dogs that have the habit of snapping at a bee or wasp sometimes succeed and then try to swallow what they have caught and are stung in the oropharynx or hypopharynx. The sting causes acute pharyngeal edema, leading to life-threatening obstruction of the airway. In these emergency cases a glucocorticoid will not act quickly enough to decrease the obstruction and a better approach is immediate sedation (medetomidine and propofol intravenously) and endotracheal intubation. The swelling will decrease spontaneously but this process takes several days, and tracheostomy and insertion of a tracheal cannula are indispensable for that period. The effects of the sting may be very painful at first and an intravenous catheter should be provided for fluid administration and intravenous feeding. The tracheal cannula may be removed after about 4 days, when inspection of the pharynx reveals a satisfactory airway. Drinking and feeding orally will be possible by that time.

Chronic pharyngitis is characterized by retching independent of the intake of food, normal swallowing, and sudden periods of pica. In most cases in dogs and in cats the cause is obscure. Inspection of the oropharyngeal and hypopharyngeal mucosa reveals thickening and irregular reddening. Since the cause of the disease is not clear, only symptomatic treatment can be given, directed at diminishing the irritation of the mucosa by giving moistened food and feeding it in smaller portions. Administering a spoon of syrup or honey before the meal is sometimes helpful, probably by stimulating salivation, which moistens the mucosa. Bouts of pica can be subdued by giving phenobarbital in a moderate dose upon the appearance of the first signs, which are licking and restlessness.

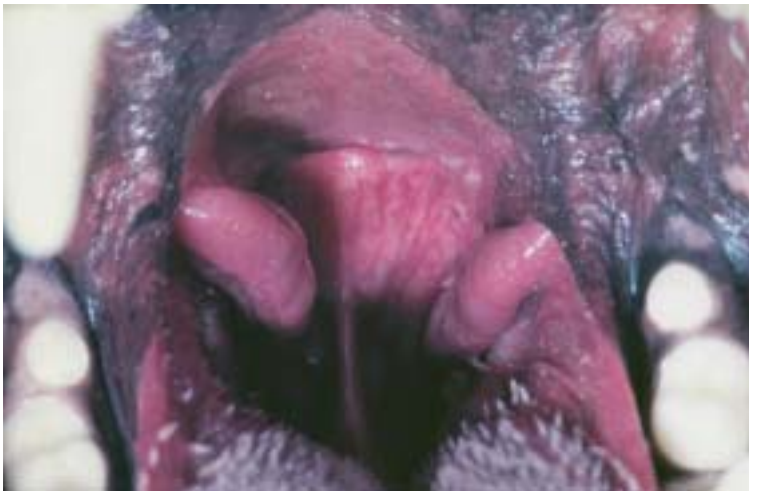


Figure 3.8: Chronic tonsillitis in a dog. The tonsils are enlarged and indurated, causing pain during swallowing.

Chronic tonsillitis is found more often in dogs than in cats. The tonsils are enlarged and indurated, and there is no response to repeated antibiotic treatment. The enlarged tonsils may cause obstruction and therefore pain during swallowing and even hinder the passage of air through the oropharynx (Figure 3.8). This is one of the rare indications for tonsillectomy.

Tonsillectomy of the palatine tonsils in dogs is performed under anesthesia and after endotracheal intubation. The dog is placed in lateral recumbency. An assistant sits at the dorsal side of the dog beside the head and neck and opens the dog's mouth and extends its neck, using one hand to hold the upper jaw and the other to hold the lower jaw and flatten the tongue. With the dog in this position the tonsillar fold on the lower side is retracted medially and the tonsil is grasped with an Allis tissue forceps, avoiding the deeper part of the tonsil covered by mucosa. The mucosa is then severed from the tonsil by the use of fine-pointed scissors, first at the rostral end—this is a firm attachment—and then along the lateral and the medial lines of attachment to caudal end. Some bleeding may occur when the caudal attachment is severed, but the tonsil is thereby removed and a collagen or gelatin sponge can be folded into the freed tonsillar fossa to provide hemostasis. The procedure may be complicated by bleeding but ligation of arteries is seldom necessary. Thermocauterization is avoided in this, as in most pharyngeal surgery. The usually minor portion

of the tonsil that is covered by mucosa may be the major part when the tonsil is chronically enlarged.[16] After the tonsil on the lower side has been removed, it may be advantageous to turn the dog to its other side for removal of the other tonsil.

When the tonsils of the cat are enlarged, feline leukemia or lymphoma should be suspected and biopsy of the tonsil will lead to the diagnosis.[71] See Chapter 3.6 for diagnosis and treatment of tumor in the pharynx and tonsils.

3.5.3 Pharyngeal mucocele

The pharyngeal mucocele is a rather common anomaly in dogs and rare in cats.[19, 74] A mucocele is a cyst arising from distended mucosal mucous glands. As the glands distend, they often rupture.[58] In the pharynx these cysts are found originating from the pharyngeal wall and sometimes from the soft palate. The signs are dyspnea and snoring caused by the voluminous cyst obstructing the oropharyngeal airway (Figure 3.9). The diagnosis is made by oropharyngeal inspection under sedation. To enable endotracheal intubation it may be necessary to grasp the cyst with a tissue forceps and move to one side until the larynx is visible. A 1-cm incision in the cyst allows its thick mucous contents to be removed, which usually solves the problem, but removal of part of the cyst wall may be necessary to open the airway sufficiently, after which sutures may be needed.

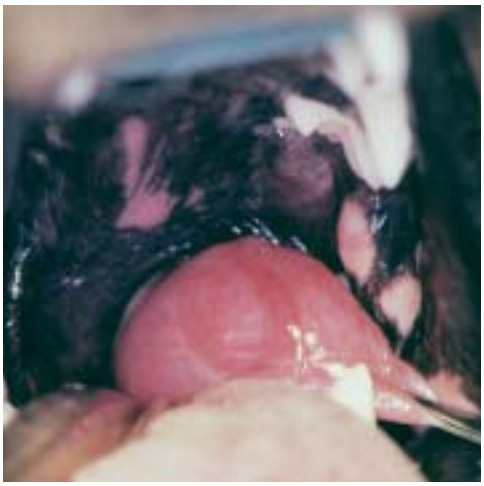


Figure 3.9: Pharyngeal mucocele causing obstruction in the oropharyngeal airway in a dog.

3.6 Tumors of the pharynx

Most tumors in the pharyngeal area of dogs and cats are malignant. In a survey of 361 oral and pharyngeal neoplasms in dogs, there was 1 malignant melanoma in the pharynx and there were 80 squamous cell carcinomas and 1 malignant melanoma in the palatine tonsils.[62] In cats, malignant lymphomas and squamous cell carcinomas are found in the rostral part of the nasopharynx.

Malignant tonsillar tumors in the dog. Malignant lymphomas (Figure 3.10) and malignant melanomas (Figure 3.11) in the canine tonsil may be unilateral or bilateral. The tonsillar carcinoma in the dog is remarkable because of its misleading presentation as a painful process rather than as an obvious mass in the oropharynx. The major clinical signs are slowly progressive difficulty in swallowing—first manifested by frequent interruptions of eating—frequent swallowing independent of eating, and then weight loss. In many cases there is a large mass

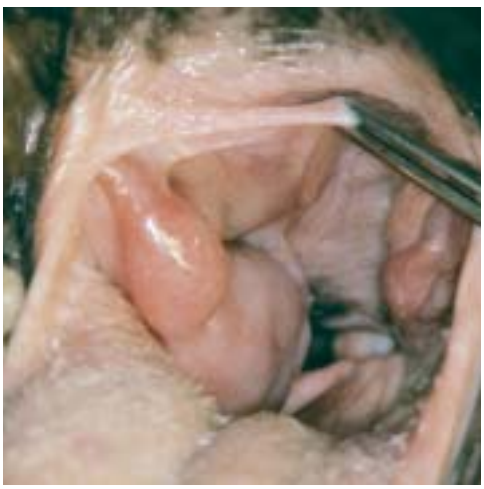


Figure 3.10: Malignant lymphomas in the right tonsillar area of a dog.

lateral to the larynx, the enlarged retropharyngeal lymph node. Pharyngeal inspection reveals a slightly enlarged tonsil. Although it may not be much enlarged, its color is yellowish-pink and the surrounding tissue is retracted and indurated owing to infiltration by the tumor (Figure 3.12). The dog should be sedated to allow careful inspection of the area and to obtain a biopsy of the tonsil for histological examination. Surgical removal is not an option because of the extensive infiltration by the tumor, and neither chemotherapy nor radiation is effective. The pain caused by the tumor gradually increases, and together with the anorexia and emaciation, it provides adequate justification for euthanasia.[67]

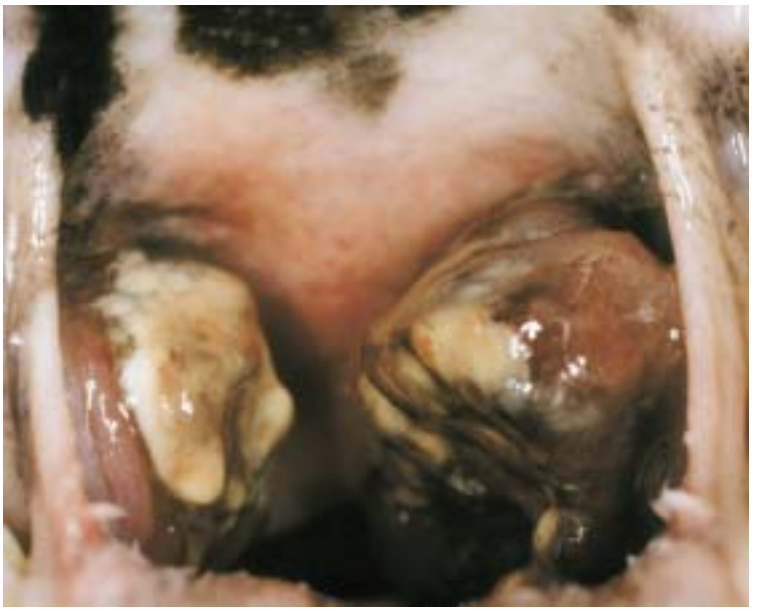


Figure 3.11:
Bilateral malignant melanomas in the tonsillar area of a dog.

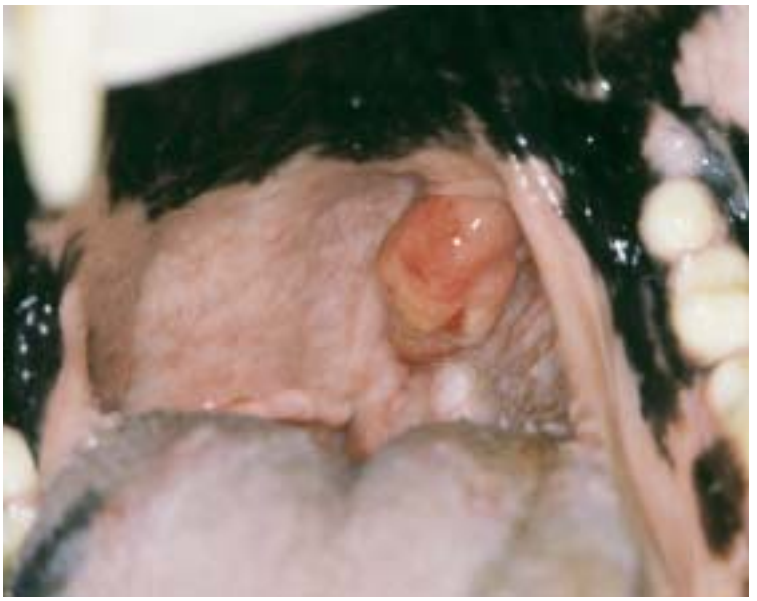


Figure 3.12:
Tonsillar carcinoma in the left tonsil of a dog. The color of the tonsil is yellowish-pink and the surrounding tissue is retracted and indurated, owing to infiltration by the tumor.

Malignant pharyngeal tumors. In cats malignant lymphoma and squamous cell carcinoma occur in the rostral part of the nasopharynx, and the nasal cavities are usually not involved. The clinical signs of snoring and dyspnea without nasal discharge point to obstruction in the nasopharyngeal airway. Laterolateral radiographs of the head reveal a density dorsal to the hard palate and extending caudally. There are no reliable criteria to differentiate between squamous cell carcinoma and lymphoma on these radiographs. Further diagnosis requires endoscopic examination and biopsy under anesthesia, using a flexible endoscope with 180°-retroflexion. Only in case of lymphoma is there a good prognosis for remission of the tumor with chemotherapy.[61] Palliative debulking of a squamous cell carcinoma may help the cat for less than 4 weeks, after which the dyspnea recurs.

Melanomas occur in the oropharynx in both dogs and cats. They are generally malignant and often metastasize before causing signs that come to the attention of the owner.

Exceptional tumors that have been reported in the nasopharynx of dogs include a neuroendocrine carcinoma,[44] an angioleiomyoma,[4] a mast cell tumor,[43] and a lymphangioma.[59]

Craniopharyngiomas in dogs are discussed in Chapter 3.4.5.

3.7 Blunt and penetrating injuries of the pharynx

The most familiar type of pharyngeal trauma in dogs is that caused by penetration by a stick. In cats fissure of the hard and soft palate caused by a fall from a height is well known. In both cats and dogs automobile accidents and fights (dog-on-cat or dog-on-dog) cause many kinds of trauma. Interruption of the wall of the pharynx causes hemorrhage, subcutaneous emphysema, and severe inflammation due to leakage of the pharyngeal contents into the peripharyngeal area. The peritonsillar area is known for the occurrence of abscesses, the consequences of which can be life threatening because of the risk of involvement of cranial nerves, the carotid artery, and even the meninges and brain stem via the foramina at the base of the skull. Similar complications can also be a consequence of acute tonsillitis or rupture of the pharyngeal wall during tonsillectomy. Broad-spectrum antibiotic therapy and drainage of the abscess through the skin of the neck over the affected pharyngeal area should be considered as an emergency intervention.

3.7.1 Blunt pharyngeal injuries

Road accidents may cause blunt trauma to the pharynx, resulting in edema and hematomas that obstruct the pharyngeal airway. It is important to recognize pharyngeal trauma at an early stage, because even minor trauma can hamper the passage of food, liquid, and air. When the clinical signs include drooling of bloody mucous, increased swallowing, and possibly pain in the pharyngeal area, further examination is necessary. The pharynx is somewhat protected anatomically and for it to be traumatized, the trauma is likely to have been invasive and severe. The patient should therefore be given a thorough examination for signs of shock—prolonged capillary refill time, weak pulse, rapid respiration, depression—as well as the possibility of other wounds or fractures. If there are no immediate signs of dyspnea, an intravenous catheter is emplaced for emergency intravenous therapy if needed, and intravenous fluid administration is started. When the animal's condition becomes stable, sedation is administered for examination of the oral, oropharyngeal, laryngopharyngeal, and nasopharyngeal cavities. If dyspnea occurs, endotracheal intubation is performed. Blunt injury to the pharynx usually causes hematomas and edema which can affect the passage of air, food, and liquid. If obstruction of the airway is found or is considered likely to develop within a few hours following the examination, a tracheostomy should be performed and the tracheal cannula should remain in place until the pharyngeal airway is unobstructed. Analgesics are given if the pharynx is painful on palpation. Food and oral liquids are withheld until swallowing is adequate.

If inspection of the pharynx leaves uncertainty about the extent of the trauma, radiographs and CT may be indicated to determine whether there are fractures of the base of the skull and/or the laryngeal cartilages. The associated soft tissue injuries may include swelling, abrasions, and ecchymoses of the skin, subcutaneous tissues, and neck muscles, and particularly the pharyngeal muscles. Surgical repair is not always indicated, but treatment may involve longer hospitalization for supportive care and enforced rest.

Acquired cleft palate in cats. In cats, acquired cleft palate is commonly associated with falls from windows of apartment buildings.[76] The cleft sometimes involves the soft palate as well as the hard palate and results in an opening between the mouth and the nasal cavity or the oropharynx and the nasopharynx. Only severe trauma to the skull can cause a fracture of the hard palate, and thus further examination of the cat for other injuries and brain damage should precede detailed examination of the

palatal cleft. A cleft less than 2 mm wide will usually heal without surgical intervention if soft food is provided for several weeks.[31, 56] A cleft wider then 2 mm should be compressed by a wire passing around the upper canine teeth or by an intraoral pin and wire construction that compresses the two halves of the maxilla.[56] Only when long-term infection of the cleft has caused bone atrophy and fistula formation is it necessary to carry out secondary surgical repair involving mucoperiosteal flap reconstruction.[39]

3.7.2 Penetrating pharyngeal injuries

Penetrating wounds caused by animal bites or a knife or bullet may involve the pharynx, larynx, and trachea. Particularly the skin wounds caused by dog bites in the neck may be misleading in suggesting only minor injury. Subcutaneous emphysema is the most important sign of severe trauma from a penetrating wound in the pharynx, larynx, or trachea. Deeper damage may include injury to vessels and nerves and disruption of muscles. The patient should be examined for signs of shock, such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilated pupils, hypothermia, muscle weakness, and depression, and for other fractures or wounds.[60] The clinical signs of penetrating wounds of the pharynx may include dyspnea due to mucosal edema and cellulitis. If dyspnea is prominent, management of the airway obstruction has priority. Endotracheal intubation and tracheotomy then take precedence over further inventory clinical examination, but general sedation or anesthesia is necessary for this. An intravenous catheter is emplaced for emergency intravenous therapy, if needed, and intravenous fluid administration is started. ECG monitoring should be functioning before sedation or anesthesia. For emergency endotracheal intubation, tubes of several sizes, smaller in diameter than the usual size for the animal, are prepared together with a laryngoscope and a long forceps, about twice as long as the muzzle of the animal, with fine-toothed or grasping jaws for removal of obstructing soft tissues from the laryngeal inlet. Remember that there is always an entrance through the larynx to the trachea as long as the animal is breathing. Intubation may be particularly difficult if there are pharyngeal wounds, and removing blood by suction may be necessary to identify the laryngeal opening. After the endotracheal tube is in place, attention is turned to further monitoring of the patient and to homeostasis. Radiographic examination follows, under anesthesia and with careful ventilation by hand if required. The neck and thorax are examined first but other sites may be included, according to the findings of the general examination. The radiographs of the neck will be difficult to interpret in detail because of the subcutaneous emphysema. The radiographs of the thorax will show mediastinal emphysema or pneumothorax. These findings are of particular interest to the anesthetist if surgical intervention is required, for prolonged anesthesia will necessitate specific additional techniques.

Surgical exploration of the region of the injury begins with a long ventral midline incision of the skin over the pharynx, larynx, and trachea. The location of skin wounds does not necessarily indicate the location of the penetrating injury, and the goal of the surgery is to explore the area to determine the integrity of the pharynx, larynx, trachea, and esophagus. Wounds in the pharyngeal mucosa are débrided and then closed with simple inverting sutures with monofilament suture material. Lacerated and thrombosed vessels are ligated. Lacerations of the pharynx, esophagus, and trachea are closed after débridement. Lacerated muscles are repaired where possible and drains are put in place.

Internal penetrating wounds can be caused by wooden sticks, fish hooks, sewing needles, or other sharp objects. Stick wounds usually

result from the dog's running into a stick with the mouth wide open, as can happen in the woods, or catching a stick thrown in play. When the dog is presented as an emergency, it is usually in pain and panic, and is bleeding from the mouth. In a survey of 65 dogs examined for penetrating stick wounds of the pharynx, an abscess or cellulitis was found in the soft tissue adjacent to the site of the wound and the dogs were frequently depressed, febrile, and reluctant to eat.[21, 75]

After clinical examination the dog is sedated to allow a thorough inspection of the painful mouth and pharynx and the larynx. Most penetrating wounds occur under the tongue or near the tonsil or in the caudal laryngopharynx or in the pyriform recess. Radiographs usually reveal emphysema and cellulitis but no foreign material, since wood is radiolucent, but injury to the hyoid bone or the retrobulbar and other regions may be observed. Diagnostic examination can be carried out most effectively under anesthesia.

As in the case of blunt injuries in the pharynx, the dog may be depressed and there may be severe blood loss or shock, and thus an intravenous catheter is emplaced for emergency intravenous therapy if needed, and intravenous fluid administration is started prior to sedation or anesthesia. The ECG should be monitored. It may be necessary to remove blood by suction in order to carry out endotracheal intubation. The inspection for a stick wound should include the mouth, the area under the tongue, the oropharynx, soft palate, tonsillar crypts, laryngopharynx, and nasopharynx. Parts of the stick are sometimes found. In the survey of 65 dogs noted above,[75] foreign bodies were recovered in 37. In cases in which no foreign body was found, the pharynx was packed off and the wound and residual tissue cavity were lavaged vigorously to remove any wood fragments. Fresh lacerations of the soft palate and the pharyngeal mucosa were closed with absorbable sutures and drains were placed in the contaminated areas.[75] The severe complications of penetrating wounds in the pharynx include rupture of the esophagus, laceration of arteries, and severe inflammation in contaminated areas.

Dogs playing with sticks and fish hooks. In dogs with a history of playing with sticks and having recurrent abscesses in the neck, the chronic inflammation in the pharyngeal area usually causes clinical signs only when an abscess reaches a substantial volume. After the abscess has been drained and antibiotics administered, most dogs eat well and are lively. Yet even in these cases, long sticks may be found when the neck is explored, although several surgical sessions may be necessary. The area to explore is that alongside the larynx and the first few tracheal rings, for here a stick can penetrate deeply without obstruction by muscle. In some cases ultrasound imaging is useful to locate the foreign material.

A dog that snaps at the bait on a fish hook may be literally caught when someone gives a quick tug on the line, hoping to retrieve the hook before it is too late. Depending on how successful the dog was in trying to swallow the bait, the hook may thereby be lodged in the tongue, the mucosa of the pharynx, the upper esophageal sphincter, or further down the esophagus. When presented to the veterinarian, the dog may be salivating and swallowing repeatedly, but does not usually appear to be in great pain. With the dog under anesthesia and its mouth held open, and with the aid of a laryngoscope and/or other strong light source, very slight tension on the fish line usually points the way to the hook. If there is sufficient space to work under direct vision, it is often possible to use a long forceps to dislodge the hook by grasping the shaft, or the curvature if possible, and pushing in reverse. A simpler and often more effective approach is to thread a rigid tube over the line attached to the hook until, with slight tension on the line to keep it straight, the

tube is stopped by the curve of the hook. While maintaining very slight tension on the line, pushing the tube ahead dislodges the hook. If the line attached to the hook is too short for this maneuver, a length of strong suture material can be spliced to it. However, if the hook is lodged deep in the pharynx or in the cranial esophageal sphincter and the attached line is too short even for splicing, it will be necessary to use a flexible endoscope with a working channel through which endoscopic grasping forceps can be passed to manipulate and remove the hook. The barb on the fish hook unavoidably causes a small tear in the mucosa as the hook is removed, but this will heal and requires no attention.

Penetrating sewing needles in cats. Cats may not have the opportunity to snap at a baited fish hook, but they do like to play with thread. If a sewing needle happens to be attached to it, the cat may be presented as an emergency because the needle has penetrated the pharynx. The thread or even part of the needle may be visible and radiographs taken in both the ventrodorsal and the laterolateral direction will help to locate the needle and reveal the way to remove it. It may be very easy or very time consuming, and anesthesia should be planned for a lengthy procedure. A sewing needle may also be found by chance on a radiograph long after its entry. A broken needle in the pharyngeal area with a somewhat irregular outline may be an interesting radiographic finding, but is almost never the cause of the clinical signs for which the radiograph was taken. Attempting to remove it is not advised, because surgery in the peripharyngeal area may cause more serious lesions than it resolves.

3.8 Dysphagia

Dysphagia is difficulty in swallowing. Swallowing is a complicated function triggered by activation of sensory nerve endings in the pharyngeal mucosa. Under both voluntary regulation in a small area of the cortex and involuntary regulation in the brain stem, sequential activity is initiated in the tongue, masseter muscles, pharyngeal muscles, and esophagus to transport food or liquid to the stomach. Functional considerations concerning swallowing are discussed in Chapter 3.1.

3.8.1 Causes of dysphagia

Neurogenic and muscular disorders. The cause of neurogenic dysphagia is not always clear. It is difficult to demonstrate sensory loss in the pharynx by diagnostic tests, even though the clinical signs of such sensory loss are easily recognized when the sensory nerves are transected. In the same way, it is possible to study the clinical signs and characteristics of dysphagia following transection of the cranial nerves involved in swallowing. In muscular disorders causing dysphagia, such as the muscular dystrophy described in Bouviers,[48] the diagnosis can be based on EMG studies and muscle histology. Neurogenic causes of dysphagia can be recognized because the resulting muscular dysfunction also involves other muscle groups, as in myasthenia gravis.

Peripheral sensory nerves. The cranial laryngeal nerve, or superior laryngeal nerve (SLN) in biomedical terminology, is the most important sensory nerve for triggering the swallowing action.[27] Peripheral neurogenic dysphagia caused by loss of sensory innervation was studied in 10 dogs. In these dogs the effect of unilateral or bilateral transection of the superior laryngeal nerve on electromyographic activity in the hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles was studied during eating and during unilateral electrical stimulation of the solitary nucleus.[70] The difference in effect of stimulation via peripheral pathways (eating, unilateral stimulation of the SLN) ver-

sus central pathways (solitary nucleus (NTS)) on pharyngeal muscle contraction timing during swallowing was studied previously in eight dogs.[69] The duration of pharyngeal swallowing was significantly shorter during eating than during stimulation of the NTS in the anesthetized dog (Figures 3.13 a, b). It was also significantly shorter during eating than during stimulation of the SLN in the conscious dog, and it was significantly shorter during stimulation of the SLN under anesthesia than during stimulation of the NTS under anesthesia. The difference in duration of pharyngeal swallowing during stimulation of the SLN between the conscious and anesthetized states was not significant. It was concluded that stimulation of peripheral or central neural pathways resulted in different timing of pharyngeal muscle contraction during swallowing in dogs. In all recordings the sequence of activity during eating, during stimulation of the SLN, and during stimulation of the NTS was always such that the onset of activity in the hyopharyngeal muscles preceded that in the thyropharyngeal muscles, which in turn preceded that in the cricopharyngeal muscles; this being the normal sequence of activity in these muscles during swallowing.[66, 69, 70]

The effect of unilateral or bilateral transection of the SLN on electromyographic activity in the hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles was studied in 10 dogs during eating and during unilateral electrical stimulation of the NTS. In all groups of dogs after unilateral (left or right) or bilateral transection, the sequence of activity in the pharyngeal muscles was disturbed in some swallowing actions during eating and during stimulation of the NTS (Figures 3.14 a, b). After unilateral transection, 18 % of the sequences were abnormal in the ipsilateral muscles during eating and 7 % were abnormal in the contralateral muscles. After bilateral transection, 8 % of the sequences were abnormal in the muscles on the left during eating and 16 % were abnormal in the muscles on the right. The percentages of abnormal sequences when swallowing was evoked by stimulation of the solitary nucleus were not significantly different. Comparison of swallowing actions having a normal sequence with those in dogs with intact SLNs[69] showed that contraction timing was not significantly different during eating, but during stimulation of the NTS it was significantly shorter than in dogs with intact nerves. It was concluded that transection of the SLN modulates the central pattern generator for pharyngeal swallowing in dogs.[70] Dysphagia was expected after transection of the SLN,[29] but dogs continued to eat normally after unilateral transection. After bilateral transection the first dog ate normally, but the second ate with reluctance, and the third did not attempt to eat at all, and in both of the latter two there were fluid sounds in the trachea. For this reason the remaining two dogs were not allowed to awaken after the bilateral transection; instead, the placement of the wire electrodes in the pharyngeal muscles and stimulation of the NTS were carried out on the same day. The difference in the proportion of irregular sequences in pharyngeal muscle activity between the dogs with a unilateral SLN transection and those with a bilateral transection was not significant, and yet there were no signs of aspiration after unilateral transection of the SLN. The bilateral loss of sensation in the laryngeal mucosa[29] and the consequent loss of normal triggering of swallowing may have allowed leakage of food and saliva into the trachea, with greater consequences than those of irregularities in the sequence of pharyngeal muscle activity. That one dog continued to eat normally after bilateral transection of the SLN suggests the possible existence of a compensatory mechanism.

Peripheral motor nerves. Peripheral neurogenic dysphagia caused by loss of motor innervation was studied in 10 dogs in which the effect of unilateral or bilateral transection of either the glossopharyngeal nerve (IX) or the pharyngeal branch of the vagus nerve (Xph) was investi-

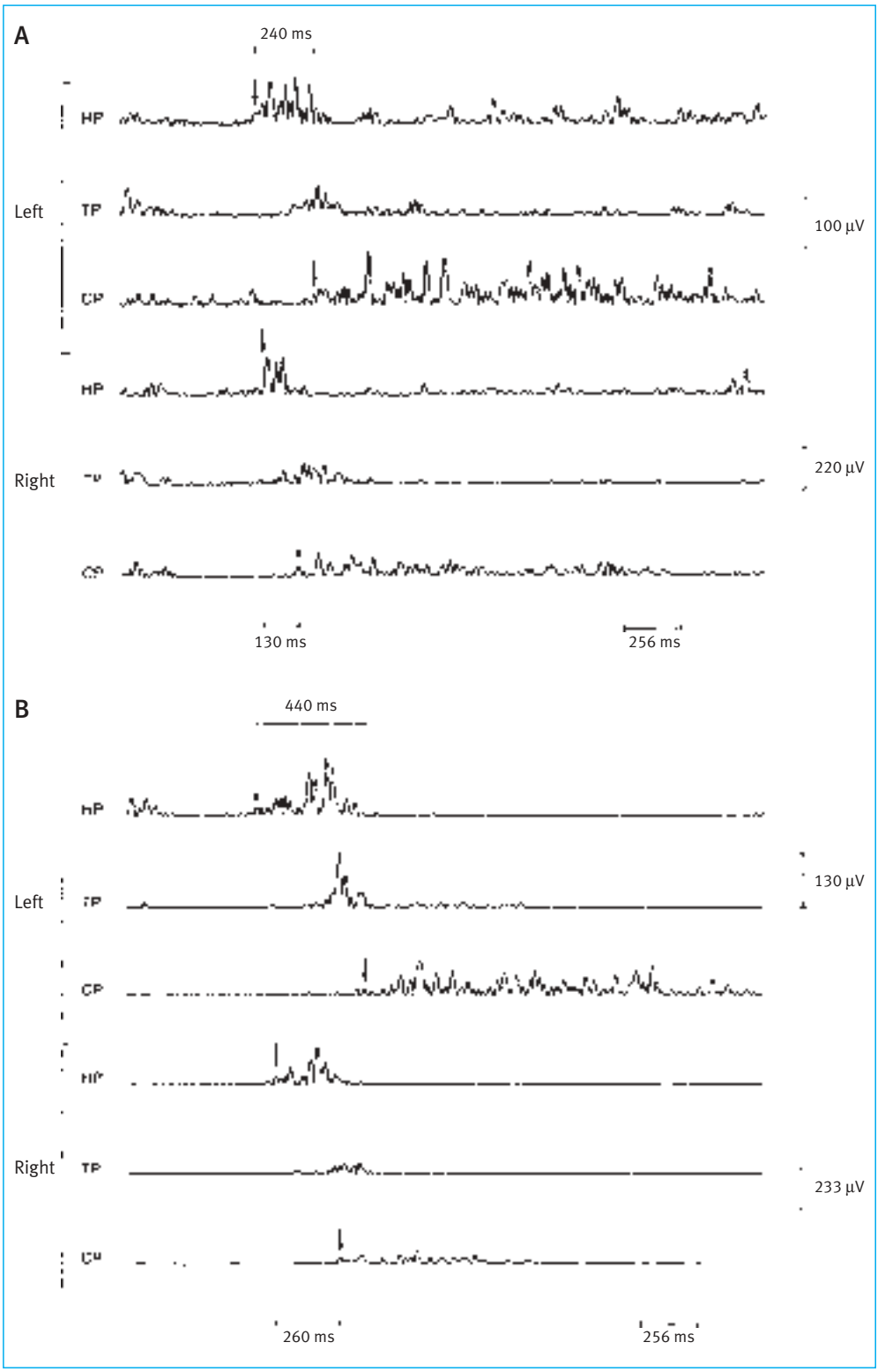


Figure 3.13 a, b: Representative examples of the digitally high-pass filtered and rectified recordings of swallowing activity in the left and right hyopharyngeal (HP), left and right thyropharyngeal (TP), and left and right cricopharyngeal (CP) muscles during eating (A) and during stimulation of the right nucleus tractus solitarius (B) in one dog. Arrows indicate the onset of activity. The bars connecting the dotted lines indicate the intervals in milliseconds. The duration of pharyngeal swallowing was significantly shorter during eating than during stimulation of the NTS; see text for details. (From: Venker-van Haagen AJ, Van den Brom WE, and Hellebrekers LJ. Effect of stimulating peripheral and central neural pathways on pharyngeal muscle contraction timing during swallowing in dogs. Brain Res Bull 1998; 45: 131–136, Fig. 1).

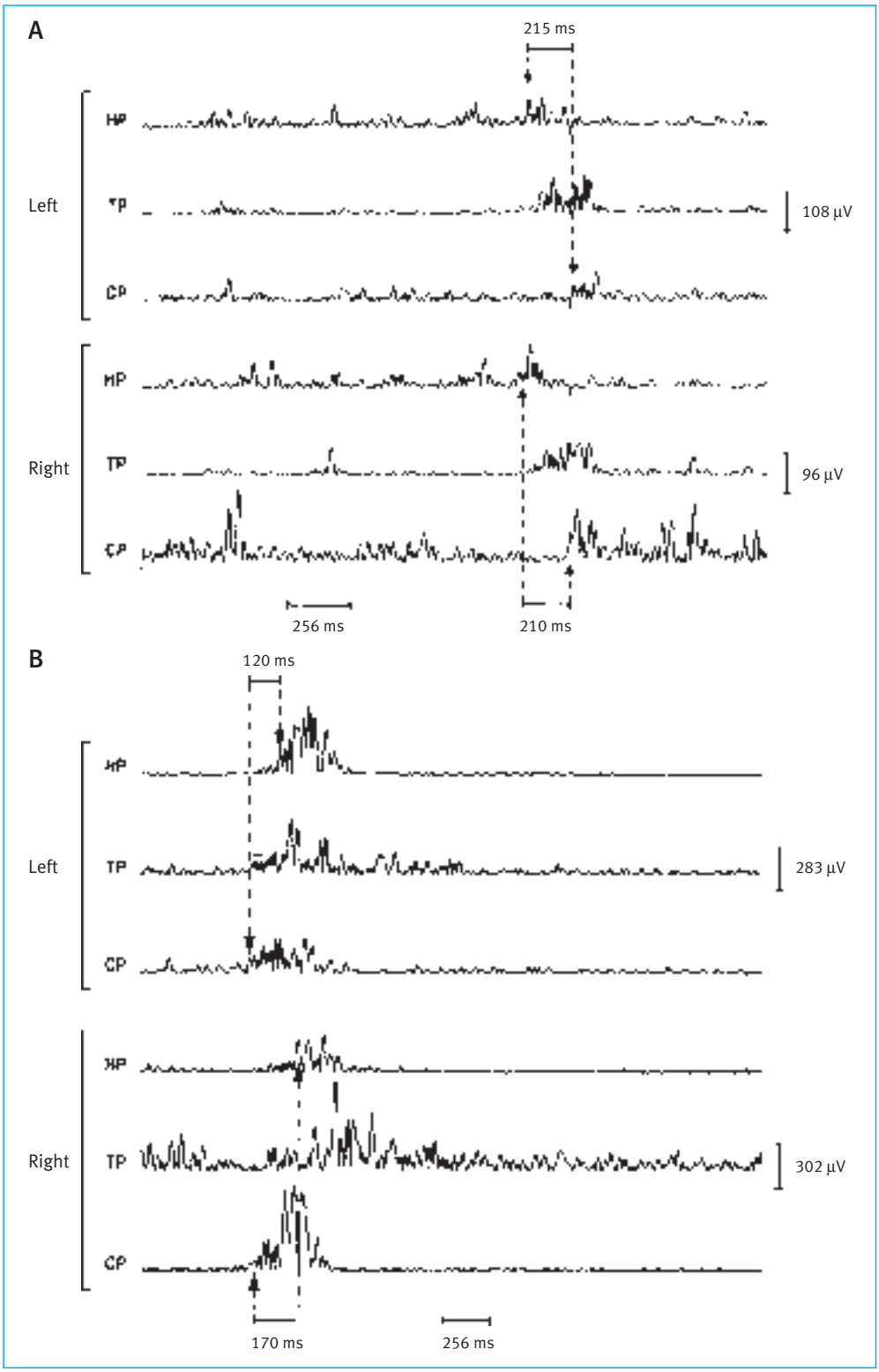


Figure 3.14 a, b: Representative examples of digitally high-pass filtered and rectified recordings of regular intervals (A) and irregular intervals (B). The recordings are from the same dog, following transection of the left superior laryngeal nerve, (A) recorded during eating and (B) during stimulation of the right nucleus tractus solitarius. They show the swallowing activity in the left and right hyopharyngeal (HP), left and right thyropharyngeal (TP), and left and right cricopharyngeal (CP) muscles. The bars connecting the dotted lines indicate the intervals in milliseconds. Transection of the left superior laryngeal nerve modulated the central pattern generator for pharyngeal swallowing in dogs; see text for details. (From: Venker-van Haagen AJ, Van den Brom WE, Hellebrekers LJ. Effect of superior laryngeal nerve transection on pharyngeal muscle contraction timing and sequence of activity during eating and stimulation of the nucleus solitarius in dogs. Brain Res Bull 1999; 49: 393-400, Fig. 1).

gated.[65] These two nerves had been found to be the sole source of motor innervation of the pharyngeal muscles. They have a sensory component, and the neurogenic dysphagia caused by their transection should be regarded as being the result of mixed—motor and sensory—nerve loss. The loss of motor innervation was demonstrated by electromyography; the sensory loss was not demonstrated. In 9 of the 10 dogs there were no outward signs of dysphagia after unilateral or bilateral transection of either the IX or the Xph nerves. Following transection of the second Xph nerve in the remaining dog, the taking of a mouthful of food resulted in dyspnea and regurgitation of most of the food, which was covered with thick mucus. The food was eaten again and, as the process was repeated over and over, the meal was swallowed in small portions. More than one year after surgery, this situation was unchanged.[65] Contrast videofluorography of swallowing in these dogs after unilateral IX transection revealed normal swallowing in 4 and minor changes in 1. After unilateral transection of the Xph nerve there were minor changes in 2 dogs and moderate abnormalities in 3. After bilateral transection of the IX nerves contrast videofluorography revealed normal swallowing in 1 dog and minor abnormalities in 4, while after bilateral transection of the Xph nerve there were minor irregularities in 1 dog and moderate abnormalities in 1, and swallowing was severely disturbed in 3.[65] All 10 dogs were still alive 2 years after the surgery.[66] Although dysphagia ranged from absent to severe in these 10 dogs, there was no acute and life-threatening leakage of food and fluid into the trachea as occurred in all but 1 of the dogs in which the SLN was transected bilaterally.[29] These differences in clinical signs may be helpful in determining the cause in clinical cases of neurogenic dysphagia.

Muscular disorders causing dysphagia were studied in 24 Bouviers and 14 dogs of various breeds.[45, 48] The Bouviers had a familial form of muscular dystrophy,[47] while 2 of the 14 other dogs had myositis and 12 had a myogenic disease of unknown origin.[48]

Difficulty in swallowing was the sole clinical abnormality in all 24 of the Bouviers; there were no other signs of muscle disease. Contrast videofluorography was used to observe the swallowing action and passage through the esophagus. For the evaluation, the swallowing process was divided into three stages: (1) the *oral stage:* prehension and mastication; (2) the *pharyngeal stage:* the combined muscle action of the base of the tongue, the soft palate, and the pharyngeal muscles, and passage through the cricopharyngeal muscle; and (3) the *esophageal stage*: transport through the cervical and thoracic esophagus and the cardia of the stomach.[45] In one dog scarcely any movement of the tongue was observed during the intake of food and water. In 7 dogs the cricopharyngeal muscle did not relax following pharyngeal contractions. In 5 dogs the pharyngeal contractions were very ineffective and uncoordinated, resulting in accumulation of food in the pharyngeal cavity. In 1 dog the cricopharyngeal muscle was permanently relaxed. In 12 dogs there was little or no peristalsis in the cervical part of the esophagus. In 3 dogs peristalsis in the thoracic part of the esophagus was decreased. In addition, aerophagia was observed in 7 dogs, in 1 of which it was the only abnormality observed during videofluorography.[45] In 6 dogs more than 1 stage of the swallowing action was abnormal.

In these 24 Bouviers recordings of spontaneous EMG activity from of the tongue, the soft palate, the hyopharyngeal, thyropharyngeal, and cricopharyngeal muscles (Figure 3.3), and the cervical esophagus revealed a variety of abnormalities, such as fibrillation potentials, positive sharp waves, continuous potentials, and complex repetitive discharges.

Nine Bouviers were available for necropsy and a biopsy of the cricopharyngeal muscle was obtained from another. In the 9 dogs examined

at necropsy, histological examination revealed progressive degenerative myopathy of the altered pharyngeal and esophageal muscles, resembling muscular dystrophy. There were slight abnormalities in the cricopharyngeal muscle examined by biopsy. In none of the 9 dogs examined at necropsy were abnormalities found in the thoracic limbs, pelvic limbs, brain, or peripheral nerves, but in 2 there were changes in the temporal and masseter muscles, and in one there were changes in the intrinsic laryngeal muscles.[45] In a follow-up study in 8 other Bouviers with severe dysphagia, specimens for histological examination were taken from the masticatory muscles (temporal and masseter), pharyngeal muscles (stylopharyngeus, hyopharyngeus, thyropharyngeus, cricopharyngeus, and soft palate), esophagus (cervical and thoracic), diaphragm, and truncal, spinal, and limb muscles. In addition to routine staining techniques, enzyme histochemical methods were used and immunocytochemical methods were used for spectrin and dystrophin. In the tongue, pharyngeal muscles, and esophagus of these dogs there was myopathy characterized by necrosis and phagocytosis of muscle fibers, progressive loss of muscle fibers, variable mononuclear cell inflammation, and replacement by connective tissue and fat (Figure 3.15). The muscular dystrophy was graded according to the degree of fibrosis, from 1 = slight to 4 = replacement of most muscle fibers by connective tissue and fat. In all dogs there were grade 1 to 4 changes in several of the pharyngeal muscles and the esophagus, and in 2 dogs the masticatory muscles were slightly affected, but in none of the dogs were the diaphragm, forelimb, hind limb, or spinal muscles affected. Based on the histological findings and the genealogical characteristics of the disease, it was classified as a form of muscular dystrophy, with predilection for the upper part of the digestive tract. Dystrophin was present in normal amounts in the muscle fibers.[48]

In the 14 dogs of other breeds the diagnosis of myogenic dysphagia was based mainly on the EMG findings, since biopsy or necropsy material was seldom available.

Dysphagia and myasthenia gravis. Dysphagia can be one of the clinical signs in myasthenia gravis, a disease characterized by inefficient neuromuscular transmission secondary to a reduction in acetylcholine receptors on the postsynaptic muscle membrane.[25] Two forms of the disease are recognized in dogs and cats, the acquired form being the most common.[25] In cats, acquired myasthenia gravis can result in generalized weakness but it can also produce focal signs, including dysphagia and megaesophagus, without general weakness.[57] Nine of 25 dogs with acquired myasthenia gravis had no history or clinical signs of generalized muscular weakness but rather the focal form of the disease. Three of the 9 had swallowing difficulties, as did 4 dogs with an acute fulminating myasthenia gravis and generalized weakness.[8]

Megaesophagus and dysphagia. Dysphagia may thus occur in both cats and dogs having the focal form of acquired myasthenia gravis. In cats megaesophagus may be a more prominent sign of myasthenia gravis and in dogs myasthenia gravis is recognized as one of the causes of megaesophagus.[73]

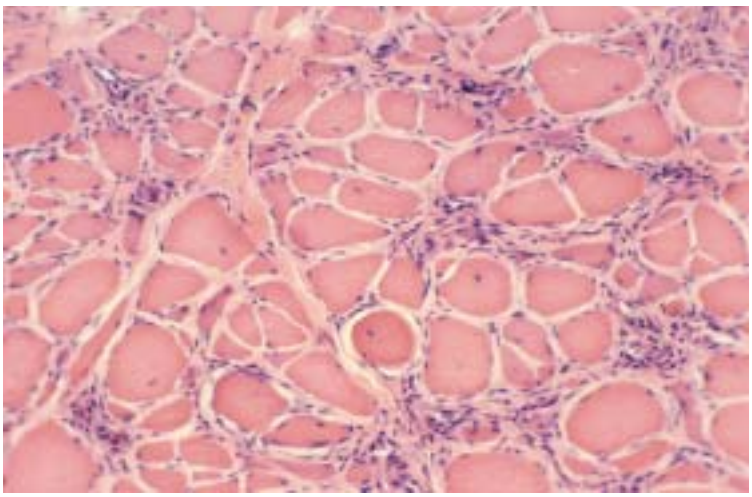


Figure 3.15: The myopathy of the pharyngeal muscles in Bouviers with dysphagia caused by muscular dystrophy was characterized by necrosis and phagocytosis of the muscle fibers, progressive loss of muscle fibers, variable mononuclear cell inflammation, and replacement by connective tissue and fat. (Courtesy of Dr. M.E. Peeters, Division of Surgery, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University).

Central neurogenic lesions causing dysphagia are well known in humans and include cerebral or brain stem ischemic or hemorrhagic strokes, Alzheimer's disease, Parkinson's disease, traumatic brain injury, and motor neuron disease. Both brain stem and cerebral neoplasms occur in dogs and cats, and dysphagia may be one of the signs. A tumor may involve structures such as the nucleus ambiguus, the nucleus solitarius, or the medullary »swallowing center« early in its development, thereby resulting in dysphagia as a presenting sign.[2] With the growing use of CT and MRI in veterinary medicine, it is important to anticipate future developments by studying clinical signs of the most relevant causes of dysphagia in humans.

Obstructive disorders causing dysphagia. Obstruction of the passageway for food can cause dysphagia. The obstruction can be in the oropharynx or laryngopharynx, or in the area around the pharynx. Tumors in the pharynx are described in Chapter 3.6. The clinical signs of dysphagia are probably due to the physical size of the tumor but pain is certainly a factor. The pharynx is the passageway for air as well as for food and liquid, and the clinical signs of obstruction in the pharynx often consist of dyspnea as well as dysphagia. If the dyspnea is severe, the dysphagia may prove to be caused by obstruction of the airway rather than the passageway for food. Peripharyngeal masses are especially known for obstruction of the pharyngeal cavity. The hypopharynx may be deformed by a tumor in the neck and its metastases in the retropharyngeal lymph nodes. Snoring and difficulty in swallowing may be caused by a peripharyngeal process pressing down on the roof of the pharynx.

Radiography can be used as the first approach in the diagnosis of an obstructive disorder causing dysphagia and it can be performed without anesthesia if the dyspnea is not severe. Following radiography, inspection of the pharynx is performed under anesthesia, which should be prepared with extra care such as preoxygenation, since endotracheal intubation may be delayed by the obstruction. Pharyngoscopy should be scheduled as a potentially longer procedure because the diagnosis may not be easy and biopsies may need to be taken. Biopsy of tumors in the pharyngeal cavity often causes profuse bleeding and thus fine needle aspiration biopsy is preferred. Peripharyngeal masses obstruct the pharyngeal passageway but leave the pharyngeal wall intact. Fine needle aspiration biopsy through the wall of the pharynx is possible, but anatomical orientation and fixation of the mass for aspiration is often easier from the outside, through the skin of the neck. Fine needle aspiration biopsy of thyroid tumor metastases in retropharyngeal lymph nodes often obtains blood, in which case multiple biopsies should be taken to ensure that sufficient tissue is also acquired. When biopsy of a tumor in the pharyngeal area is being planned, arrangements should be made for emergency cytological examination of the biopsies, if possible, to ensure that satisfactory biopsies have been obtained. Surgery should be planned after the results of the cytology are known and preferably after CT to determine which structures are involved. Lung metastasis should be known or excluded before surgery. The possibility of complications of surgery in the peripharyngeal area should be anticipated; they may be multiple and can occur postoperatively as well as during surgery. The original problem of dysphagia may not be resolved, because of lesions already present or caused during surgery.[72]

Pain causing dysphagia. Pain serves an essential function in guaranteeing immediate awareness of actual or threatened injury, thereby enabling the individual to adopt a protective behavior.[1] It may seem natural to assume that the sensation of pain arises from excessive stimulation of the same receptors that generate other somatic sensations, but this is not the

case. Although similar in some ways to the sensory processing of routine mechanical stimulation, nociception (noci = hurt) depends on specifically dedicated receptors and pathways. Since alerting the brain to dangers implied by noxious stimuli differs substantially from informing it about more ordinary somatic sensory stimuli, it makes good sense that a special subsystem be devoted to the potentially threatening circumstances.[49] The brain's reaction to a sharp pain in the pharynx may modulate triggering of the swallowing action via the normal sensation of the pharyngeal and laryngeal mucosa. Acute pain may cause contraction of the pharyngeal muscles, which can be interpreted as a defense mechanism, rather than the normal sequence of activity involved in swallowing.[68]

Pain in the pharynx may be caused by sharp injuries, inflammation, or the growth of a tumor. That it makes swallowing difficult is evidenced by multiple rapid contractions of the pharyngeal muscles and profuse salivation. An extreme example of pharyngeal pain is found in the clinical signs of glossopharyngeal neuralgia. This uncommon but dramatic phenomenon is characterized by attacks of severe pharyngeal pain, causing the dog to scream and to salivate. Cramping of the neck muscles may also be observed. The attacks last for several seconds but may recur many times daily. More about neuralgias and their treatment and prognosis is given in Chapter 6.1.

Dysphagia in systemic diseases. Myasthenia gravis has already been mentioned as one of the systemic or neurogenic and muscular disorders in which dysphagia is a clinical sign. Distemper is a systemic viral disease that can cause dysphagia in dogs. Human diseases in which dysphagia can occur include systemic lupus erythematosus, dermatomyositis, and Sjögren's syndrome,[3] as well as benign mucous membrane pemphigoid, polymyositis, and other diseases.[28] When dysphagia and diminished food intake can be differentiated more reliably in dogs and cats, other systemic diseases causing dysphagia are likely to be recognized.

3.8.2 Diagnosis in dysphagia

History and clinical signs. »Difficulty in swallowing« may be mentioned spontaneously by the owner as a problem in a dog or cat. Questioning typically reveals that the animal takes a bite of food from the pan and attempts to swallow it but does not succeed in emptying the pharynx, as is apparent from repeated swallowing actions. If the animal takes in a quick breath of air between swallowing actions, food may enter the larynx and trachea, causing coughing. A cat with such a difficulty in swallowing soon stops eating, apparently because the effort is associated with unpleasant sensations, but a dog will try again and again, and will sometimes succeed in achieving a more adequate swallowing action. Although the dog's attempt to drink from the water pan appears to be normal, the water becomes clouded with mucus and even after a prolonged effort, the level in the pan is scarcely changed. Emaciation is an important clinical sign and other signs include coughing, regurgitating, vomiting, and nasal discharge. The history may be confusing and therefore a standardized questionnaire was developed not only to detect dysphagia but also to determine whether it occurs in the oral, pharyngeal, or esophageal phase.[46]

Questionnaire for diagnosis in dysphagia. The questionnaire was developed and evaluated for dogs. For assessment of its sensitivity, specificity, and predictive value, 69 dogs with »swallowing problems« were investigated. The results of contrast videofluorography were used as the definitive standard. The history was recorded on a standardized questionnaire concerned with the uptake of solid food and liquid

and their transport through the pharynx and esophagus. The questions were short and could be answered by the dog's owner with a »yes« or »no«. Questions about the oral phase of swallowing sought information about normal or abnormal use of the lips and tongue, about abnormal chewing, and about leakage of water from the mouth during drinking. Questions about the pharyngeal phase asked whether swallowing was normal or whether there was gagging, choking, and repeated swallowing of the bolus and/or repeated attempts to swallow water. Questions about the esophageal phase dealt with regurgitation of food or water.[46] The results of the 69 questionnaires and the 69 videofluorographic examinations were compared. The diagnostic accuracy of the questionnaire was investigated by calculating its sensitivity, specificity, and positive and negative predictive values. The questionnaire was found to have a high sensitivity and a high positive predictive value for dysphagia in general and it was useful for exclusion of oral phase dysphagia. Most dogs with pharyngeal phase dysphagia could be detected by using the questionnaire, but it was not of specific value for the detection or exclusion of the esophageal phase of dysphagia. The author's conclusion was that the questionnaire was useful to detect dysphagia in general and sometimes allowed predictions about its localization. Contrast videofluorography remains an indispensable diagnostic tool for the evaluation of dysphagia.[46]

Specific diagnostic techniques. Plain radiographs of the pharyngeal area are useful in the diagnosis in dysphagia when obstruction of the passageway for food is suspected. Masses and foreign bodies can be detected. The hyoid bone should be inspected carefully because a fracture of the bone or arthritic changes in its articulations can cause pain during swallowing and may cause dysphagia.[53] Usually a laterolateral radiograph gives sufficient information.

Contrast videofluorography to evaluate swallowing. For many years contrast videofluorography has been the gold standard for evaluation of patients with swallowing disorders.[41, 46] A standardized procedure should be used to study each patient in the same way so that experience with the technique can be acquired and reliable conclusions can be drawn. The video images should be described in detail, as was illustrated by the descriptions of contrast videofluorographic images in 6 normal beagles and then in these and 4 additional beagles after unilateral and then bilateral transection of the glossopharyngeal nerve or the pharyngeal branch of the vagus nerve .[65] In all 10 dogs, contrast examinations were performed 3 weeks after the unilateral nerve transection and again 3 weeks after the second nerve was transected.[65] For videofluorographic contrast examinations, each dog was placed in standing position in a transparent cage constructed of low x-ray-absorbing polymethylmethacrylate and adaptable to the dog's size. The dogs were fed a meal of either ground meat or pelleted dry food, wetted with barium suspension. Swallowing movements and passage through the esophagus were observed by image-intensified fluoroscopy, using a horizontally directed x-ray tube linked to an image intensifier, with automatic maintenance of alignment. The fluoroscopic images were recorded on videotape. The dogs were conditioned to the cage in advance and food was withheld for 2 days before each examination.[65]

In the preoperative contrast studies, the pattern in all 6 normal beagles was similar. After prehension of the food by the stripping action of the tongue, a bolus was formed at the base of the tongue and then passed into the oropharynx. This action was immediately followed by contractions of the pharyngeal muscles, lifting the bolus up and passing it through the relaxed cranial esophageal (cricopharyngeal) sphincter into the cervical part of the esophagus. At the same time, the exits from the pharynx, includ-

ing the nasopharynx, oral cavity, and laryngeal opening, were closed to prevent regurgitation and leakage into the trachea. The cranial esophageal sphincter closed immediately after the bolus passed through and then esophageal peristalsis transported the bolus in one slow and regular peristaltic wave from the cervical region to the gastroesophageal junction, where it passed slowly through the relaxed sphincter into the stomach. Usually, the passage of food through the cricopharyngeal sphincter was followed by immediate esophageal peristalsis. However, in some dogs, especially those that bolted their food, not every swallowed bolus was picked up immediately by esophageal peristalsis; sometimes, peristaltic activity started only after a second bolus or even a third had been swallowed. Another frequent phenomenon was the stopping of a bolus of food somewhere in the esophagus until it was passed along with the following bolus. Similarly, sometimes a bolus stopped at the gastroesophageal sphincter and did not enter the stomach until it was carried in with the following bolus. Reflux of food from the stomach into the terminal portion of the esophagus occurred occasionally, but it was always followed by secondary peristalsis that cleared the esophagus. In all dogs, the pharynx and esophagus were always completely cleared of food at the end of the meal.

Contrast videofluorography after nerve transection. After transection of one glossopharyngeal nerve in each of 5 dogs, swallowing was unchanged in 4. In the fifth dog it was changed slightly in comparison with the preoperative findings, in that the pharyngeal response after arrival of the food bolus from the tongue was retarded, as was the peristaltic response of the cervical part of the esophagus to the passage of the bolus through the cricopharyngeal sphincter.

After transection of the second glossopharyngeal nerve, swallowing dynamics were still normal in one dog. In the other 4 dogs there were slight irregularities similar to those just described. However, in all 5 dogs, cricopharyngeal relaxation and passage were undisturbed and there were no signs of regurgitation or leakage into the trachea.

There were minor irregularities in swallowing in 2 of the 5 dogs in which the pharyngeal branch of the vagus nerve was transected on one side. In one of these two, the peristaltic response in the cervical part of the esophagus was delayed and in the other the pharyngeal response was retarded. In the third dog swallowing dynamics were normal but there was leakage of material into the trachea. In the fourth dog there was leakage of material into the trachea, the pharyngeal contractions were weak, and relaxation of the cricopharyngeal sphincter was preceded by repeated swallowing movements. In the fifth dog unusually large boluses were formed before being passed into the esophagus and there was no reflex peristaltic response in the cervical portion of the esophagus.

After transection of the pharyngeal branch of the vagus nerve on the opposite side, there were minor irregularities in the swallowing mechanism in one dog, not differing from the irregularities after unilateral transection of the nerve. In the dog in which there was leakage of food into the trachea after unilateral transection of the nerve, the leakage was no longer observed but the pharyngeal contractions were still weak. In three dogs irregular pharyngeal boluses were formed and there were vehement pharyngeal contractions to pass some food through the cricopharyngeal sphincter, but the sphincter remained closed or opened only after a delay. Some food passed fortuitously into the esophagus but peristaltic activity in the cranial part of the esophagus was absent or greatly delayed. When the cricopharyngeal sphincter remained closed, there was regurgitation into the nasopharynx and leakage into the trachea. Peristaltic activity in the thoracic part of the esophagus was normal in all dogs, as was the

passage of food through the gastroesophageal sphincter into the stomach.

The detailed descriptions of the contrast videofluorography images made it possible to classify swallowing dynamics in these ten dogs.[65] The use of the same protocol in dogs with spontaneous dysphagia made it possible not only to classify the severity of the dysphagia, but also to localize the stage—oral, pharyngeal, or esophageal—of the dysfunction.[46] In a long-term follow-up study of muscular dystrophy causing dysphagia in Bouviers, it was concluded that the clinical course in dysphagic Bouviers depended on the localization of the dysphagia as observed by contrast videofluorography. A fairly optimistic prognosis can be given in dogs having only esophageal dysphagia.[48]

Pharyngoscopy in dysphagia. Pharyngoscopy is important in the diagnosis of dysphagia, to recognize and differentiate among lesions of the tongue, oral cavity, oropharynx, laryngopharynx, and nasopharynx. Anatomical malformations in the pharynx, such as hypoplasia of the soft palate, may cause dysphagia or dysphagia-like symptoms. Lesions in the mucosa may cause pain and should be evaluated as the possible cause of dysphagia. The technique of pharyngoscopy is described in Chapter 3.3.1. When no lesions are found to explain the dysphagia, electromyography of the pharyngeal muscles and the tongue should be carried out after the inspection.

An abnormal pattern in the swallowing action observed by contrast videofluorography does not identify the underlying disease nor the affected muscles. Electromyography is required to diagnosis neurogenic or myogenic diseases of the tongue or pharyngeal muscles and, in particular, to determine which muscles are affected. The technique is described in Chapter 3.3.3. Electromyography should in principle be followed by biopsy of the affected muscle(s). Although this is possible for the tongue, the pharyngeal muscles in dogs and cats are too small and too difficult to reach by routine biopsy techniques.

Comparing pharyngoscopy and EMG findings. A noteworthy combination in the diagnosis of dysphagia is the finding of normal electromyographic results together with severe abnormalities on contrast videofluorography. This suggests defective sensory innervation of the pharyngeal mucosa, which is difficult to test. The best approximation is the exclusion of motor neuron defects in the pharyngeal muscles together with normal findings by pharyngoscopy. It is sometimes possible to touch the mucosa in the hypopharynx of the dog without sedation and this mechanical stimulation should provoke contraction of the pharyngeal muscles. The absence of this defense mechanism is highly suggestive for defective sensory innervation in the pharynx.

Manometry in dysphagia. Manometry is the most common technique for the diagnosis of dysphagia in humans,[14] but it requires the cooperation of the patient. Specially designed balloon catheters are passed through the nose to situate one balloon in the esophagus, one in the upper esophageal sphincter, and one in the hypopharynx. The balloons are filled with water and connected to pressure transducers. Pressure measurements are made while the patient is instructed to swallow. Sedation is not used because it affects the results, which is why the technique is not applicable for use in animal patients.

3.8.3 Therapy in dysphagia

The management of dysphagia caused by neurogenic and muscular disorders is often disappointing. It may be helpful to feed semi moist food and, for dogs, to feed from an elevation while the animal remains sitting. In a survey of

long-term survival of Bouviers with muscular dystrophy of the pharyngeal muscles, the dogs with esophageal dysphagia alone survived the longest and their owners reported improvement using this feeding technique.[48] In the rare cases in which EMG examination reveals neuromuscular disease affecting only the cricopharyngeal muscle, cricopharyngeal myotomy may have good results. This cannot be expected to be beneficial in any other case and may even increase the swallowing difficulties and cause aspiration, leading to pneumonia and death.

The treatment of obstructive disorders causing dysphagia depends on the nature of the obstruction. Tumors in the pharynx are usually malignant and difficult to remove surgically. Biopsy will reveal the type of neoplasia, which should guide the oncologist in making a prognosis for the available therapies in each specific case.

Pain causing dysphagia should be treated according to the cause and with the help of analgesics. The pain should be suppressed during the course of the treatment of the underlying disease. It is important for the patient to resume eating and drinking as soon as possible and this guides the choice of analgesic protocol. The depression caused by pain in the pharyngeal area is often underestimated. Swallowing is a phenomenon that occurs about once a minute and the majority of the swallows occur subconsciously between meals. Pain in the pharynx is felt acutely during these swallowing actions, which are involuntary and cannot be suppressed. Pain caused by neuralgias is very difficult to suppress. More details are given in Chapter 6.1.

The treatment of dysphagia in systemic diseases depends on the underlying disease, while the treatment of myasthenia gravis should take into account the management of megaesophagus associated with it,[73] adapted to each affected animal.

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4 The Larynx

4.1 Functional considerations

The larynx acts as a sphincter at the cranial end of the tracheobronchial tree. In order of priority, its functions are to protect the lower airways, to regulate the respiratory airflow, and to vocalize. The cartilages of the larynx—the cricoid, thyroid, and arytenoid cartilages and the epiglottis—interact under the control of the neuromuscular system of the larynx to perform these functions. The cartilages of the larynx simultaneously support and respond to the activity of the extrinsic and intrinsic laryngeal muscles. These muscles move the larynx in swallowing and they open and close the vocal folds in order to protect the larynx and lower airways, and to facilitate respiration and vocalization. Knowledge of the neurophysiology of laryngeal innervation is necessary for understanding laryngeal dysfunction.

4.1.1 The glottic closure reflex

Laryngeal reflexes play important roles in several physiologic functions such as respiration, circulation, phonation, and protection of the lower airway from aspiration. Chemical or mechanical stimulation of the supraglottic mucosa or direct electrical stimulation of the cranial laryngeal nerve (SLN) may result in several responses, one of which is the laryngeal closure reflex. The others are centrally mediated and could be characterized as laryngeal chemoreflexes.[20, 56] The mucosal receptors in the glottic area respond to stimuli such as touch and contact with liquids. The glottic closure reflex, which enables the larynx to protect the lower airways from aspiration of solids or liquids, is a polysynaptic reflex. Mechanoreceptors are located in the superficial mucosa of the glottis and in the muscles and laryngeal joints.[48, 49] There are chemical and thermal sensors in the supraglottic laryngeal mucosa, and many taste buds in the mucosa of the epiglottis and the aryepiglottic folds. The vocal folds also have touch receptors that are more abundant caudally than rostrally. The afferent impulses that are generated are carried by the afferent (sensory) internal branch of the ipsilateral cranial laryngeal nerve, through the distal vagal ganglion (nodose ganglion) to the tractus solitarius and nucleus solitarius (NTS) in the brain stem. The cells of the NTS project to the reticular formation and to the nucleus ambiguus. The nucleus ambiguus is located in the brain stem and contains the motor neurons of the efferent (motor) innervation to the ipsilateral laryngeal intrinsic muscles. The axons of these motor neurons form the ipsilateral recurrent laryngeal nerve. The left recurrent laryngeal nerve passes through the thoracic inlet with the vagus nerve and then returns cranially along the left dorsolateral aspect of the trachea to the larynx, where it innervates the left intrinsic laryngeal muscles. The right recurrent laryngeal nerve also runs caudally with the ipsilateral vagus nerve, but it returns cranially without having entered the thoracic inlet, along the right dorsolateral aspect of the trachea to the larynx, to innervate the right intrinsic laryngeal muscles. The innervation of the paired intrinsic laryngeal muscles is basically ipsilateral, while that of the interarytenoid muscle is bilateral. Bilateral closure of the larynx is effected via bilateral stimulation of motor neuron cells of the nucleus ambiguous by interneurons in the reticular formation. Unilateral stimulation of the SLN caused closure of the glottis together with evoked electromyographic (EMG) responses in both thyroarytenoid muscles.[21] Unilateral stimulation of the SLN also produced evoked responses in the cricopharyngeal and thyropharyngeal muscles or a synchronous bilateral swallowing action, depending on the character of the stimulus (Chapter 3.8).[67, 68]

Reflex closure of the glottis has been shown to be stimulated by water in the pharynx.[52]

4.1.2 Respiratory movements of the glottis

The respiratory movements of the glottis—opening during inspiration and closing during expiration—are regulated in the respiratory center in the brain stem and higher central nervous systems. Chemoreceptors for O_2 and CO_2 in the blood are located in the glomus caroticum and the glomus aorticum, from which afferent fibers of the glossopharyngeal and vagus nerves carry impulses to the respiratory centers in the medulla oblongata and the pons. The motor neurons in the left and right nucleus ambiguus are activated from these centers.

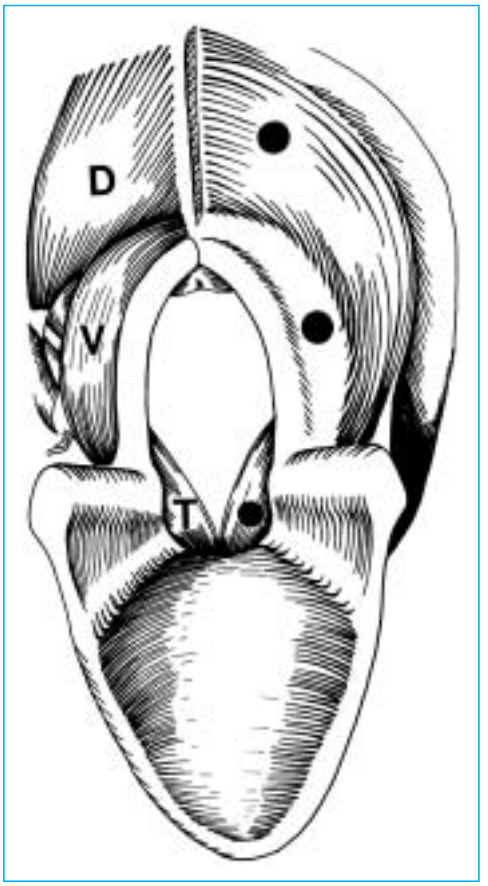


Figure 4.1:
Diagram of the oral view of the canine larynx. The mucosa is omitted on the left side to show the underlying muscles. The black dots indicate placements of the electrodes. D = dorsal arytenoid muscle, V = ventricular muscle, T = thyroarytenoid muscle. (From: Venker-van Haagen AJ, Hartman W, Goedegebuure SA, Wentink GJ. The source of normal motor unit potentials in supposedly denervated laryngeal muscles of dogs. Zentralbl Veterinarmed A 1978; 25: 751–761, Fig. 1).

4.1.3 Movements of the glottis in vocalization

The larynx must respond to commands from linguistic and motor centers for vocalization, signals being relayed to the motor cortex in the precentral gyrus and to motor nuclei in the brain stem and spinal cord. These functions are well described for humans and probably have similar pathways in dogs and cats, but speech in humans is much more complex than vocalization in dogs and cats.[49]

4.1.4 Action of the glottis in coughing

Coughing is a reflex triggered by stimulation of »cough receptors« in the mucosa of the larynx, trachea, carina, and bronchi. Activation of the receptors triggers a fixed-action motor pattern: a rapid deep inspiration, closure of the glottis, expiratory muscle activity that raises the pressure against the closed glottis, and then abrupt opening of the glottis to forcefully expel the air and whatever substance stimulated the cough. This reflex is one of the defense mechanisms for protection of the deeper airways.

4.1.5 Supplementary innervation of the dog's intrinsic laryngeal muscles

The innervation of the intrinsic laryngeal muscles was investigated to facilitate interpretation of EMG findings in dogs with laryngeal paralysis.[60] EMG recordings of spontaneous activity in the intrinsic laryngeal muscles were made before and after unilateral recurrent laryngeal nerve neurectomy and during long-term follow-up. Nerve stimulation studies with stimulation-evoked EMG recordings and acute nerve transection experiments during continuous EMG recordings from the intrinsic laryngeal muscles were also performed. The long-term effects of unilateral recurrent laryngeal nerve transection

on the bilateral intrinsic laryngeal muscles were studied histologically.

In five dogs spontaneous EMG recordings were made from the intrinsic laryngeal muscles using an oral approach to these muscles (Figure 4.1). After unilateral recurrent laryngeal nerve resection, EMG recordings from the bilateral intrinsic laryngeal muscles were repeated monthly in a long-term follow-up study. In four of these 6 dogs the vocal fold ipsilateral to the resected recurrent laryngeal nerve was immobile or vibrated, and in one there was moderate abduction on the side of the resected nerve. After disappearance of the denervation potentials in the ipsilateral intrinsic laryngeal muscles, observed 3 to 9 months after the neurectomy, motor unit potentials synchronous with respiration were recorded in all intrinsic laryngeal muscles. The number of action potentials was lower in all muscles ipsilateral to the resected nerve than in muscles contralateral to the resected nerve. Histological examination of the intrinsic laryngeal muscles revealed denervation atrophy in all muscles ipsilateral to the resection and an amputation neuroma of the proximal nerve end of each resected recurrent laryngeal nerve in all five dogs, indicating that the nerve ends were successfully separated. To search for the pathway of cross innervation, longitudinal sections of the medial part of the dorsal wall of the larynx of these 5 dogs were examined histologically for nerve branches. Several transversely-sectioned nerve branches were found just above the caudodorsal portion of the cricoid cartilage. The finding of action potentials synchronous with respiration in the intrinsic laryngeal muscles ipsilateral to the resected recurrent laryngeal nerve after the disappearance of denervation potentials was inconsistent with the sole innervation of these muscles by the ipsilateral recurrent laryngeal nerve. The site of crossing over of the recurrent laryngeal nerves appeared to be the dorsal side of the cricoid cartilage.

Nerve stimulation studies were then performed in 18 dogs to determine the source of supplementary innervation of the intrinsic laryngeal muscles. In 9 dogs the evoked EMG potentials were recorded from the muscles bilaterally during stimulation of each (left and right) recurrent laryngeal nerve. The results indicated that 11 of the 18 dorsal cricoarytenoid muscles had supplementary innervation from the contralateral recurrent laryngeal nerve. Similar supplementary innervation was found in 5 of the 12 thyroarytenoid muscles from which recordings could be made. In the other 9 dogs not only the recurrent laryngeal nerves but also the external branch of the left and right cranial laryngeal nerves were stimulated electrically. Stimulation potentials were recorded from the left and right cricothyroid, dorsal cricoarytenoid, thyroarytenoid, and ventricular muscles. In 8 of these 9 dogs four or more of the muscles had supplementary innervation and in 2 of these dogs all muscles had supplementary innervation. The supplementary innervation was derived from the contralateral recurrent laryngeal nerve or the ipsilateral or contralateral external branch of the cranial laryngeal nerve, or from more than one of these. Hence in these 18 dogs supplementary innervation of the intrinsic laryngeal muscles was not at all uncommon. In 3 of the dogs with supplementary innervation of the contralateral cricoarytenoid muscles, splitting of the ventral wall of the larynx during continuous stimulation of the right recurrent laryngeal nerve did not affect the evoked EMG response, but splitting of the dorsal wall caused it to cease. This lent support to the supposed pathway for the cross innervation over the dorsal wall of the larynx.

In 4 of the dogs with supplementary innervation, additional EMG recordings were made during spontaneous activity in the intrinsic laryngeal muscles and during one-by-one cutting of the innervating nerves. The results of these studies indicated that the supplementary innervation was effective in sustaining spontaneous

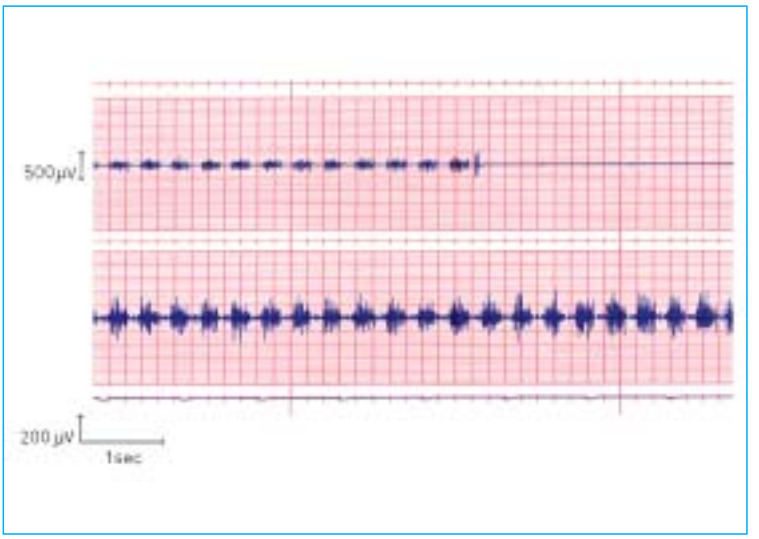
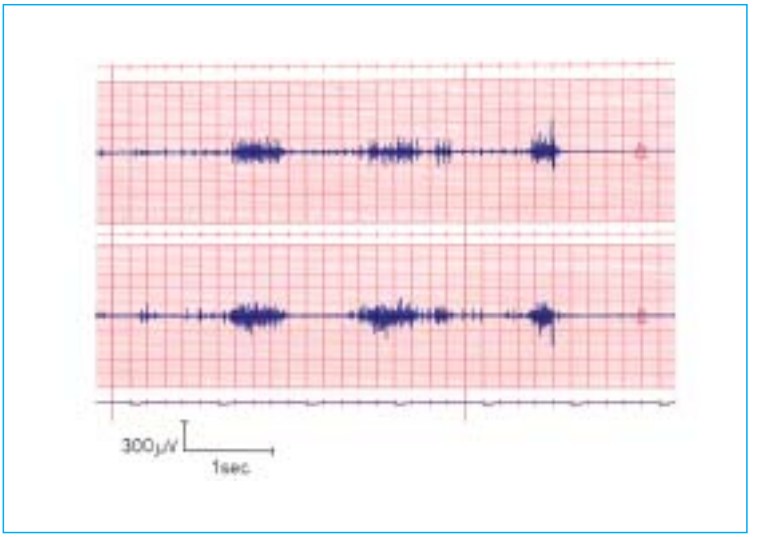


Figure 4.2 a, b:
(a) Simultaneous EMG recordings of spontaneous activity in the left ventricular muscle (upper trace) and left cricothyroid muscle (lower trace) in one dog after the left recurrent laryngeal nerve had been transected. Normal motor unit potentials continued in the left ventricular muscle until the right recurrent laryngeal nerve was also transected (producing the stimulus artifact in the tracing). However, activity still continued in the left cricothyroid muscle and the transection did not produce a stimulus artifact in this tracing.



(b) Simultaneous EMG recordings of spontaneous activity in the left thyroarytenoid muscle (upper tracing) and left cricothyroid muscle (lower tracing) in the same dog as in (A), after both recurrent laryngeal nerves had been transected. Normal motor unit potentials persisted until the left cranial laryngeal nerve was also transected (producing the stimulus artifact), which caused immediate electrical silence in both muscles. (From: Venker-van Haagen AJ, Hartman W, Goedegebuure SA, Wentink GJ. The source of normal motor unit potentials in supposedly denervated laryngeal muscles of dogs. Zentralbl Veterinarmed A 1978; 25: 751–761, Figs. 5 and 6).

laryngeal muscle activity after resection of the principal innervation (Figures 4.2 a, b).[60]

Summary of results from experiments. The combined results of these three experiments can be summarized as follows: (1) The classical description of laryngeal neuroanatomy represents the principal innervation. (2) Supplementary innervation occurs frequently in several intrinsic laryngeal muscles and is provided by the contralateral recurrent laryngeal nerve and the cranial branch of the ipsilateral and the contralateral extrinsic laryngeal nerves; the pattern varies from dog to dog. (3) The supplementary innervation is of substantial importance in EMG recordings from the intrinsic laryngeal muscles, in that the finding of normal action potentials in long-term denervated intrinsic laryngeal muscles does not necessarily prove that the supposedly damaged or transected nerve remained partially intact. (4) The finding of normal motor unit potentials in intrinsic laryngeal muscles does not by itself indicate functional capability.[60]

4.2 History and clinical signs

4.2.1 History

The medical history in laryngeal disease often includes clear statements of specific problems caused by laryngeal dysfunction. The most specific of these are a dry cough and dyspnea with remarkable respiratory sounds. Additional questions are then asked about the animal's general condition, appetite, drinking, physical activity, and endurance, and about changes in its habits. The answers to these questions and a general physical examination may lead to an overall impression of the condition of the patient.

4.2.2 Clinical signs

Coughing. When coughing is the prominent sign, the time and specific circumstances of onset, the frequency, the sound, the productivity, and changes in severity since the cough began will provide information about the nature of the disease. An acute onset may indicate a foreign body in the trachea or bronchi. The onset of coughing shortly after a stay in a kennel or cattery may indicate infectious disease, while the frequency of coughing indicates the severity and persistence of the stimulus. The cough may be sharp and short and be followed by gagging, as in laryngitis, or deep and soft, as in chronic bronchitis. It is important to know the productivity of the cough because neither the larynx nor the trachea produces an abundance of mucus, but mucopus may be expelled during coughing if there is mucopurulent bronchitis. Swallowing after coughing may indicate that material has been expelled through the larynx but not beyond the pharynx. Persistent coughing for several months can be caused by many diseases, but rarely by infectious disease alone. In almost all dogs and cats with persistent coughing, a thorough general and specific examination is indicated to differentiate among heart failure, disease of the respiratory tract other than the larynx, and external stimulation of the cough receptors (smoke, chemicals, dust, etc.).

Dyspnea. When dyspnea is the leading sign in the medical history it is usually described as labored breathing in cats and as diminished endurance in dogs. When the dyspnea is caused by laryngeal dysfunction, a laryngeal stridor is to be expected as an additional sign. Not all owners are aware of the stridor as a particular sign, and therefore the question should be raised. Dyspnea is caused by hypoxemia and hypercapnia, which are detected by the peripheral and central chemoreceptors, respectively. Dyspnea may be caused by insufficient ventilation or insufficient oxygen in the inspired air, or by insufficient circulation, or anemia, or abnormal hemoglobin. Respiration and circulation regulate the oxygen, carbon dioxide, and hydrogen ion environment of the cells. Respiration is controlled by central respiratory centers, central and peripheral chemoreceptors, pulmonary reflexes, and nonrespiratory neural input.

In dogs and cats, labored breathing is recognized by an increased respiratory frequency and strong outward movement of the chest wall. It is accompanied by labial respiration, the mouth being held slightly open with the corners of the lips retracted. Distress and fear of being handled are also common in dyspneic patients. When an obstruction in the upper airways causes dyspnea, there may be a stridor or wheeze. A stridor is the sound produced by the passage of the respiratory airflow through a narrowed pathway. When the narrowing is in the nose, breathing through the nose produces a rustling sound or wheezing. Obstruction of the larynx is recognized as the sound of a handsaw sawing wood, and it occurs during both inspiration and expiration. In the dog these sounds are produced through the open mouth and they are loud. When dyspnea is caused by lung dysfunction or severe anemia, respiration is labored and there is distress, but there is no stridor.

Even when the history indicates stationary or only slowly progressive signs of dyspnea, the signs can be increased rapidly by the stress of the visit to the veterinarian. During the general physical examination of the dyspneic patient, physical and emotional stress should therefore be avoided. While handling the patient gently, continuously observe the character of the respiration, the sound of the stridor, and the color of the tongue. The changing of a laryngeal stridor to a high-pitched inspiratory sound indicates the need for immediate intravenous sedation (medetomidine and propofol) and endotracheal intubation. The color of the mucosa changes rapidly to a dull grayish-blue and without immediate sedation and intubation, lung

edema and death may ensue. Oxygen and intravenous infusion of fluid may help to overcome the critical situation, after which the examination can proceed.

When there is no stridor and the mucosa of the dyspneic patient is not anemic but dark red or cyanotic, oxygen may be administered via the nose during the physical examination and during further examination such as radiography. Remember to avoid stress as much as possible and never attempt to press an oxygen mask on a conscious dyspneic dog or cat, for the ensuing struggle may cause death.

Especially in dyspneic dogs with laryngeal obstruction or hypoplasia of the larynx or laryngeal paralysis, the forced, panting respiration may cause hyperthermia. Body temperature may rise above 40 °C, even within a matter of minutes, at which point, while the mucosa is still red, cooling is more important than oxygen. Spraying or sponging cool water over the entire surface of the dog will lower the body temperature to normal in about 20 minutes. If the panting does not stop, sedation and endotracheal intubation may then be required, but oxygen is not always necessary. In this situation, intravenous administration of fluid is a useful addition, for the dog has probably lost a large amount of fluid by prolonged panting. When the dog is stable, further diagnostic examination can be carried out.

4.3 Special diagnostic techniques

Direct inspection of the larynx with a laryngoscope via the oropharyngeal cavity is the most informative diagnostic procedure. When care is taken in the choice of anesthetic and the depth of anesthesia, the respiratory movements of the arytenoid cartilages and the vocal folds can be assessed. Laryngeal inspection should take place as soon as the dog or cat just loses resistance to opening of the mouth. The larynx of the cat should be examined with the least possible touching of the laryngeal mucosa, for it is very prone to develop edema.

4.3.1 Laryngoscopy

When laryngoscopy is performed for diagnosis of laryngeal disease, the dog or cat is usually in a certain state of dyspnea. The laryngoscope is fitted with a blade suitable for the size of the animal and lubricated endotracheal tubes of several sizes are prepared. The anesthetic is then administered to effect, preferably by intravenous injection. Propofol is satisfactory and may be used after premedication with medetomidine. Medetomidine premedication is given to cats intramuscularly and to dogs intravenously. If laryngeal function is to be investigated the anesthesia should be superficial, for if it is too deep the activity of the intrinsic laryngeal muscles, abduction and adduction, is absent. When the laryngeal movements are absent and the depth of anesthesia may be the cause, the short half-life of propofol is advantageous because after a short pause there is sufficient recovery for the inspection to proceed. For diagnostic laryngoscopy, the animal is placed in a sphinx posture with its head supported by an assistant standing at its side. The mouth is then opened and the neck extended by the assistant, using one hand to hold the upper jaw and the other to hold the lower jaw and flatten the tongue. The laryngoscope is introduced over the tongue and the mouth, oral pharynx, and ventral side of the epiglottis are inspected before the epiglottis is depressed for inspection of the glottis. The size of the laryngeal opening (rima glottidis) is the first concern, for spontaneous breathing requires an adequate laryngeal opening and if the opening is obstructed, endotracheal intubation must be performed immediately. Artificial ventilation via the tube will then be needed, at least for a short while. If intubation is not required, the glottis (the paired arytenoid cartilages dorsally and

the paired vocal folds ventrally) is inspected, the movement of the glottis and the color of the laryngeal mucosa are evaluated, and the cartilages are inspected for deformities. The cavity of the larynx, caudal to the glottis, can be partially observed with the light of the laryngoscope, but the blade of the laryngoscope should not be passed through the glottis until the level of anesthesia is deeper, the laryngeal mucosa has been anesthetized locally with lidocaine spray, and the glottis has become immobile. Laryngoscopy under deeper general anesthesia as well as local anesthesia of the laryngeal mucosa allows fine-needle aspiration biopsy to be performed. In cats the laryngeal mucosa is prone to develop edema and hence following aspiration biopsy both endotracheal intubation and the intravenous administration of a glucocorticoid should be considered. Laryngeal inspection during the recovery period will reveal when the endotracheal tube can be removed safely.

Laryngoscopy is the most important tool for diagnosis of laryngeal disease, since not only the size of the laryngeal opening, the glottis, and the structures around the glottis can be examined, but also laryngeal function can be evaluated under the appropriate level of anesthesia.

4.3.2. Diagnostic imaging of the larynx

Radiographs, Computed tomography (CT), and Magnetic resonance imaging (MRI). Radiographs of laryngeal structures are not easy to interpret. The radiographic anatomy of the hyoid cartilage and the larynx of the dog has been described.[13, 40] In the lateral projection the overlapping of structures and the presence of »air pockets« are unpredictable, particularly in the dyspneic patient. The extension of neoplastic or cystic masses and the presence of calcification of the laryngeal cartilages can be recognized. When surgery is being considered for removal of a laryngeal tumor, CT or MRI will be found indispensable for estimating the involvement of laryngeal and surrounding structures by the tumor. CT is less expensive than MRI and almost always answers the question. MRI is added in only a few cases.[9] In human patients these techniques do not always require anesthesia and endotracheal tubes are avoided. In dogs and cats the use of anesthesia and endotracheal intubation cannot be avoided and this will influence the aspect of the processes in the lumen of the larynx.

Ultrasonography has the advantage that it can be performed without anesthesia, provided that the patient is not dyspneic. It has been used to study the normal larynx in dogs and cats, to examine laryngeal masses and guide fine-needle aspiration biopsy of them, and to identify indications of laryngeal paralysis.[6, 43, 45, 46]

4.3.3 Electromyography of the intrinsic laryngeal muscles

Laryngeal dysfunction is often an indication for electromyography (EMG) of the intrinsic laryngeal muscles. EMG can be performed routinely in the dog but the cat is not a good candidate because it has a small larynx and is too prone to develop laryngeal edema after the larynx is touched. When a dog has signs of laryngeal dysfunction and laryngoscopic examination does not produce a diagnosis, EMG of the intrinsic laryngeal muscles can be helpful, for it can distinguish among normal activity, neurogenic paralysis, ankylotic paralysis, and muscular disease. The dog is anesthetized as for laryngoscopy, to a level at which some muscular activity is still present, and is placed in the sphinx posture. An assistant supports the dog's head, extends its neck, and opens its mouth, as described for laryngoscopy. The epiglottis is depressed with the blade of the laryngoscope,

giving access to several of the intrinsic laryngeal muscles. Recordings are made via a bipolar needle electrode (Danica, 9013L0601), which is fixed in a long, rigid holder that enables the tip of the electrode to be inserted through the mucosa into separate laryngeal muscles under visual guidance. The thyroarytenoid, ventricular, and dorsal cricoarytenoid muscles are accessible beneath the mucosa in this way (Figure 4.1). In the paired abductors (dorsal cricoarytenoid muscles) of normal dogs action potentials are observed predominantly during inspiration. In the paired thyroarytenoid and vocal muscles, visible as the paired vocal folds, action potentials are observed mainly but not exclusively during expiration. In the paired ventricular muscles action potentials are synchronous with expiration. No action potentials are observed if the level of anesthesia is too deep, but abnormal potentials such as fibrillation potentials and complex repetitive discharges (CRDs) will be observed irrespective of the level of anesthesia. Fibrillation potentials are the result of denervation, which can be due to recurrent laryngeal nerve trauma or progressive neurogenic laryngeal paralysis. CRDs are also a common finding in the latter and they are also abundant in muscular disease.

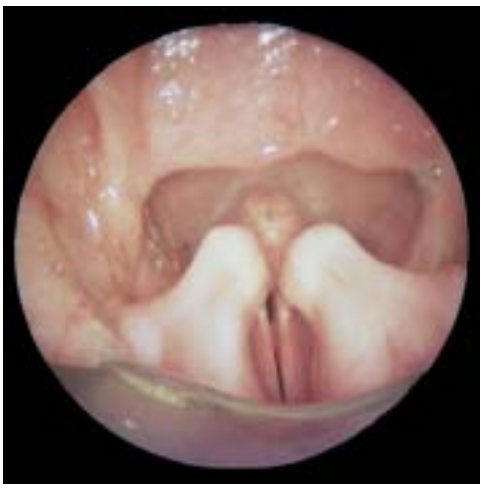


Figure 4.3: Glottic stenosis in a young dog due to joining of the corniculate processes of the left and right arytenoid cartilages.

4.4 Congenital deformities and disorders of the larynx

Congenital deformities of the larynx are uncommon in dogs and cats, and only those causing partial obstruction of the airway will be presented to the veterinarian, because they cause dyspnea or abnormal sounds during breathing. Dyspnea may also be caused by heart or lung failure, but when it is accompanied by stridorous breathing, the most likely cause is obstruction in the upper airway—nose, pharynx, larynx, or trachea.

Laryngeal obstruction is characterized by an inspiratory and expiratory stridor. In congenital deformities, the severity of the obstruction and the clinical signs determine the age at which the pup or kitten is recognized to be breathing abnormally. Sometimes the abnormality is only found when the animal is old enough for its disappointing growth or performance to be recognized. The stridor may not have been noticed or may not have been found alarming. In brachycephalic breeds stridorous breathing is often accepted as a characteristic of the breed and only recurring periods of dyspnea are found to be alarming.

4.4.1 Congenital glottis stenosis

When laryngeal paralysis is found in a very young pup the larynx is often underdeveloped in size. It is, however, difficult to classify this functional disorder as stenosis. The arytenoid cartilages and the vocal folds are not disproportionately small and there are no other deformities causing obstruction, only the paralysis of the vocal folds and arytenoids. The diagnosis is made by laryngoscopy under sedation and by laryngeal electromyography. Congenital laryngeal paralysis is discussed further in Chapter 4.8.

Glottic stenosis is a rare deformity and not recognized to have a breed predisposition. It

occurs in dogs and cats, and results from webbing of the vocal folds or deformities of the arytenoid cartilages. Whether treatment is possible or necessary depends entirely on the development and functioning of the larynx as a whole. Given the wide variety of findings, no general recommendations about treatment can be offered. Glottic stenosis in one young dog consisted of the joining of the corniculate process of the left and right arytenoid cartilages (Figure 4.3). The glottis was obstructed by the deformity and lateral movement of the vocal folds resulted in very little abduction. Electromyography revealed normal periodic activity in the thyroarytenoid muscles and no denervation potentials. Under anesthesia the cartilaginous connection between the left and right arytenoids was found to be thick and lacking an indication of the midline. After step-by-step separation of the corniculate tubercles using a pointed Beaver knife (no. 65), the glottis could be widened only slightly, yet following recovery from anesthesia, respiration was improved. There was partial recurrence of webbing within 6 weeks and it was again necessary to separate the cartilages, but the follow-up revealed progressive improvement in breathing, culminating in a life with almost no restrictions.

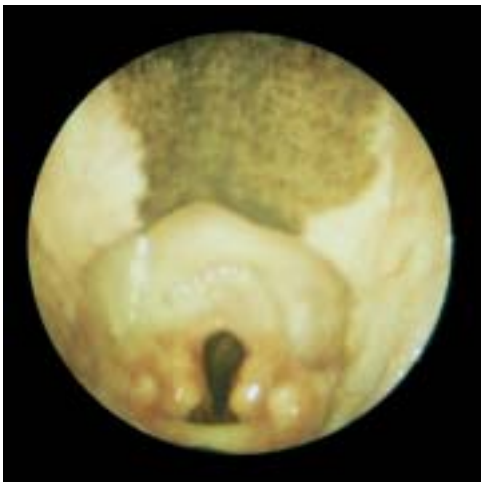


Figure 4.4: Congenital subglottic stenosis in a dog, at the oral margin of the cricoid cartilage. (From: Venker-van Haagen AJ, Engelse EJJ, Van den Ingh ThSGAM. Congenital subglottic stenosis in a dog. J Am Anim Hosp Assoc 1981; 17: 223–225, Fig. 1).

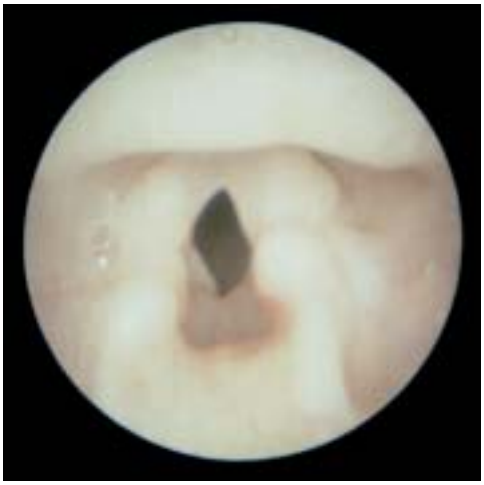


Figure 4.5: Congenital subglottic stenosis in a cat, at the oral margin of the cricoid cartilage.

4.4.2 Congenital subglottic stenosis

Congenital subglottic stenosis occurs in humans, dogs (Figure 4.4), and cats (Figure 4.5) as a congenital deformity of the cricoid cartilage.[34, 63] There may also be deformities in the glottic part of the larynx, but the name refers to the location of the deformity that obstructs the airway. Depending on the severity of the obstruction it causes, the deformity may come to attention in younger or older animals. Some minor obstructive stenosis may be diagnosed in adult life (Figure 4.6). Congenital subglottic stenosis must be distinguished from acquired subglottic stenosis by means of the history. It is

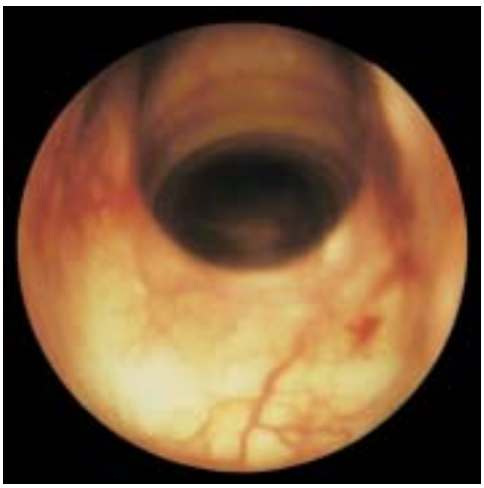


Figure 4.6: Congenital subglottic stenosis in a dog, at the caudal margin of the cricoid cartilage.

very important to examine all other structures and functions of the larynx before attempting surgical correction. The overall size of the larynx may be abnormally small. The arytenoid cartilages may be deformed and the vocal folds may not have developed normally. The aryepiglottic folds should be inspected to determine whether they are of sufficient length to permit ventral movement of the epiglottis. A case report of the disorder in a dog serves to illustrate its clinical and pathological features.[63] The 5-month-old male dog of mixed breeding had dyspnea and no voice since the age of 6 weeks. No information was available about its parents or siblings. The dog became cyanotic upon slight exertion and there was a distinct laryngeal stridor. No other abnormalities were found by clinical examination except for brachygnathia. Laryngoscopy revealed an apparently small larynx with severe subglottic stenosis at the oral margin of the cricoid cartilage. On both arytenoid cartilages the corniculate process was lacking and the cuneiform process was only half the appropriate size. The vocal folds consisted of no more than mucosa. The abnormalities were bilateral and symmetrical.

Since spontaneous improvement was not to be expected and there was no acceptable treatment, the dog was euthanized. Pathological examination confirmed that the arytenoid cartilages lacked both cuneiform and corniculate processes and showed the cricoid cartilage to be dome-shaped, resulting in a very small laryngeal inlet. The intrinsic laryngeal muscles were partly absent and the aryepiglottic folds were short. Histologically the cartilages, muscles, and mucosa of the larynx were normal. It was concluded that the disorder was congenital because of the perfect symmetry of the malformations and the absence of inflammatory or traumatic lesions.

The normal larynx of the cat varies in appearance from one individual to another. A thorough laryngoscopic examination under sedation should furnish a detailed description of the visible laryngeal structures and an impression about their functioning. The corniculate processes normally vary in size and location. Apparent congenital subglottic stenosis in a dyspneic cat should be supported by the history and by complete clinical and specific examinations to exclude other causes of dyspnea, before its relevance can be considered certain. In the cat with congenital subglottic stenosis shown in Figure 4.5, the inadequacy of the laryngeal lumen is also indicated by the eversion of the laryngeal ventricles, a rare finding in cats.

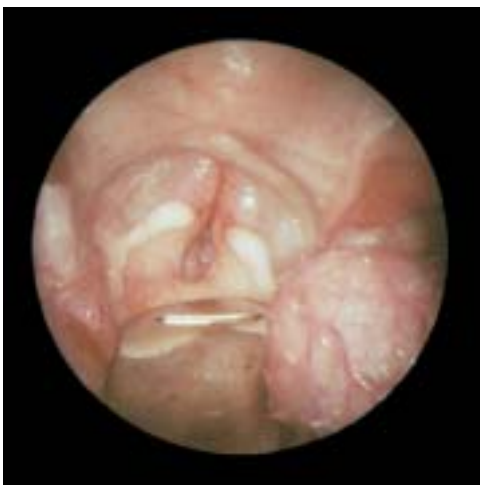


Figure 4.7: Laryngeal hypoplasia in an English bulldog, resulting in a small larynx with a small inlet, midline bending of the cuneiform processes, a flaccid epiglottis, and eversion of the lateral ventricles.

4.4.3 Laryngeal hypoplasia

Laryngeal hypoplasia in brachycephalic dogs. In brachycephalic dogs the laryngeal structures are invariably found to be hypoplastic. The normal larynx has strong cartilaginous structures supporting the laryngeal opening and the laryngeal lumen to sustain its function as the narrowest and most dynamic part of the upper airway. The intrinsic laryngeal muscles abduct and adduct the arytenoid cartilages while being attached to the cricoid cartilage (cricoarytenoid muscles) and thyroid cartilage (thyroarytenoid and vocal muscles). All of these cartilages normally have a certain rigidity in order to perform

this primary function. The hypoplastic larynx is smaller than that of a normal dog of similar stature. The laryngoscopic oral view of the larynx reveals a small and flaccid epiglottis, softly bending over the laryngeal inlet. The left and right aryepiglottic folds and the left and right cuneiform processes tend to fall medially, thereby covering the laryngeal inlet when the epiglottis is softly pressed downward. Pressing the epiglottis further downward causes the cuneiform processes of the arytenoid cartilages to bend to the midline (Figure 4.7). The glottis can be inspected by advancing the blade of the laryngoscope and gently spreading the aryepiglottic folds. The vocal folds are not directly visible because of eversion of the laryngeal ventricles, also called the laryngeal saccules, which have the appearance of small bilateral cysts rostral to the vocal folds. In the hypoplastic larynx, abduction of the glottis opens the laryngeal inlet to a narrow, pear-shaped passage which will only admit small-diameter endotracheal tubes, whereas the normal larynx of a dog of similar stature will admit tubes two sizes larger.

Hypoplasia of the larynx is almost always found in brachycephalic dogs. Laryngeal function should always be included among special clinical examinations in dogs with brachycephalic airway obstructive syndrome before any surgical intervention to diminish the dyspnea is attempted.[77] The brachycephalic airway obstructive syndrome, also known as the brachycephalic obstructive syndrome (B.O.S.), consists of a combination of congenital deformities which obstruct the airway and cause varying degrees of dyspnea, slowly progressive with age. The nostrils may be narrow because the wings (alae nasi) are inadequately supported by the flaccid dorsolateral nasal cartilages. In addition, the pharyngeal cavities are narrow and short, the mucosa is abundant and thickened, the base of the tongue is massive, and the soft palate is relatively long and sometimes passes through the glottis during inspiration. The progressive dyspnea is caused by increasing body weight, the relatively insufficient growth of the laryngeal structures, the increasing mass of the pharyngeal mucosa, and insufficient opening of the glottis. There is progressive collapse of the laryngeal structures and eversion of the laryngeal ventricles because of the increased negative pressure in the laryngeal glottis and the vestibule of the laryngeal cavity.[77] The negative pressure is caused by the increased velocity of exhaled air passing the relatively small laryngeal opening and the increased centrifugal traction on the softer tissues of the vestibule and glottis, causing the lateral ventricle to evert through the supposedly wider and more flaccid opening to the laryngeal ventricle (Figure 4.8).

Collapse of the larynx, as in laryngomalacia in infants, is less common in dogs.[28, 34] In collapse of the larynx and laryngomalacia in infants, the larynx may collapse during inspiration and this results in insufficient intake of food, recurrent dyspnea, and aspiration. In infants the mild form of the disease may be self-limiting by 18 to 24 months of age, but in the severe form complications may arise when surgery of the larynx and tracheostomy become necessary, making the outcome less predictable.[34] In dogs laryngeal collapse is usually fatal, and especially in

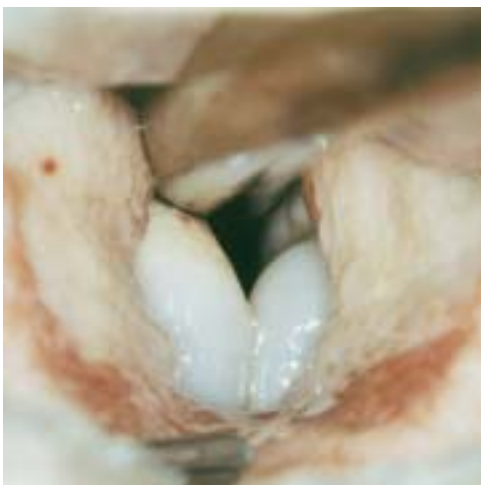


Figure 4.8: Eversion of the lateral ventricles in an English bulldog with laryngeal hypoplasia.

brachycephalic dogs no spontaneous functional improvement is to be expected.

Management of the dog with a hypoplastic larynx. In brachycephalic dogs, the relatively small size of the hypoplastic larynx and the flaccidity of the laryngeal cartilages argue against attempts at surgical correction. A permanent tracheostoma can be a practical solution, but in a young dog it is far from ideal.[74] The life-long care of a dog with a permanent tracheostoma constitutes a continuous anxiety for the family owning the dog (Chapter 5.8.4).

4.5 Laryngitis

Acute laryngitis in cats

Laryngitis is a common disease in dogs and cats. It is characterized by reddening of the mucosa due to dilatation of capillaries and infiltration of leukocytes, and the symptoms are swelling of the laryngeal mucosa, redness sometimes with pain, and hoarseness and coughing. The disease may be caused by infection (usually viral), irritation, trauma, or allergy, or it may be idiopathic. Laryngitis may be an isolated disease or one of the symptoms in a systemic infectious disease. In dogs, acute laryngitis occurs as a primary viral disease known as kennel cough, predominantly caused by canine adenovirus 2 and canine parainfluenza virus. In cats, acute laryngitis occurs in infectious upper respiratory disease caused by feline herpesvirus 1 and calicivirus. A chronic idiopathic laryngitis occurs in older dogs, resulting in chronic hoarseness and sometimes increased raspy sounds during panting.

Therapy for laryngitis should be based on the underlying cause, which should therefore be identified. The medical history is important in identifying the cause correctly. Questions should include the duration of the disease, recent contact with a kennel or cattery, exposure to caustic agents such as smoke in the house, and, in dogs, the use of a choke collar or chain during training and excessive barking in a kennel. Preventing infectious or traumatic causes of laryngitis is important because chronic laryngitis may cause irreversible damage to the larynx.

Acute infectious laryngitis. Viral upper respiratory disease in cats may cause acute laryngitis, often together with pharyngitis. The clinical symptoms are then fever, reluctance to swallow, stridorous breathing, and sometimes dyspnea. Inspection of the larynx under sedation reveals its mucosa to be red and edematous; the laryngeal opening diminished by the edema of the glottis. The obstruction of the laryngeal opening only rarely necessitates endotracheal intubation or tracheostomy. The treatment is symptomatic, for no antiviral therapy is available. In addition to broad-spectrum antibiotics, intravenous fluid therapy should be given as long as the cat is not eating and drinking, and secretions should be removed from the eyes and the nasal openings. Feline herpesvirus type 1 is highly contagious in the acute phase and the patient should be strictly isolated. Feline calicivirus infections are typically mild.[76] The accompanying signs are vesicles and erosions of the mucosa of the tongue and the hard palate. Treatment is aimed at maintaining hydration and supporting the nutritional status. Broad-spectrum antibiotics may be effective for secondary bacterial infections. In the acute phase it is sometimes difficult to differentiate herpesvirus type 1 infection from feline calicivirus infection. In the acute phase the therapy is basically the same and in the both infections the patient should be isolated.

The cats should be monitored for eventually increasing signs of dyspnea. Feline herpesvirus type 1 vaccines do not completely prevent infection but are effective in reducing clinical signs. Feline calicivirus vaccine protects the cat, but there are virus subtypes against which the vaccine provides no protection. After both of

these viral infections cats become carriers of the virus.

Acute laryngitis of unknown cause. Acute laryngitis and laryngeal edema may be found in a cat without signs of infectious disease and the cause may remain unknown after further history and examination. Such a cat should be kept under observation because progress of the disease is unclear and lethal laryngeal obstruction may occur. The cat should be prepared for emergency intubation or tracheostomy. A glucocorticoid may be effective in reducing the laryngeal edema. All handling of the cat should be aimed at avoiding stress. When no underlying cause is found the edema will disappear in about three days to one week.

Acute laryngitis in dogs

Acute infectious laryngitis. Acute laryngeal inflammatory disease in the dog is associated with infectious laryngotracheitis, usually called kennel cough. The clinical symptoms are paroxysms of a harsh, dry cough and sometimes decreased appetite, and when drinking evokes coughing there may be mild dehydration. The voice of the affected dog may be hoarse. The disease is usually self-limiting in three weeks. Complications may lead to bronchitis and even bronchopneumonia, with fever and malaise as well as coughing. The history of contact with a kennel or other coughing dogs in the neighborhood together with the clinical signs lead to the diagnosis. Soft palpation of the larynx and trachea evokes a hard, dry cough. If laryngoscopy is performed, the mucosa of the larynx will be found to be red and edematous. The disease is caused by various viruses, including canine adenovirus type 1, canine parainfluenza virus, canine adenovirus type 2, herpesvirus, and canine reovirus types 1, 2, and 3.[19] The viral infection can become complicated by bacterial infections in which *Bordetella bronchiseptica* can be important. Treatment of the mild form, laryngotracheitis without fever, is symptomatic. House or kennel rest with only short walks and avoidance of excitement is important so long as the dog is coughing. Daily monitoring of body temperature is helpful in following the progress of the disease. The coughing will diminish if the dog is kept calm. Leading the dog on a leash will provoke coughing and should be avoided. When drinking provokes coughing, dogs tend to avoid the water pan. However, a drink of water activates the glands that moisten the laryngeal mucosa, diminishing the irritation, and thus water should be given orally (20 cc for a dog of 15 kg) several times daily according the frequency of the cough. Excessive coughing may be treated by sedatives, especially during the night. Phenobarbital is satisfactory in the dose of 2 mg/kg once or twice daily, depending on the effect. Most dogs recover without complications. When kennel cough becomes a serious problem in a kennel, intranasal vaccines are recommended for use in puppies as young as 2 to 4 weeks of age. Intranasal vaccines may contain attenuated canine parainfluenza virus together with attenuated *Bordetella bronchiseptica*. Attenuated canine adenovirus 2 vaccines are also available.[19]

Acute laryngitis of noninfectious origin. The sting of a bee or wasp at or near the laryngeal supraglottic area causes acute laryngeal inflammation. In countries where these insects are common in and around the house, dogs may catch them and the edema immediately following the sting may cause life-threatening obstruction of the airway. Administration of a glucocorticoid will not quickly decrease the edema and a far better approach is immediate sedation (medetomidine and propofol intravenously) and endotracheal intubation, followed by tracheostomy and the insertion of a trachea cannula (Chapter 5.8.3). The edema disappears in about one week, leaving slight laryngeal inflammation, but abduction and adduction of the glottis can then be observed. The tracheal cannula can be removed and after

Figure 4.9: Thickening of the epiglottic mucosa in a cat due to chronic laryngitis.

another week of rest the dog can resume normal activity.

Chronic laryngitis in cats

Chronic laryngitis of unknown origin. The clinical symptoms of chronic laryngitis in cats are a soft laryngeal stridor and occasional swallowing during purring. There may be loss of voice or a change in the voice, but coughing is rare. Laryngoscopic examination reveals thickening of the laryngeal mucosa and sometimes an irregular surface. In most cases there are no distinct signs of acute onset indicating a distinct cause. The medical history reveals that the signs progressed slowly in the beginning and became stationary over a period of a few months. Treatment with a glucocorticoid is not always satisfactory, but the signs are mild and the cat does not change its habits because of the laryngitis. Explaining that no diseases are present other than chronic laryngitis will usually satisfy the owner.

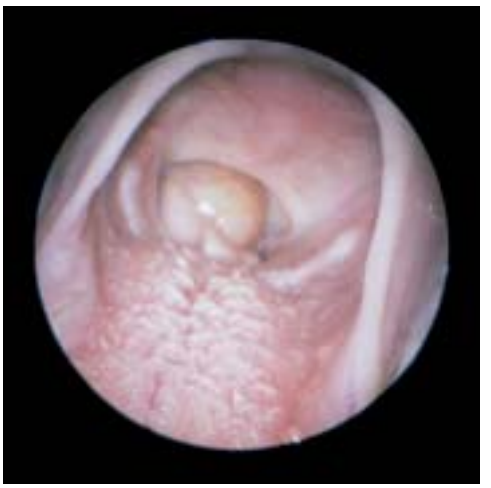


Figure 4.10: Laryngeal abscess in a cat, partly covering the laryngeal inlet.

Chronic granulomatous laryngitis causes stridorous breathing and dyspnea, and laryngoscopy reveals multiple granulomatous proliferations in the laryngeal mucosa which narrow the laryngeal opening. Histologically the granulomas are nodular lesions characterized by a central mass of epithelial cells and giant cells surrounded by lymphocytes and other inflammatory cells. Chronic granulomatous laryngitis in humans is associated with microorganisms that thrive in an immunocompromised host.[24] In cats granulomatous laryngitis may be associated with chronic viral disease under similar conditions, but this has not yet been established. Treatment consists of long-term antibiotic administration to minimize the effect of microorganisms. Regression of the disease is possible but the prognosis is unpredictable.

A single granulomatous mass containing macrophages and lymphocytes has been described.[59] Biopsy of such a lesion is necessary to differentiate inflammation from neoplasia. A solitary mass should be removed, after providing the cat with a tracheostoma to avoid suffocation by laryngeal edema during and after surgery; administering a glucocorticoid alone will not be effective in preventing laryngeal edema. A curious case of chronic laryngitis was found in a cat with a thickened epiglottic mucosa (Figure 4.9). Histological examination revealed granulation tissue and the process regressed with treatment, whether spontaneously or due to the prescribed broad-spectrum antibiotic.

Laryngeal abscess. A laryngeal abscess is sometimes found in cats with slowly progres-

sive signs of laryngeal stridor and dyspnea, usually being recognized on radiographs of the larynx. Laryngoscopy reveals a round, yellowish mass that partly covers the laryngeal inlet (Figure 4.10). A biopsy incision releases thick, yellowish-white fluid that can be freed from the capsule, and culture may reveal *Pasteurella multocida*. Abscesses in this region are usually initiated by penetration of the mucosa by a sharp fish bone, giving entry to oral microorganisms. After removal of the pus and sufficient widening of the opening in the capsule for good drainage, no further treatment is necessary.

Chronic laryngitis in dogs

Chronic laryngitis in the dog may follow kennel cough and be the cause of recurrent coughing and retching. Further specific examination is required to determine whether there is coexisting tracheitis and bronchitis, for *Bordetella bronchiseptica* infections or eosinophilic tracheobronchitis may be the cause of the chronic cough. Therapy depends upon the final diagnosis.

Chronic laryngitis in older dogs. Many older dogs have a mild laryngitis, which may be found during laryngoscopy for various reasons. The laryngeal mucosa is thickened and congested. The clinical signs, if any, are limited to a rasping sound during panting, a roughening of the bark, and gagging after the infrequent single cough. If the clinical signs increase, the history should determine whether there is excessive straining against the collar when the dog is on a leash and whether there is frequent barking or panting. Each of these habits can cause chronic laryngitis and should be eliminated to prevent chronic changes in laryngeal structures which can cause permanent dysfunction. In all cases of chronic laryngitis in the dog, therapy begins with the use of a harness instead of a collar. When possible, other contributing habits should be changed. Nothing is to be expected from medication.

Calcification of the laryngeal cartilages is occasionally found on laryngeal radiographs in older dogs and is not always associated with clinical signs of laryngitis. But when there are signs of mild to severe laryngitis, and by palpation the larynx is found to be enlarged and stiff, there have been considerable changes influencing laryngeal function. Palpation of the larynx evokes a cough followed by gagging and swallowing, possibly an indication of pain. Laryngoscopy reveals that the epiglottis is enlarged and rigid and the mucosa on its dorsal surface is red and contains yellow nodules (Figure 4.11). The mucosa of the glottis and adjacent structures is also red and sometimes edematous. In cases of severe laryngitis vocalization is impaired and breathing is stridorous, both indicating laryngeal dysfunction. Long-term restriction of exercise and use of NSAIDs may suppress the clinical signs.

Chronic laryngitis in laryngeal paralysis. Chronic laryngitis in older dogs can be caused by laryngeal paralysis. In an early stage, when laryngeal function is still nearly adequate, signs of laryngitis may predominate. However, the laryngoscopic finding of dysfunction of glottic adduction or abduction suggests laryngeal paralysis. EMG of the intrinsic laryngeal muscles will confirm the diagnosis.

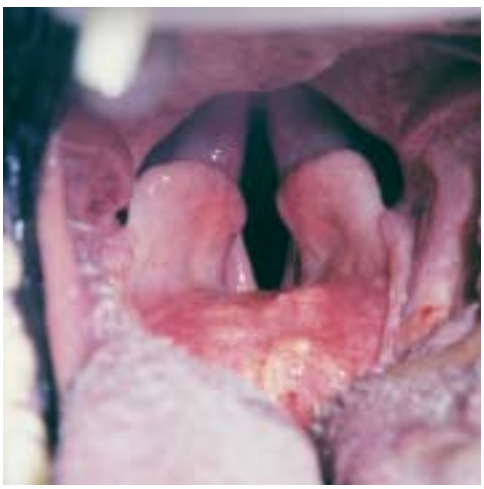


Figure 4.11: Calcification of the epiglottis in an elderly dog. The epiglottis is enlarged and rigid and there are yellow nodules in the reddened mucosa.

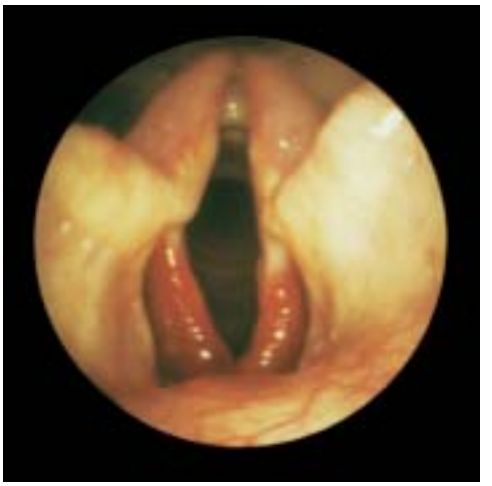


Figure 4.12:
Laryngitis in a dog due to vocal fold abuse, characterized by redness and thickening of the vocal folds.

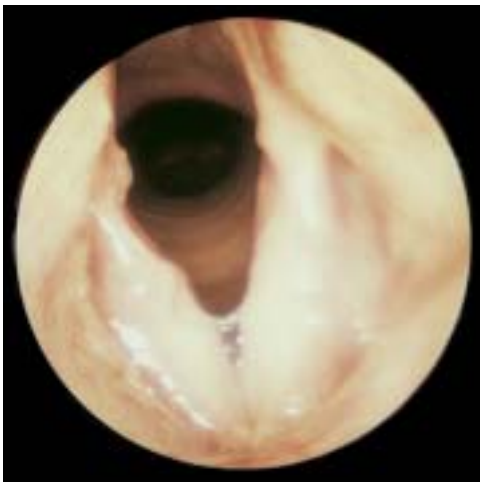


Figure 4.13:
Progressive fibrous replacement of the muscular tissue of the vocal folds in a dog, associated with longstanding vocal fold abuse.

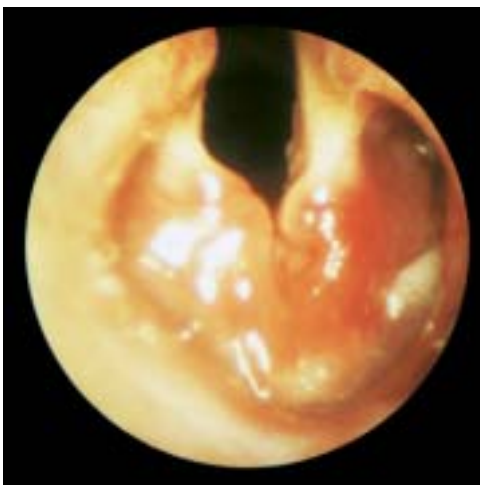


Figure 4.14:
Extremely thick and red vocal folds in a dog. Biopsy revealed only inflammation.

Vocal fold abuse. In dogs certain laryngeal changes indicate vocal fold abuse. These include redness and thickening of the vocal folds (Figure 4.12), sometimes with proliferations or polyps. There may be progressive fibrous replacement of the muscular tissue of the vocal folds (Figure 4.13). There are clinical signs of laryngeal dysfunction, including hoarseness and stridorous breathing, indicating loss of functional abduction of the vocal folds. The history does not always disclose the barking habits of the dog. When the predominant laryngoscopic findings are changes in the vocal folds, the medical history should reexamine the dog's habits and living conditions. Dogs left alone may bark and squeal all day long, a fact not always readily admitted in the history. When the vocal folds are extremely thick and red, only histological examination of biopsy material can exclude diagnoses other than inflammation (Figure 4.14). Therapy consists of extreme rest and providing a situation for the dog in which it does not need to bark. If the dog is at first very restless in the new situation, sedation during the first week may be helpful. If a collar has been worn, it should be replaced by a harness. Laryngoscopy should be repeated after six week of restriction of exercise and in most cases the vocal folds will by then be normal in appearance or nearly so. Exercise can then be increased gradually but situations provoking panting and barking should still be avoided. It should be explained to the owner that if the former situation is allowed to recur, the dog's vocal folds may become fibrous, after which normal functioning of the larynx in respiration and in vocalization can never be restored. A calmer life and the avoidance of excessive activity and excitement will be of the greatest benefit to the dog. For a watchdog this means that a different occupation must be found.

4.5.1 Benign laryngeal masses

Laryngeal cysts have been reported in dogs and cats.[44, 74] If a cyst is located in the laryngeal lumen, dyspnea is the principal sign. The cyst can be recognized by radiographic and ultrasonographic examination of the larynx. It can be localized more precisely by laryngoscopy, during which it can also be incised and drained. Single cysts usually do not recur. Multiple small cysts in the vocal folds are rare in dogs and cats. In humans they are associated with voice abuse and smoking and may recur after removal if the voice abuse or smoking continues.[32]

Other benign laryngeal masses may occur in dogs and cats. According to their size and location, they can cause signs of laryngeal obstruction and a change in the voice. When histological or cytological examination of biopsy material from a mass indicates that it is benign, it should be removed. Depending on size and location, small masses can be removed via the oral cavity, but opening the larynx on the ventral midline provides a safer approach to large masses.

4.5.2 Ventral midline approach to the laryngeal cavities to expose large masses

The procedure described here is essentially for the dog, in which it is safe, while all approaches to the larynx of the cat can be complicated by laryngeal edema. The aim of approaching through the ventral midline of the thyroid cartilage is to obtain satisfactory visibility of the glottis and supraglottic area. This should not be obstructed by an endotracheal tube passing through the larynx and hence the procedure begins with a tracheostomy for insertion of a tube into the trachea below the larynx.

After premedication and induction of anesthesia, an endotracheal tube is introduced through the larynx into the trachea and the cuff is inflated. The ventral side of the neck is prepared for a tracheostomy and laryngeal surgery. The tracheostomy is located midway between the larynx and the thoracic inlet (Chapter 5.8.3). The endotracheal tube will be visible through the tracheal opening and after the cuff is deflated, the tube is slowly withdrawn until it is no longer visible. A sterile endotracheal tube is then inserted through the tracheostomy opening, taking care that it does not pass the carina. Its cuff is inflated and it is connected to the gas anesthesia apparatus, and the first endotracheal tube is removed.

The skin is incised on the ventral midline over the thyroid and cricoid cartilages, the left and right sternohyoid muscles are separated, and then the ventral part of the thyroid and cricoid cartilages and the cricothyroid muscles are freed. With a pointed scalpel (Beaver, no. 65), the thyroid cartilage and the cricothyroid ligament are opened exactly on the midline, leaving the cranial 2 mm of the thyroid cartilage intact. This intact band of cartilage will prevent the two halves of the thyroid cartilage from overlapping when they are subsequently rejoined. An incision exactly on the midline through the thyroid cartilage is important to exactly separate the left and right thyroarytenoid and vocal muscles, which join at the midline of the luminal side. This insures that only cartilage and not muscle is incised, which avoids subsequent webbing of the mucosal lining. Using a small retractor to separate the halves of the thyroid cartilages allows inspection and eventual biopsy or complete excision of the process for histological examination. To prevent webbing, it is very important to avoid creating a lesion in the mucosa on both sides of the larynx. If necessary, the mucosa is sutured to the thyroid cartilage and the cartilage is closed with superficial interrupted absorbable sutures. The suturing is continued caudally to the cricothyroid ligament, taking care that there can be no leakage of air from the laryngeal lumen after subsequent removal of the endotracheal tube. The left and right sternohyoid

muscles are sutured together loosely and the subcutis and skin are closed routinely. When the dog breathes spontaneously the endotracheal tube is replaced by a tracheal cannula, which is left in place for 3 to 5 days.

4.6 Tumors of the larynx

In humans the larynx is the second most common site for cancer in the aerodigestive tract, after the oral cavity. Squamous cell carcinoma accounts for more than 95 % of laryngeal cancers.[55] Primary laryngeal tumors occur occasionally in dogs and cats.[66] In a review of 36 reported primary laryngeal tumors in dogs, 8 were oncocytomas, 6 were rhabdomyomas, and 4 were rhabdomyosarcomas. It was later suggested that two of the reported oncocytomas may have been rhabdomyosarcomas.[29, 30] The other laryngeal tumors found in this review included carcinoma, osteosarcoma, mast cell tumor, melanoma, lipoma, chondroma, chondrosarcoma, leiomyoma, adenocarcinoma, fibrosarcoma, fibropapilloma, and myxochondroma. Among 14 laryngeal tumors reported in cats, 9 were malignant lymphomas, 3 were squamous cell carcinomas, and 2 were adenocarcinomas. The reason for the great difference between these animals and humans in the type and frequency of laryngeal cancer may be that the foremost factor contributing to the development of human laryngeal cancer is tobacco, and the second is the synergistic effect when this is combined with heavy alcohol intake, neither being a habit of dogs or cats.[55]

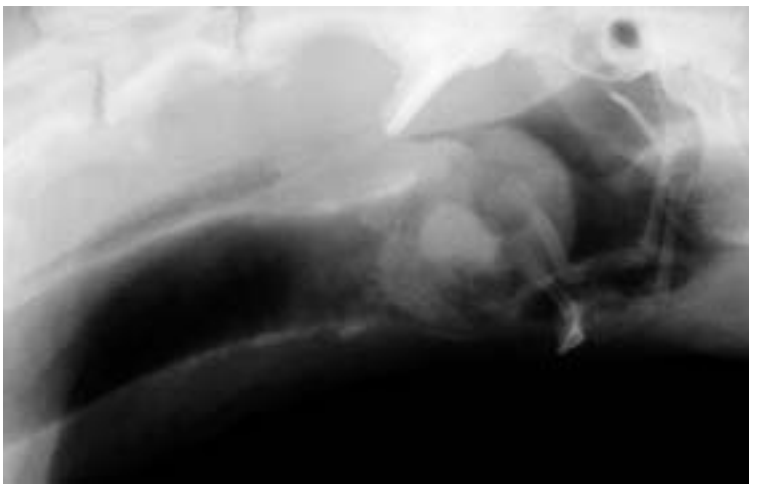


Figure 4.15: Laterolateral radiograph of the larynx of a dog, revealing a tumor and its extension.

4.6.1 History and clinical signs of laryngeal tumors

In dogs and cats the history is important for assessing the risk of further examinations. In most dogs and cats the clinical signs that alarm the owner are dyspnea and hoarseness, which usually indicate that the tumor is already large enough to partially obstruct the airway. Hoarseness does not always mean glottic involvement by the tumor, for it may also be caused by secondary edema. Also, hoarseness and loss of voice occur in most of the laryngeal diseases. What strongly arouses suspicion of tumor is a »breaking voice«, which describes what happens when vocalization—a bark or mew—begins normally and then suddenly changes to a breathy sound or is lost completely, while the animal continues the barking or mewing behavior. This phenomenon is caused by growths protruding into the glottal space, preventing the glottal borders from touching firmly during the cyclic vibration. Also, a cat may start to purr but suddenly stop, possibly because the sensation of purring is unpleasant, for this is followed by swallowing and movement.

Dyspnea and stridorous breathing are common signs of an obstruction in the larynx. Laryngeal stridor is usually both inspiratory and expiratory when the obstruction is great enough to cause dyspnea, and the sound is rasping. In cats the stridor is softer but is clearly recognized as a rasping sound. In dogs the dyspnea is accompanied by listlessness and lack of endurance, whereas in cats the behavioral change it causes is slow and interrupted eating.

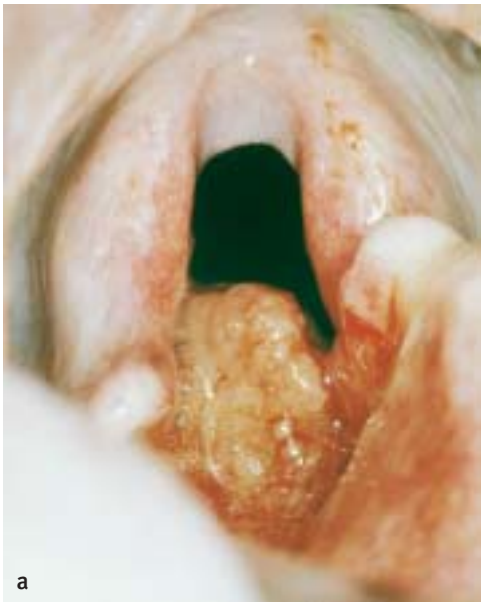
In cats there is usually enlargement of the larynx, which is not too difficult to detect by palpation, but in dogs this is often not the case. The reason for the difference is partly that laryngeal tumors in cats usually involve multiple laryngeal structures, while in dogs they often involve only the glottis and do not cause an overall enlargement of the larynx. In addition, the cat's larynx is a soft and flaccid structure, while many older and larger dogs have a rather rigid larynx. Enlarged lymph nodes are not usually found with laryngeal tumors and hence staging of the tumor should be done on the basis of CT or MRI and thoracic radiographs, when laryngeal surgery is being considered.

4.6.2 Imaging of laryngeal tumors

Further examination of the process in the larynx can be made by ultrasonography, radiography, CT, and MRI. Ultrasonography has been found to provide an accurate indication of the presence and location of laryngeal tumors in cats.[46] Lateral radiographs of the larynx can reveal a tumor and give some idea of its extension (Figure 4.15), but do not clearly distinguish left- and right-sided involvement. Both CT and MRI show fine anatomical detail and provide useful information about invasion of the cartilage, invasion of the base of the tongue, or other extralaryngeal extension, and the status of the lymph nodes.[70] If treatment of the tumor is being considered, CT and MRI are indispensable. However, the cat's larynx is small and even these techniques may not provide detailed information about it or distinguish small lymph nodes. CT and MRI should be considered complimentary to, and not substitutes for, physical examination and laryngoscopy.

4.6.3 Laryngoscopy for laryngeal tumors

Laryngoscopy is the most important technique for the diagnosis of laryngeal tumor, for it reveals the appearance of the tumor, gives some information about its location and its involvement of visible structures, and facilitates biopsy for histological diagnosis. Fine-needle



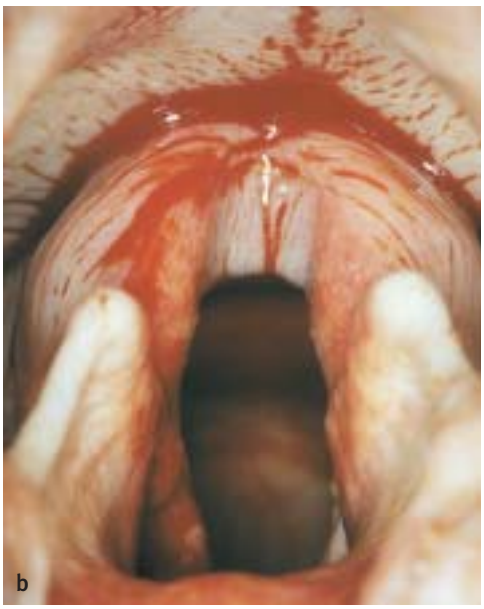


Figure 4.16 a, b: Rhabdomyosarcoma protruding into the glottis of a dog (a). The tumor had a very small base of attachment and was removed (b) via the ventral midline approach to the larynx. (From: Venker-van Haagen AJ. Diseases of the larynx. In: McKiernan BC, Editor. Vet Clin North Am, Sm Anim Pract; Update on respiratory disease. 22: 1155–1172, Figs. 2 and 3. Copyright 1992, WB Saunders Company, with permission from Elsevier).

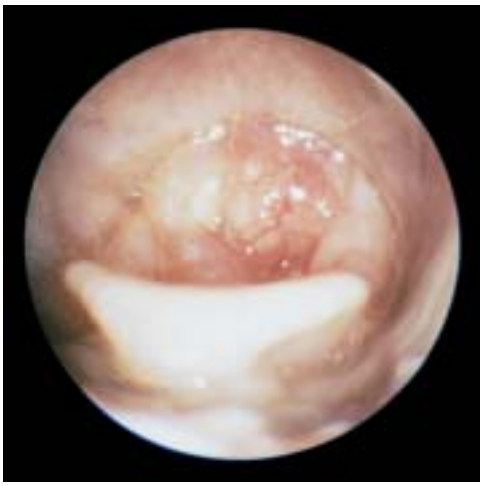


Figure 4.17: Laryngoscopic view of a squamous cell carcinoma invading the larynx of a cat.

aspiration biopsy with emergency cytological diagnosis is quite adequate, especially if the owner does not wish treatment for the animal in the event that a malignancy is found or does indeed wish chemotherapy if the diagnosis proves to be malignant lymphoma. While the cytological examination is being carried out, the animal can be kept under anesthesia and provided with an endotracheal tube. If more time is needed, a temporary tracheostomy is preferable. In dogs and cats an obstruction in the larynx that causes serious dyspnea is not accepted as a mode of living by most owners or veterinarians.

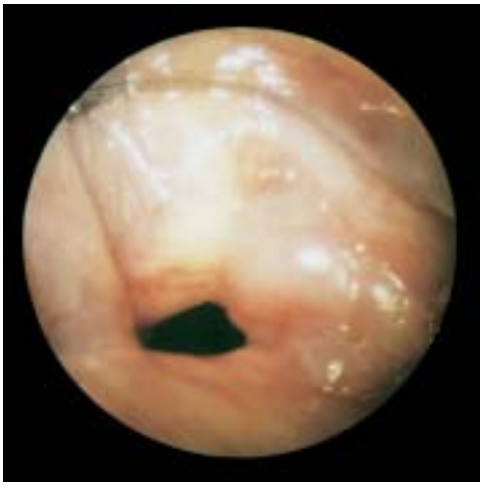


Figure 4.18: Laryngoscopic view of the tumor invading the larynx shown in the radiograph in Figure 4.15.

4.6.4 Therapy for laryngeal tumors

Surgical approach. Some laryngeal tumors have a very small base of attachment and can be removed surgically. If the attachment is in the glottic or subglottic area it is best to use the ventral midline approach to the larynx (Chapter 4.5.2) (Figures 4.16 a, b). If the tumor has so obviously invaded a large part of the larynx, no treatment may be possible other than the hazardous removal of the larynx and formation of a permanent tracheostoma (Figures 4.17 and 4.18). Euthanasia may be well advised. When surgery is not an option, costly CT and MRI examinations should be omitted.

Surgical techniques have been described for hemilaryngectomy and total laryngectomy in dogs and cats.[3, 8] Use of the procedure presumes that the tumor has not spread beyond the larynx and thus lymph node dissection is not included. Formation of a permanent tracheostoma is part of the procedure. The ventral midline approach has been described in detail.[37] The procedure begins with severance of the trachea from the cricoid cartilage and insertion of a sterile endotracheal tube into the trachea for continuing administration of anesthetic gases. The left and right thyropharyngeal and cricopharyngeal muscles are separated on the ventral midline and severed from the thyroid and cricoid cartilages. Then the sternothyroid and thyrohyoid muscles are transected at their laryngeal attachments. The sternohyoid muscle is left undisturbed. The caudal aspect of the larynx is then grasped, lifted, and dissected free from remaining attachments. The bilateral pharyngeal plexus is mainly left undisturbed in this approach.[65] The sensory branches to the laryngeal mucosa, however, are partly severed with the laryngeal mucosa and thus one of the very important triggers for the swallowing action is removed.[69] The mucosa is incised along the rostral edge of the larynx, and the entire larynx, including the epiglottis, is removed.

The pharyngeal mucosa is closed with a con-

tinuous inverting pattern, using absorbable suture material. The transected ends of the paired thyropharyngeal and cricopharyngeal muscles are sutured together ventral to the pharynx and esophagus, and a Penrose drain is put in place to prevent seroma formation.

A stoma is created in the trachea at the level selected for the permanent tracheotomy wound and a new sterile endotracheal tube is inserted through the stoma while the other tube is withdrawn through the mouth. The severed end of the trachea is closed after removal of three tracheal rings, so that the dorsal ligament can be used as a flap. The trachea is then replaced and a standard permanent tracheostoma is constructed (Chapter 5.8.4). The procedure is concluded by emplacement of a gastrostomy feeding tube, which is used for 7 to 14 days, until oral feeding can be resumed.

Radiation therapy is used for human laryngeal carcinoma only following very early diagnosis, when only the vocal fold is affected, and it may be curative. At the stage at which laryngeal tumors become evident in dogs and cats, radiation is clearly no longer an option.

In conclusion, laryngeal tumors occur occasionally in dogs and cats, in a wide variety of histological types. When a tumor is found to be attached to the larynx by a small base, surgical removal may be curative, and malignant lymphoma of the larynx may be susceptible to chemotherapy. However, when a tumor is found to be invasive and to have already metastasized, the only choices are either a hazardous total laryngectomy with a permanent tracheostoma, or euthanasia.

4.7 Blunt and penetrating injuries to the larynx

Because of the relative infrequency of laryngeal trauma, most veterinary clinicians have little experience with this type of injury. There are no typical patterns of injury—each is unique—but the clinician and the surgeon must be familiar with certain signs and techniques in order to manage these patients. A similar situation exists in human medicine, but reviews of laryngeal trauma have led to useful classifications which have improved standardization and evaluation of diagnosis and treatment.[54]

Emergency. It is most important to recognize laryngeal trauma when it is acute and the dog or cat is presented as an emergency. The history will reveal the circumstances leading to the injury and the approximate time at which it occurred. In dogs the most common cause is a dog-to-dog fight, whereas in cats the injury usually occurs out of sight of the owner and thus the circumstances and the exact time at which the trauma occurred are often unknown.

Clinical signs suggesting laryngeal trauma are dyspnea together with hematomas and skin lesions on the neck. A penetrating injury to the larynx is indicated by the surrounding subcutaneous emphysema. The larynx is somewhat protected by the head and the neck muscles and by its ventral location in dogs and cats, particularly when the head is bent downward. Hence for trauma to the larynx to have occurred, the injury must have been invasive and severe. The patient should therefore be given a thorough examination for signs of shock, such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilation of the pupils, hypothermia, muscle weakness, restlessness, and depression or even coma, as well as other fractures or wounds.[58] In most cases of acute injury to the larynx, dyspnea is the predominant sign and management of the airway obstruction must be given priority. Endotracheal intubation or tracheostomy then takes precedence over further inventory clinical examination, but general sedation or anesthesia is necessary for this. An intravenous catheter is emplaced for emergency intra-

venous therapy, if needed, and intravenous administration of fluid is started. ECG monitoring should be functioning before sedation or anesthesia is administered. For emergency endotracheal intubation, tubes of several sizes, smaller in diameter than the usual size for this animal, are prepared together with a laryngoscope and a long forceps, about twice as long as the muzzle of the animal, with fine-toothed or grasping jaws to remove obstructing soft tissues from the laryngeal inlet. Remember that there is always an entrance through the larynx to the trachea as long as the animal is breathing. After the endotracheal tube is in place, attention is turned to further monitoring of the patient and to homeostasis. Preparations are made for the surgical procedure for tracheostomy (Chapter 5.8.3) and the laryngoscopic findings are recorded. Before tracheostomy is begun, another quick inspection of the pharynx is possible, but the endotracheal tube should remain in place until the tracheal cannula is functional. Only then should a plan be made for further diagnostic procedures, in accordance with the respective priorities.

4.7.1 Blunt laryngeal injuries

Road accidents and severe straining on the leash are causes of external blunt injury to the larynx. Acute injury in a road accident can produce endolaryngeal hematomas and edema, causing laryngeal obstruction. The injury may be minor, with small laryngeal hematomas and some lacerations but no fractures of laryngeal structures. The patient is usually alert, coughing, and painful in the area of the larynx, and some bloody mucous might be dripping from the mouth. There is a mild stridor with mild signs of dyspnea that increase during soft palpation of the larynx. General physical examination may reveal further injuries such as broken teeth, bruising or laceration of the tongue, and other trauma usually associated with road accidents. The laryngeal injury may not at first be very alarming. The hematomas and edema may increase rapidly, however, and the most careful approach is to plan laryngoscopy and tracheostomy as soon as the extent of the laryngeal mucosal trauma has been estimated. Prolonged endotracheal intubation is always injurious to the laryngeal mucosa and should certainly be avoided after laryngeal trauma. Imaging techniques, radiographs in the first instance, will detect fractures of the larynx. When there has been substantial dyspnea before endotracheal intubation, radiographs of the thorax will reveal accompanying causes of dyspnea. When there are no fractures of the larynx and no other injuries, the dog or cat is hospitalized for cage rest and care of the tracheal cannula. After about 5 to 7 days laryngoscopy is repeated to evaluate the resolution of the hematomas and edema. When the laryngeal inlet and passage are unobstructed the tracheal cannula can be removed. The larynx will still be very tender and restriction of activity is advised. The wearing of a collar is not allowed for at least 3 weeks and in place of it the dog or cat is fitted with a harness. A cat should be kept in the house for an additional 3 weeks.

Severe dyspnea. When the dyspnea is more severe and the stridor is louder and occurs during both inspiration and expiration, severe laryngeal edema and/or larger laryngeal hematomas can be expected. Since dogs and cats seldom vocalize in this situation, there will be no indication of the function of the vocal folds. When there are large lacerations of the mucosa, the loss of blood or bloody saliva will be evident and further clinical evaluation will reveal associated injuries. Intravenous fluid administration should be started and the patient should be prepared for emergency laryngoscopy and tracheostomy, as described above. If laryngoscopy reveals that the cartilages of the larynx are covered with mucosa and are not exposed, no surgical intervention is indicated. Before a tracheal cannula is introduced via the mouth, a trache-

ostomy is performed and an endotracheal tube is introduced through it to provide anesthesia for diagnostic imaging. If radiographs reveal fractures of laryngeal structures without dislocation, which can be expected if there is no obvious subcutaneous emphysema, a tracheal cannula can replace the endotracheal tube in the tracheostoma. The radiographic examination should be repeated after 2 days. If there are no complications during hospitalization, sufficient resolution of the edema and hematomas can be expected in 10 to 14 days. Laryngoscopic examination should help in deciding when to remove the cannula. As after milder trauma, a harness is used in place of a collar for 3 weeks. A cat is kept at home for 3 weeks following the period of hospitalization necessary for care of the tracheal cannula.

Fractures with discontinuity of the laryngeal cartilages should be repaired immediately or granulation tissue will cause contraction and narrowing of the laryngeal passageway. Depending on their location, most cartilaginous fractures can be stabilized with sutures and fine wire. Apposition of the fragmented parts should be gentle, to avoid tearing the cartilage, but precise, to prevent formation of granulation tissue. Radiographic examination should be repeated after 2 days. If there are no complications during hospitalization, sufficient resolution of edema and hematomas can be expected in 10 to 14 days. After removal of the cannula, a cat is kept at home for 3 weeks and a dog's exercise is restricted for this length of time. For both dogs and cats lifelong avoidance of the use of a collar is advised.

Associated soft tissue injuries may include swelling, abrasions, and ecchymoses of the skin, subcutaneous tissues, and neck muscles, particularly the muscles attached to the larynx or hyoid bone. Any of these injuries necessitating repair should be attended to in the same surgical session as the repair of the fractured cartilages.

Sudden or violent straining against a collar or pulling on a choke chain or collar may cause laryngeal trauma. The injury is usually mild but fractures of the hyoid bone and cricoid cartilage can occur. After other lesions have healed, unilateral recurrent laryngeal nerve paralysis may appear, presumably caused by trauma at the dorsocaudal border of the thyroid cartilage where the nerve enters the larynx, passing between the thyroid and cricoid cartilages. When there is swelling of the laryngeal and pharyngeal mucosa causing dyspnea due to laryngeal obstruction, the approach is similar to that for blunt laryngeal trauma caused by road accidents. Fractures of the laryngeal cartilages should be repaired as described for other blunt trauma to the larynx. When only the hyoid bone is fractured, usually no intervention is necessary, for the fracture is usually single and not often associated with functional disturbance. Trauma to the articulations between the hyoid bones may result in local arthritis, which may cause dysphagia due to pain during swallowing. Resection of the joint will resolve the pain and no further repair will be necessary.

When laryngeal paralysis develops, a wait-and-see approach is usually advisable. Recurrent laryngeal nerve injury will result in some degree of dyskinesia because of mixing of the regenerating abductor and adductor nerve fibers.[25] This occurs with or without surgical repair.[54] In dogs and cats, unilateral laryngeal paralysis is acceptable because it rarely results in dyspnea (Chapter 5.8.1).

Incautious insertion of an endotracheal tube can cause blunt laryngeal injury. Particularly in the cat, laryngeal edema occurs after any touching of the laryngeal mucosa and hence prevention of injury to the cat's larynx is most important. Use of the correct intubation technique and prevention of laryngospasm by the correct method of anesthesia and local application of anesthetic spray are helpful in preventing postanesthetic laryngeal obstruction by laryngeal edema.[16]

Prolonged endotracheal intubation. In a study of prolonged endotracheal intubation in dogs, laryngeal mucosal inflammation was observed after 24 hours, and after one week there was loss of mucosal architecture and inflammatory infiltrates were seen in the arytenoid cartilages.[2] In another study, ulceration was found where the tube rubbed and pressed against contact points in the cricoarytenoid region.[75] Both of these reports have shown that tracheostomy is better than prolonged endotracheal intubation when the laryngeal cavity is obstructed as result of injury.

4.7.2 Penetrating laryngeal injuries

Penetrating wounds caused by an animal bite, knife, or bullet may involve the larynx and other cervical structures. The injuries may be extensive and are generally even more extensive than the skin wounds suggest. Particularly the initial skin wounds from dog bites in the neck may suggest minor injury. Subcutaneous emphysema is the most important sign of severe trauma from a penetrating laryngeal wound. Damage to deeper structures may include penetration of the trachea, laceration of the pharynx and esophagus, injury to vessels and nerves, and disruption of muscles. The patient should be examined for signs of shock, such as tachycardia, hypotension (prolonged capillary refill time, weak pulse), rapid respiration, dilation of the pupils, hypothermia, muscle weakness, restlessness, and depression or even coma, and other fractures or wounds. In most cases of acute laryngeal injury dyspnea is the predominant sign and management of the airway obstruction has priority.[58] Endotracheal intubation and tracheostomy then take precedence over further inventory clinical examination, but general sedation or anesthesia is necessary for this. An intravenous catheter is emplaced for emergency intravenous therapy, if needed, and intravenous fluid administration is started. ECG monitoring should be functioning before sedation or anesthesia. For emergency endotracheal intubation, tubes of several sizes, smaller in diameter than the usual size for the animal, are prepared together with a laryngoscope and a long forceps, about twice as long as the muzzle of the animal, with fine-toothed or grasping jaws for removal of obstructing soft tissues from the laryngeal inlet. Remember that there is always an entrance through the larynx to the trachea as long as the animal is breathing. After the endotracheal tube is in place, attention is turned to further monitoring of the patient and to homeostasis. Radiographic examination follows, under anesthesia and with careful ventilation by hand if required. The neck and thorax are examined first but other sites may be included, according to the findings of the general examination. The radiographs of the neck will be difficult to interpret in detail because of the subcutaneous emphysema. The radiographs of the thorax will show mediastinal emphysema or pneumothorax. These findings are of particular interest to the anesthetist if surgical intervention is required, for prolonged anesthesia will necessitate specific additional techniques.

Surgical exploration of the region of the injury begins with a long ventral midline incision of the skin over the larynx and the trachea. The goal of the surgery is to explore the area to determine the integrity of the trachea, the pharynx, and the esophagus. Vessel lacerations and thrombosis are treated by vessel ligation. Lacerations of the pharynx and esophagus are closed after débridement, as are the tracheal wounds. Lacerated muscles are repaired where possible and the laryngeal trauma is explored and drains are put in place.

Mucosal continuity is the single most important factor to protect the cartilage from infection and to reduce formation of granulation tissue and thereby minimize stenosis in the larynx.[10, 37]

Cartilage fractures may be repaired as in severe blunt laryngeal trauma, but when tissue loss and the necessity for débridement complicate the alignment of the fractured parts, defects are patched to prevent cartilage collapse and stenosis by granulation tissue.[37] In these cases a tracheostomy is performed, initially for insertion of a sterile endotracheal tube to replace the tube passing through the larynx, and then following surgery for insertion of a tracheal cannula to ensure an open airway during the ensuing 10 to 14 days or longer while the wound is healing. Broad-spectrum antibiotics are given and the patient is hospitalized in the intensive care unit during the first few days. Circulation, ventilation, and body temperature are monitored carefully and any necessary supportive treatment is provided. Complications are to be expected in these patients and should be prevented if possible or dealt with in an early stage. All other procedures are similar to those described for major blunt laryngeal trauma.

Internal penetrating wounds are caused by wooden sticks or other foreign bodies that enter the larynx via the oral cavity. Stick wounds usually result from dogs running into sticks with the mouth wide open, as can happen in the woods, or catching a stick thrown in play. When the dog arrives as an emergency, blood loss from the mouth, pain, and panic are the usual signs. After clinical examination, the dog is sedated to allow a thorough inspection of the painful mouth, pharynx and larynx. Usually the laryngeal mucosa has been grazed by the stick, causing a tear in the epiglottis or cuneiform process of the arytenoid cartilage. Most penetrating wounds occur under the tongue, near the tonsil, or in the caudal laryngeal pharynx, the pyriform recess (Chapter 3.7).

Tear in epiglottis. A tear in the epiglottis is repaired by suturing the cartilage with fine wire and suturing the mucosa on the dorsal and ventral sides with absorbable material. For all other laryngeal vestibular wounds, preserving or restoring mucosal coverage of the cartilage is the primary aim of the surgery. Most of these injuries cause trauma in the pharynx followed by abscesses in the neck, rather than obstructive problems in the larynx. Tracheostomy is usually not needed.

Laryngeal webbing is caused by scar tissue formation after bilateral mucosal trauma in the glottis. The cause of the web is often surgical intervention in which bilateral coverage by mucosa was insufficient. Granulation tissue then connects the opposite sides within the glottis or the lumen of the larynx and when the granulation tissue changes into a connective tissue scar, the webbing is complete. Removal of the vocal folds bilaterally, as in debarking surgery, has often been the cause of such webs (Figure 4.19). Dyspnea and stridor develop several months after the surgical procedure. The web may appear to be easy to remove, but dur-

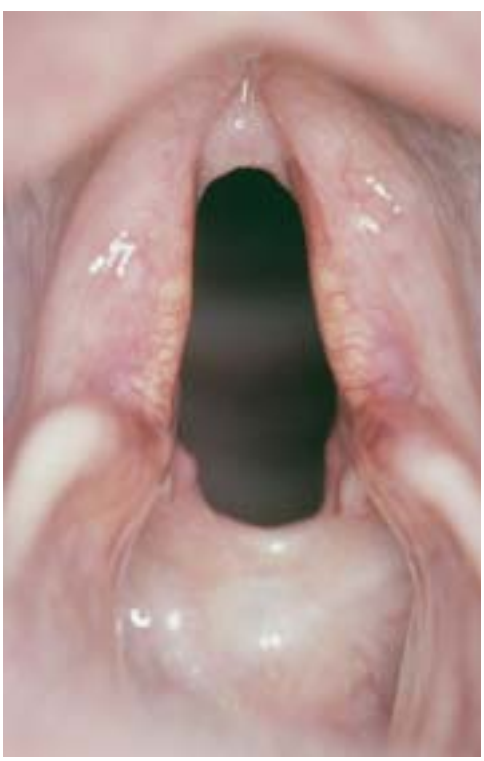


Figure 4.19: Laryngeal webbing in a dog resulting from bilateral vocal fold resection. (From: Venker-van Haagen AJ. Diseases of the larynx. In: McKiernan BC, Editor. Vet Clin North Am, Sm Anim Pract; Update on respiratory disease. 22: 1155–1172, Fig. 15. Copyright 1992, WB Saunders Company, with permission from Elsevier).

ing its formation other structures of the larynx have reacted to the injury, resulting in a less dynamic larynx and insufficient abduction of the glottis. In human patients, 50 % of the webs recur after laser resection.[42]

All surgical intervention involving the larynx necessitates careful handling of the structures and suturing of the mucosal lining to prevent subsequent stenosis and webbing.

4.8 Laryngeal paralysis and functional disorders of the larynx

Paralysis is the loss or impairment of motor function in a part due to lesion of the neural or muscular mechanism; also, by analogy, impairment of sensory function (sensory paralysis).[1] In human medicine the loss of voice is a major subject in laryngology, but loss or impairment of the voice is seldom noticed or mentioned in dogs or cats. This is partly because fine-tuned vocalization is of minor social importance in dogs and cats, and partly because dogs can produce a bark without very highly specialized vibrations of the vocal folds. Dogs use the loudness of the voice more than its tone. Some cats use their voice to produce a variety of tones in communication and thus loss of the voice is recognized by the owner and is part of the clinical history. In dogs and cats respiratory dysfunction is the primary cause of signs and symptoms of laryngeal paralysis. In most cases it is insufficient abduction of the glottis that causes the clinical signs of laryngeal stridor and sometimes dyspnea.

Neurogenic laryngeal paralysis may be caused by interruption of the recurrent laryngeal nerve or by motor neuron disease that involves the motor neurons of the recurrent laryngeal nerves in the nucleus ambiguus. Sensory loss in the laryngeal mucosa is usually caused by interruption of the cranial laryngeal nerves, also called the superior laryngeal nerve (SLN) in biomedical literature. Laryngeal paralysis caused by muscular disease affects both the abductor and adductor muscles and is usually but one component of polymyositis, and dyspnea is then part of the muscular weakness syndrome. In myoneural junction disease, laryngeal dysfunction is due to weakness of both the abductor and the adductor muscles, and is also part of a generalized disorder. Ankylotic laryngeal paralysis is found in older dogs and in the rare cases that have been diagnosed the cricoarytenoid articulations were the only joints affected. Ankylosis of the cricoarytenoid articulations also occurs in humans and arthritis of the cricoarytenoid articulations can occur as an uncommon symptom of rheumatoid arthritis.[36] Although laryngeal paralysis does occur in cats, it is more common in dogs.

Unilateral and bilateral laryngeal paralysis in cats. In cats, laryngeal paralysis is usually unilateral. The unilateral laryngeal dysfunction does not always cause dyspnea but is found by laryngoscopy, together with laryngitis or pharyngitis, and may therefore be associated with herpesvirus type 1 or calicivirus infection. Laryngoscopy should be repeated several times and under various levels of anesthesia before it can be concluded that one vocal fold is definitely immobile. One »lazy« vocal fold may be due to the level of anesthesia and there may be a spontaneous reappearance of normal abduction as the cat awakens. In a review concerning 16 cats with laryngeal paralysis, only 4 had a unilateral paralysis. The clinical signs in these patients were tachypnea or dyspnea.[50] Cases in which the signs were less obvious may not have been included in this retrospective study. In most of the reported cases of unilateral laryngeal paralysis in cats the cause of the disease was unknown. However, in one case the cause was tumor growth in the vagus nerve and in another it was infiltration by tumor originating in the tympanic bulla, both emphasizing the need for an extensive clinical history and physical examination when unilateral

laryngeal paralysis is observed by laryngoscopy.[7, 51]

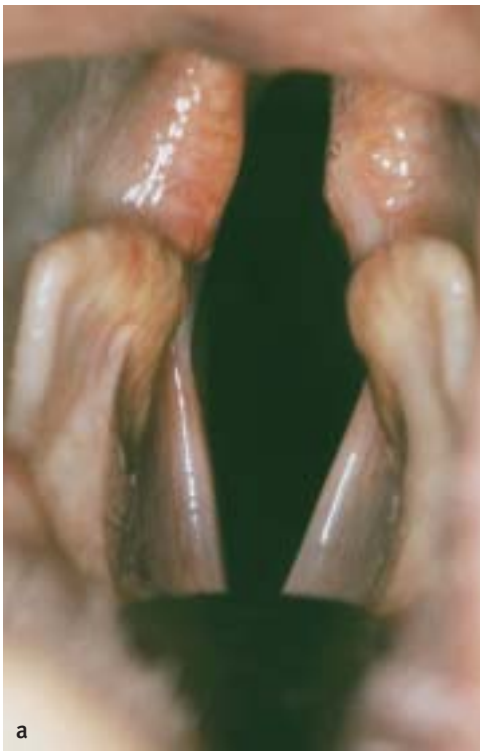
When the cause of unilateral laryngeal paralysis in a cat is not known, it is difficult to decide upon appropriate therapy. If the paralyzed vocal fold causes obstruction of the airway, it can be lateralized (Chapter 4.8.2), but since spontaneous improvement can occur, the examination should be repeated in three weeks before deciding to undertake surgery. Medical treatment is less invasive, but is not based on knowledge of the cause. Treatment with a glucocorticoid may be tried, but only for a very short period, because of the possibility of an underlying viral infection. When obstruction of the airway is not the main symptom, it is best to avoid treatment and to repeat the laryngoscopy after three weeks.

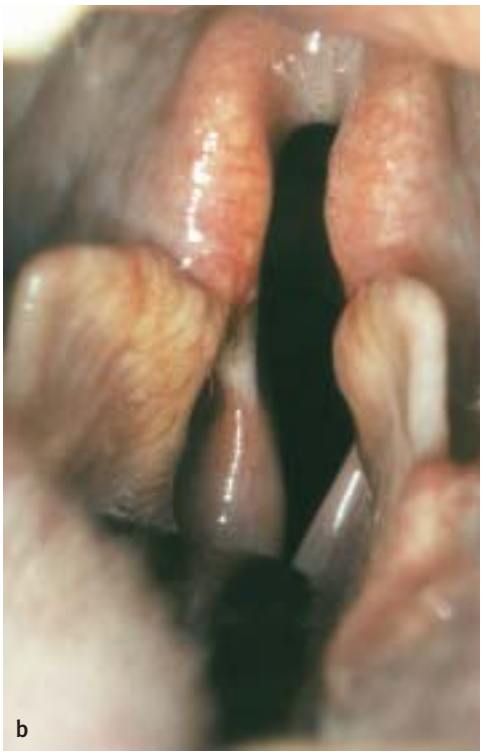
Bilateral laryngeal paralysis is characterized by severe dyspnea and a laryngeal stridor. The cat should be assured of an open airway, for which tracheostomy is often necessary. Spontaneous improvement may also occur in bilateral vocal fold paralysis when no specific cause of the disease is found. A short period of treatment with a glucocorticoid may be tried. If a tracheal cannula is needed, a broad-spectrum antibiotic should be given to prevent bronchial infection. Laryngoscopy should be repeated after one week and if there is even slight spontaneous improvement, such as restoration of activity in one vocal fold, surgical lateralization should be postponed for another week. When there is no improvement, lateralization of one vocal fold is usually sufficient to relieve the severe dyspnea.

4.8.1 Neurogenic laryngeal paralysis

Unilateral laryngeal paralysis in dogs. When one recurrent laryngeal nerve is transected in dogs, there is no loss of voice or hoarseness and there is no observable effect on endurance during normal exercise. The nonparalyzed vocal fold is observed to cross the midline during adduction and it closes the laryngeal opening when it touches the paralyzed vocal fold. Similarly, sufficient abduction of the nonparalyzed vocal fold compensates for the inactive vocal fold and provides an adequate laryngeal opening (Figures 4.20 a–c). Thus, unilateral laryngeal paralysis caused by recurrent laryngeal nerve interruption is usually subclinical. This is in contrast to laryngeal paralysis caused by degenerative neurogenic diseases. There may be asymmetry in movement of the vocal folds but the innervation of the intrinsic laryngeal muscles is affected bilaterally, as EMG recordings will clearly reveal. Trauma to the recurrent laryngeal nerve can occur in a dog fight, usually together with trauma to the trachea, or as a result of scar tissue contraction following neck wounds. Unilateral laryngeal paralysis can also result from trauma to the recurrent laryngeal nerve caused by use of a choke chain. In its course along the trachea, the recurrent laryngeal nerve is well-covered and protected, but at the site of its entrance into the laryngeal cavity near the cricothyroid articulation, compressing the thyroid cartilage against the cricoid cartilage may damage the nerve, which lies between them. The most common cause of unilateral vocal cord paralysis in humans is head and neck surgery (44.2 % of cases), followed by idiopathic cases (21.4 %), central and peripheral neural lesions, and inflammatory or infectious diseases. Congenital unilateral vocal cord paralysis is rare in humans.[38]

In dogs the paralyzed vocal fold is usually not positioned on the midline but is slightly abducted (Figure 4.20 a), similar to its position at rest. There may be a soft laryngeal stridor if the dog is in professional training and undergoing uncommon exertion. Laryngitis may then complicate the signs. In such a case the owner should be advised to stop the training or to select only the easier parts of the training. Lateralization of the paralyzed vocal fold will also handicap the dog, because lateralization is





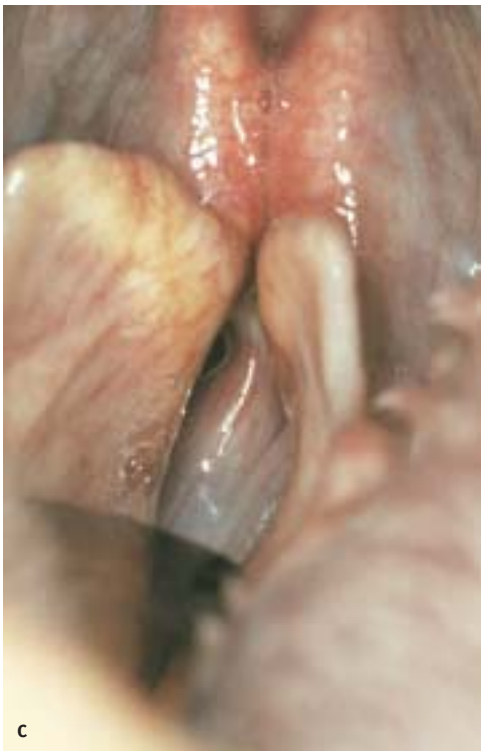


Figure 4.20 a–c: Adduction of the vocal folds in a dog with left-sided laryngeal paralysis (the dog's left side is on the right in the picture). (a) At the moment between inspiration and expiration, no asymmetry is apparent. (b) As adduction begins, only the right vocal fold moves toward the center. (c) Movement from the right continues until it closes the glottis, compensating for lack of movement from the left. (From: Venker-van Haagen AJ. Diseases of the larynx. In: McKiernan BC, Editor. Vet Clin North Am, Sm Anim Pract; Update on respiratory disease. 22: 1155–1172, Figs. 8, 9, and 10. Copyright 1992, WB Saunders Company, with permission from Elsevier).

never equivalent to functional abduction and the loss of expected performance together with the loss of voice will diminish the dog's usefulness in professional work.

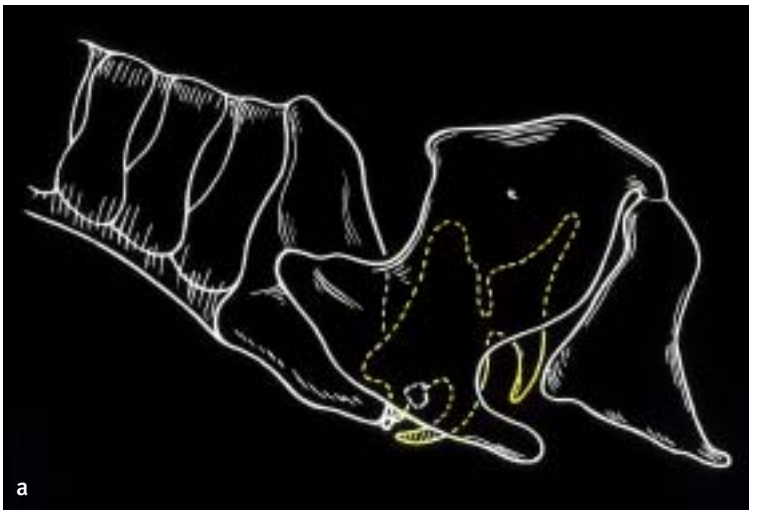
Bilateral laryngeal paralysis in dogs caused by injury. Bilateral recurrent laryngeal nerve injury can occur during severe trauma to the neck or during bilateral surgery near the trachea. The nerves may be ruptured bilaterally but more often the damage occurs during wound healing and scar tissue retraction. When both recurrent laryngeal nerves are ruptured, there is immediate bilateral laryngeal paralysis and thus immediate dyspnea following the injury. When scar tissue retraction is the cause of the injury to the nerves, the history reveals that the dyspnea is recent although the trauma occurred some weeks before. In most cases of complete bilateral laryngeal paralysis the vocal folds obstruct the airway almost completely. Laryngoscopy shows that there is no abduction of either vocal

fold but there may be some adduction, due to the action of the cricothyroid muscles, which are innervated by the external branch of the cranial laryngeal nerves. Their action is to pivot the cricoid cartilage on its thyroid articulations in order to tense the vocal folds, which may be misinterpreted as adduction. No spontaneous improvement is to be expected in these cases. The dyspnea is usually absent during sleep but the least excitement causes a high-pitched inspiratory stridor and gasping for breath. Emergency endotracheal intubation followed by tracheostomy is life saving. When there has been prolonged dyspnea it is better to restore homeostasis before undertaking surgery.

Spontaneous improvement in laryngeal function is not to be expected. Even when the recurrent nerves have been crushed rather than transected, the misdirection of the regenerating axons leads to inappropriate reinnervation and hence no effective laryngeal function.[25, 33, 35, 39] Reinnervation techniques are used to improve adduction in human patients with unilateral vocal fold paralysis.[27] In dogs, however, adequate abduction is more important than the quality of the voice and in bilateral laryngeal paralysis the restoration of the air passage through the larynx is of immediate importance. The alternative is to create a permanent tracheostoma (Chapter 5.8.4).

Arytenoid cartilage lateralization (Figures 4.21 a, b). The aim of this surgical procedure is to widen the laryngeal inlet in bilateral laryngeal paralysis. King first reported its use in humans in 1939, with the aim of fixing the arytenoid cartilage in a more lateral position during healing after transposition of the omohyoid muscle to the arytenoid cartilage.[22] The suture used to fix the arytenoid was catgut, which would be absorbed in two weeks, and then the lateralization would be taken over by the action of the omohyoid muscle. Arytenoid cartilage lateralization was later used without omohyoid muscle transposition. The first publication on its use in dogs was based on experience with this technique in young Bouviers with spontaneous laryngeal paralysis.[17, 61] The aim was to permanently widen the opening of the larynx with a minimum of laryngeal trauma, and improvement in respiratory ability was given priority over vocalization. The technique is recognized as being successful in both dogs and cats.[31] Its advantage is that it can be performed without bilaterally rupturing the laryngeal mucosa and this minimizes webbing.[73]

With the dog under anesthesia in dorsal recumbency and with an endotracheal tube in place, the procedure begins with tracheostomy (Chapter 5.8.3). The endotracheal tube passing through the laryngeal lumen is then removed and a sterile endotracheal tube is inserted through the tracheostoma, cuffed, and connected to the anesthetic equipment. A paramedian incision is made 1 cm off the midline over the larynx on the side of the intended lateralization. The subcutis is separated over the sternohyoid muscle. The caudodorsal edge of the thyroid cartilage can then be palpated through the thyropharyngeal muscle (Figure 4.21 c). Using a finger to lift the dorsal edge of the thyroid cartilage, an incision of 1 to 1.5 cm is made through the thyropharyngeal muscle over the caudodorsal edge of the thyroid cartilage (Figure 4.21 d). Using closed blunt-tipped scissors, the cricothyroid articulation is located and disarticulated without rupturing the joint cartilages. The caudodorsal edge of the thyroid cartilage is then lifted in order to view the intrinsic laryngeal muscles (Figure 4.21 e). The atrophy of the laryngeal muscles exposes the cricoarytenoid articulation as a cartilaginous elevation. If the muscles are not atrophied, the articulation can be palpated under the junction of the dorsal cricoarytenoid muscle and the thyroarytenoid muscle. The dorsal cricoarytenoid muscle is severed from its attachment to the arytenoid cartilage and the cricoarytenoid articulation is separated in the joint (Figure 4.21 f).



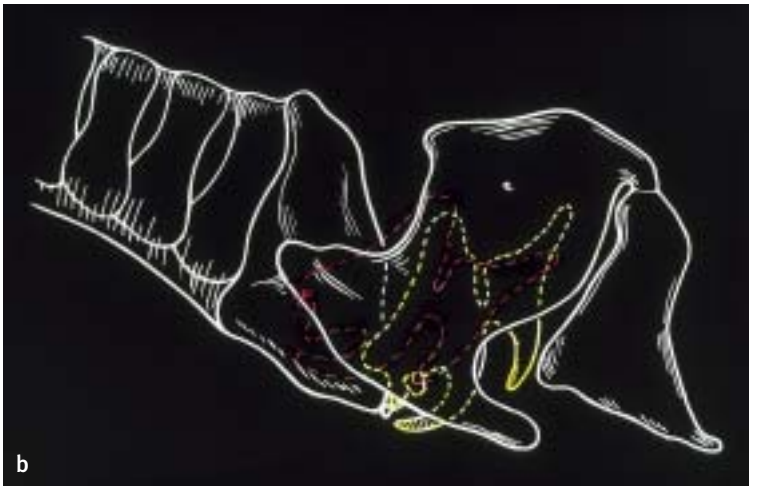
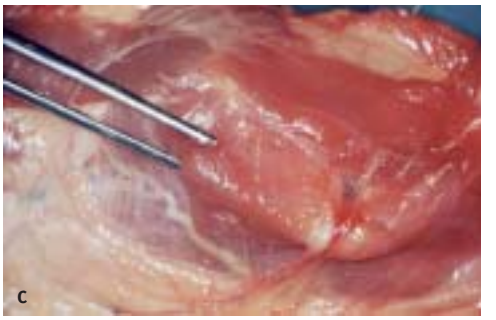
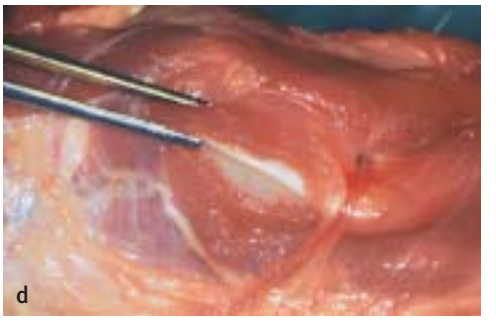
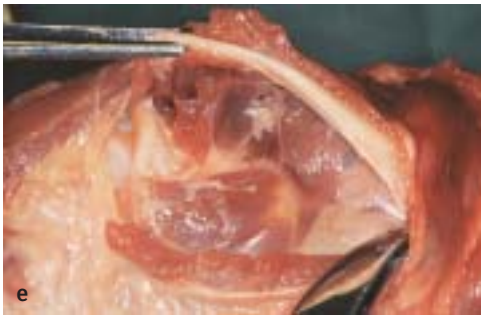


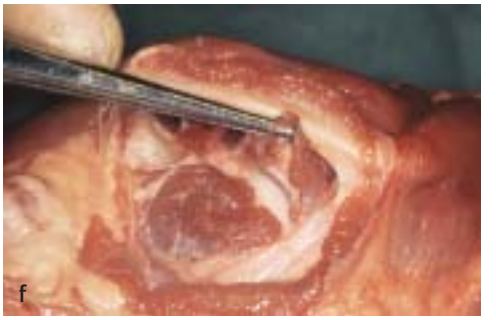
Figure 4.21 a–i:
Diagram of the cartilageous laryngeal skeleton of a dog in dorsal recumbency. The normal position of the left arytenoid cartilage is indicated by the yellow dotted line (a) and its position after lateralization is indicated by the red dotted line (b). The caudodorsal edge of the thyroid cartilage can be palpated through the thyropharyngeal muscle (c). The thyropharyngeal muscle is incised over the dorsal edge of the thyroid cartilage (d). After disarticulation of the cricothyroid cartilage, the thyroid cartilage is lifted to expose the intrinsic laryngeal muscles (e). The dorsal cricoarytenoid muscle is severed and then the cricoarytenoid joint is disarticulated (f). Atraumatic twisted stainless steel suture material is passed through the caudodorsal edge of the thyroid cartilage and through the muscular process of the arytenoid cartilage (g, h). (Routinely, a second stainless steel suture is placed through the caudodorsal edge of the thyroid cartilage just cranial to the previous suture and through the muscular process slightly dorsal to the previous suture). The incision in the thyropharyngeal muscle is sutured (i). (From: Harvey CE, Venker-van Haagen AJ. Surgical management of pharyngeal and laryngeal airway obstruction in the dog. In: Vet Clin North Am, Surgical techniques in small animal practice. 5: 515–535, Figs. 3 A–D. Copyright 1975, WB Saunders Company, with permission from Elsevier).

The arytenoid cartilage is partly separated from the cricoid cartilage ventrally and dorsally by cutting part of the mucosal connection (often indurated in older dogs) between the cricoid

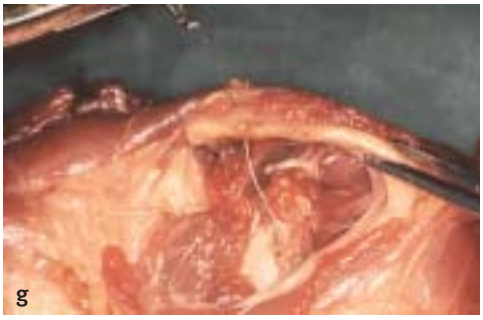


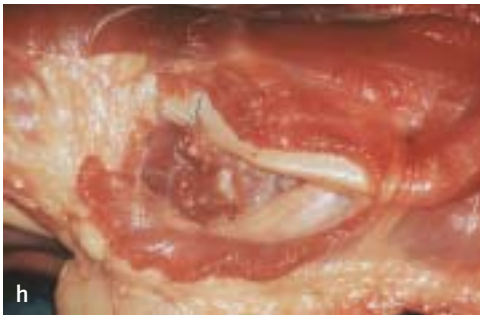


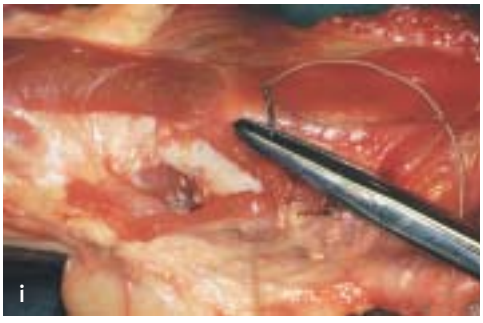


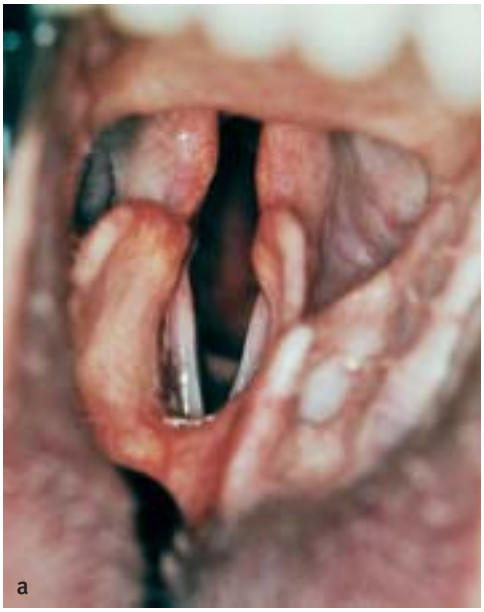


and arytenoid cartilages. The arytenoid is then separated from the contralateral arytenoid by severing the connective tissue and sesamoid cartilage joining them on the dorsal midline. The effect of this separation and the intended lateralization is inspected via the mouth with the laryngoscope while the arytenoid cartilage is pulled into the intended position under visual guidance. The effect is considered satisfactory if the detached arytenoid cartilage can be lateralized without the need for traction on the contralateral arytenoid cartilage and if there is satisfactory widening of the laryngeal opening (Figures 4.22 a, b). Atraumatic twisted stainless steel suture material is placed through the caudodorsal edge of the thyroid cartilage and then through the muscular process of the arytenoid cartilage (Figures 4.21 g, h). A second stainless steel suture is placed through the caudodorsal edge of the thyroid cartilage just cranial to the previous suture and through the muscular









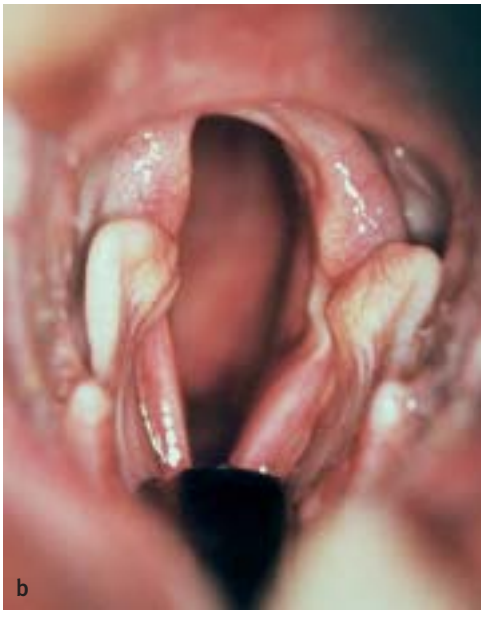


Figure 4.22 a, b: Bilateral laryngeal paralysis in a dog, resulting in a narrow laryngeal inlet (a). After lateralization, the left arytenoid cartilage and left vocal fold are fixed in abduction (b).

process slightly dorsal to the previous suture. This extra suture prevents pivoting of the arytenoid cartilage after fixation. In cats and small dogs the second suture is omitted because of the smaller size of the muscular process of the arytenoid cartilage. The incision in the thyropharyngeal muscle is sutured (Figure 4.21 i) and the wound is closed routinely. When the dog breathes spontaneously the endotracheal tube is removed from the tracheostomy wound and a tracheal cannula is put in place for five days.

There are several variations of the above procedure and some surgeons omit the tracheostomy.[31] In older dogs with a well-developed larynx the laryngeal opening that is achieved may allow this, but one should be prepared to insert a tracheal cannula if the patient becomes restless and its breathing stridorous. Edema caused by irritation of the laryngeal mucosa may cause the opening to be smaller than anticipated and severe dyspnea may occur.

Complications of laryngeal surgery often have severe consequences. The airway may again become obstructed or failure of the mucosal sensory function may result in aspiration. There are various reasons for failure of the lateralization of the arytenoid cartilage. The laryngeal opening can become obstructed if the sutures tear through the cartilage before the arytenoid becomes consolidated in its new position. To help prevent this, exercise, excitement, and barking should be strictly limited for four weeks. Secondly, a harness should be used in place of a collar—for the remainder of the dog's life—beginning from the moment of awakening after surgery. One fierce tug on a collar at that time could disrupt the fixation of the arytenoid. After surgery the larynx will be found by palpation to be hard and more voluminous and there may be recurrent laryngitis because of the unnatural laryngeal opening. If disruption of the arytenoid fixation occurs within less than a month after surgery, reattachment is usually possible. Otherwise, it is easier to lateralize the arytenoid cartilage on the opposite side.

Another reason for failure of lateralization is a lack of sturdiness of the cartilage, such as in very young dogs (before 5 to 6 months of age) and in dogs with a hypoplastic larynx. As a result of its softness, the larynx collapses after surgery and further surgery usually fails to create a sufficient laryngeal opening. A choice must then be made between a permanent tracheostoma and euthanasia.

After arytenoid cartilage lateralization the glottis is permanently abducted on one side. In most cases the contralateral side of the larynx is also paralyzed or partly paralyzed and the glottis cannot close properly. When the sensory nerves—the cranial laryngeal nerves—are intact, the normal swallowing action prevents the leakage of fluid and food into the larynx and trachea. When the internal branch of the cranial laryngeal nerve is crushed or transected during surgery, even unilaterally, the swallowing action may be impaired (Chapter 3.1.2) and aspiration may occur. In neurogenic degenerative diseases and in polyneuropathy, the sensory nerves may be involved in the disease and with the laryngeal inlet permanently open, aspiration may occur. Evaluation of the swallowing action should be included in the clinical evaluation before arytenoid lateralization (Chapter 3.1.2).

Laryngeal paralysis caused by neuronal degeneration

Slowly evolving degeneration of neurons underlies many of the heritable and sporadic disorders of the central nervous system. In some disorders progressive axonal changes precede loss of perikaryon, while in others, including motor neuron diseases, subacute perikaryal degeneration precedes loss of nerve fibers. Although these distinctions are helpful concepts, the neuron is a single functional unit and various types of neuronal changes can be found at vari-

ous stages of a disease in progress.[15] These diseases may affect the recurrent laryngeal nerves and cause laryngeal paralysis. In peripheral nerves such as these, which are most accessible for biopsy, it is useful to consider separately the two major types of axonal degeneration, wallerian degeneration and distal axonal degeneration. Wallerian degeneration follows several days after interruption of the axon, distal to the lesion. The process of distal degeneration is usually regarded as a length-dependent vulnerability and, in human neuropathology, underlies the initial involvement of the feet and ankles in many axonal neuropathies.[72] Slowly-evolving perikaryal degeneration and loss are outstanding findings in a variety of neuronal degenerative disorders. In most disorders, the perikaryon undergoes nonspecific changes. The relation of these alterations to the pathogenesis of neuronal loss is unclear.[15] Primary degeneration of the nerve cell bodies can lead to loss of axons and the pathological changes resemble those of acute or chronic axonal degeneration.[47]

The common characteristics of the neuronal degenerative diseases are: (1) they are selective, affecting one or more »systems« of neurons bilaterally in a more or less symmetrical pattern; (2) they are steadily progressive, although not necessarily fatal; (3) they are variable in their clinical and pathological features; and 4) some have a clear genetic basis.[26]

Laryngeal paralysis caused by neuronal degenerative disease in young Bouviers. During a period of 6 years, at a time when the Bouvier had become a very popular breed in the Netherlands, 105 Bouviers (78 males and 27 females) were presented at the small animal clinic of the University of Utrecht, with the leading signs of noisy breathing and decreased endurance. At the first examination, 77 of these dogs were four to eight months of age and the others were older. The onset of the clinical signs (if known) was at four to six months of age in all dogs and the signs were progressive in frequency and character. Laryngeal stridor was a dominant sign in all of them. Clinical examination and hospital admission often constituted a severe enough stress to necessitate immediate intubation, followed by tracheostomy. In three of these dogs there was bilateral absence of flexion of the tarsal joint.[61]

Laryngoscopy revealed that in 28 of the 105 affected Bouviers there was immobility of both vocal folds. In 71 there was immobility of the left vocal fold and moderate abduction of the right during inspiration, in 3 there was slight motion of both vocal folds, and in 3 there was clear abduction of both vocal folds during inspiration. In all 105 dogs there were signs of laryngitis. In 40 of the dogs EMG recordings were made from the intrinsic laryngeal muscles: in all 40 from the left and right dorsal cricoarytenoid muscles, in 34 also from the left and right thyroarytenoid and ventricular muscles, and in 8 of the latter also from the cricothyroid muscles. In three dogs recordings from the dorsal cricoarytenoid muscles were normal and in 25 there were denervation potentials bilaterally (fibrillation potentials, positive waves, and complex repetitive discharges) (Figures 4.23 a–d). Of the 34 dogs in which recordings were made bilaterally from the ventricular, thyroarytenoid, and dorsal cricoarytenoid muscles, 14 had denervation potentials in all of these muscles and all 34 had denervation potentials in at least one muscle. Thus there was no single pattern of denervation but rather various patterns of normal and denervated muscles on both sides. In 35 muscles denervation potentials and normal action potentials were recorded simultaneously (Figure 4.23 c). In all 8 dogs in which recordings were made from both cricothyroid muscles, normal action potentials were observed during expiration. In the 3 dogs in which there was bilateral absence of flexion of the tarsal joint, denervation potentials were recorded from all six cranial tibial muscles.[61]

Histology of laryngeal muscles and nerves. In 53 of the Bouviers a biopsy of the dorsal cricoarytenoid muscle was obtained for histological examination during surgery to lateralize the arytenoid cartilage. Seven dogs became available for necropsy and in these all intrinsic laryngeal muscles and the recurrent laryngeal and vagus nerves were removed for histological examination. All 53 biopsy specimens from the cricoarytenoid muscles and all of the intrinsic laryngeal muscles of the 7 dogs obtained at necropsy revealed changes characteristic of denervation atrophy. There was a constant finding of angular fibers of smaller diameter, occurring singly, in groups, or occupying entire fascicles, together with an increase in number of sarcolemmal nuclei in the atrophic fibers. These findings have been described in denervation atrophy in both humans and animals.[61, 71, 72] The occurrence of numerous digestive chambers in the myelin sheaths was the most notable feature in the recurrent laryngeal nerves. There was disintegration of the axons in the digestive chambers and fragments of axons

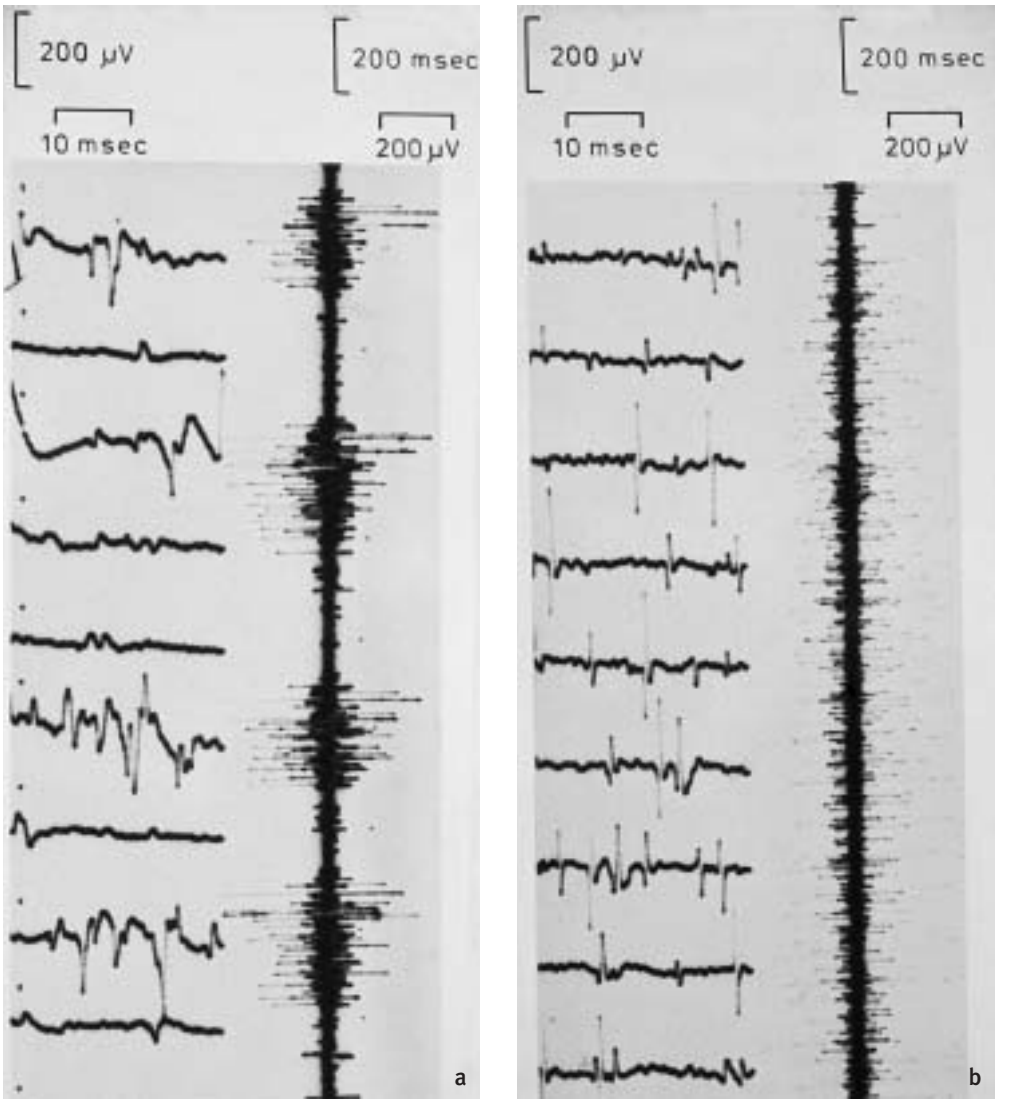
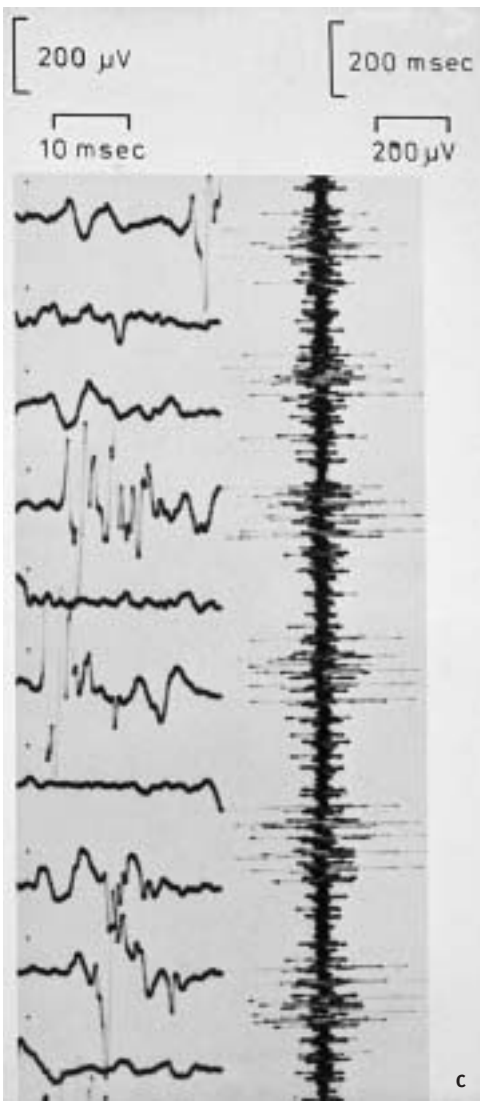
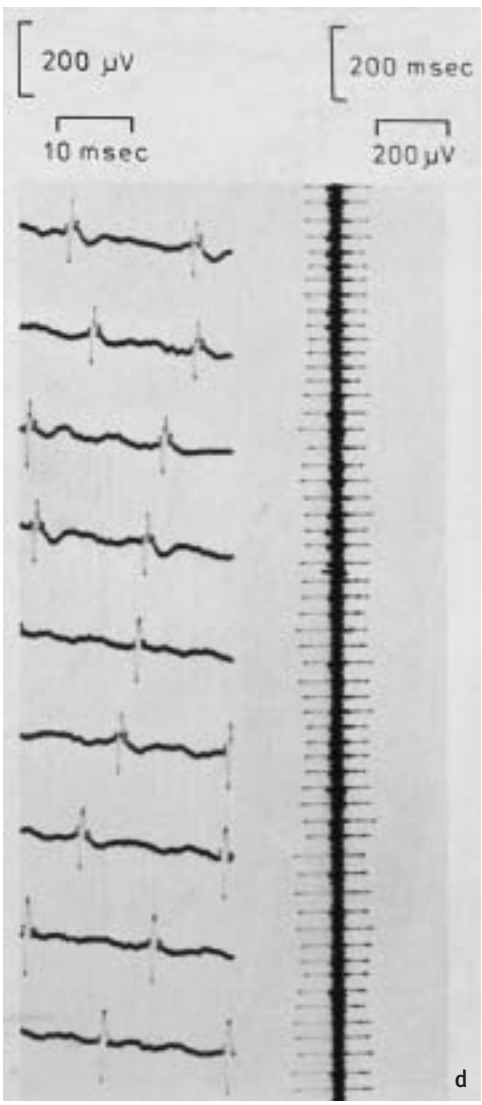


Figure 4.23 a–d: (a) Electromyogram showing normal action potentials during expiration, in the normal left ventricular muscle of an 8-month-old Bouvier. Paper speed 5 cm/sec, sweep speed 10 msec/div, amplitude 200 µV/div. (b) Electromyogram showing denervation potentials in the left ventricular muscle of an 8-month-old Bouvier. Paper speed 5 cm/sec, sweep speed 10 msec/div, amplitude 200 µV/div. (c) Electromyogram showing normal activity together with denervation potentials, in the left thyroarytenoid muscle of a 6-month-old Bouvier. Paper speed 5 cm/sec, sweep speed 10 msec/div, amplitude 200 µV/div. (d) Electromyogram showing complex repetitive discharges (CRDs) in the right ventricular muscle of a 6-month-old Bouvier. Paper speed 5 cm/sec, sweep speed 10 msec/div, amplitude 200 µV/div. (From: Venker-van Haagen AJ, Hartman W, Goedegebuure SA. Spontaneous laryngeal paralysis in young Bouviers. J Am Anim Hosp Assoc 1978; 14: 714–720, Figs. 2–5).

were often visible. There was an increase in endoneural connective tissue and in the number of Schwann cells, indicating wallerian degeneration. All of these abnormalities were present throughout the length of the nerves. In the vagus nerve, only a few digestive chambers were observed in otherwise normal nerve tissue. Macroscopically and microscopically there were no signs of traumatic lesions surrounding the vagus or recurrent laryngeal nerves.[61] The presence of wallerian degeneration in both recurrent laryngeal nerves and its occurrence throughout the length of these nerves, together with the absence of traumatic lesions in the vagus nerves, strongly indicated that the lesion was in the perikaryon.[61]

Hereditary transmission of laryngeal paralysis in Bouviers was studied in a family of 22 Bouviers and 13 Bouvier-greyhound crossbreds (Figure 4.24). The parent (P) generation consisted of two Bouviers with laryngeal paralysis, donated for this study.[64] The diagnosis of laryngeal paralysis was based on electromyographic





examination of the left and right dorsal cricoarytenoid, ventricular, and thyroarytenoid muscles. EMG studies were performed in the P generation Bouviers and the greyhounds before breeding. In all dogs of the F1, F2, and F3 generations recordings were made at 8 weeks of age. In the F1 generation recordings were repeated at three months and one year of age. Among these 6 dogs, there were 6 affected laryngeal muscles at the age of 8 weeks and 25 affected muscles at the age of 12 weeks, indicating progression of the disease during that interval. The diagnosis of laryngeal paralysis was based on the observation of denervation potentials in at least one of the intrinsic laryngeal muscles (Figures 4.23 b–d). Since 5 of the 6 Bouviers in the F1 generation had laryngeal paralysis, hereditary transmission of the disease was considered likely. The purpose of mating the dam with a son and the sire with a daughter was to concentrate the genes responsible for the disease in the resulting offspring. The mating of two of these inbred offspring with greyhounds, a breed having no relation to the Bouvier and no history of laryngeal paralysis, was undertaken in order to obtain litters which could be maximally heterozygous for the genes causing this defect. The results demonstrate that the disease is brought about by autosomal, dominant gene(s) with 100 % penetration in the F3 generation. The one unaffected male dog in the F1 generation can be explained by assuming a homozygous recessive genotype in this dog. It was concluded that since laryngeal paralysis in Bouviers is transmitted as a dominant trait and the disease can be diagnosed before the age of breeding, its prevention by controlled breeding was possible. Now, twenty years after the study, laryngeal paralysis appears to have become only a sporadic occurrence in Bouviers.

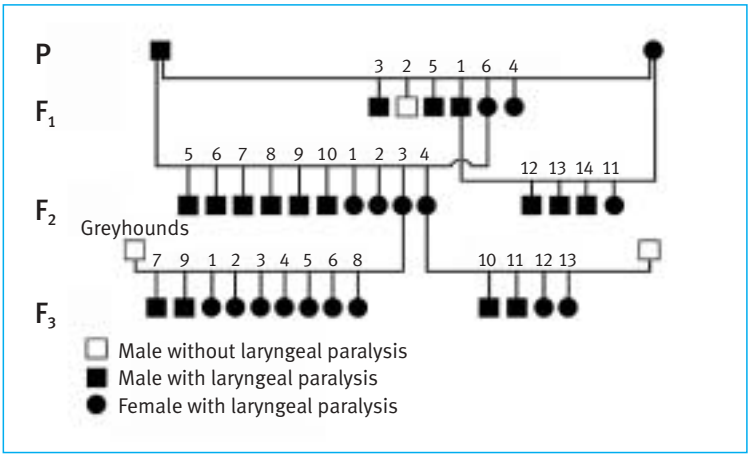


Figure 4.24:
Pedigree of a family of 22 Bouviers and 13 Bouvier-greyhound crossbreds in a study of hereditary transmission of laryngeal paralysis in Bouviers. (From: Venker-van Haagen AJ, Bouw J, Hartman W. Hereditary transmission of laryngeal paralysis in Bouviers. J Am Anim Hosp Assoc 1981; 17: 75–76, Fig. 1).

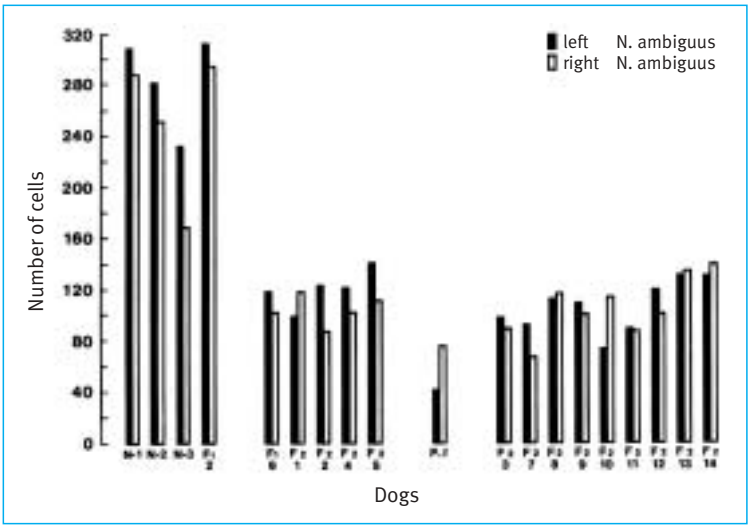


Figure 4.25:
Number of cells in the nucleus ambiguus, counted in one of every five serial sections (15 µm thickness) of the entire brain stem in 4 Bouviers without laryngeal paralysis (N-1, N-2, N-3, unrelated to the family shown in Figure 4.24, and the normal Bouvier in the F1 generation of the family, and in 15 Bouviers with laryngeal paralysis identified in the pedigree in the P generation, the F1 generation, and the F2 generation. (From: Venker-van Haagen AJ. Investigations on the pathogenesis of hereditary laryngeal paralysis in the Bouvier, Thesis, 1980, Utrecht University, Fig. 25).

Histological studies of the nucleus ambiguus in Bouviers with laryngeal paralysis. It is generally recognized that the nucleus ambiguus is the motor nucleus for the striated muscles of the larynx and pharynx and, in dogs, the striated muscles of the esophagus. Kosaka (1909) investigated the motor function of the nucleus

ambiguous for the intrinsic laryngeal muscles in dogs and concluded that it contains all of the motor neurons for the intrinsic laryngeal muscles.[23] Later investigations found evidence of a rostral-to-caudal pattern of motor neurons in the nucleus ambiguus corresponding to the caudal-to-cranial localization of the intrinsic laryngeal muscles.[11, 57, 62]

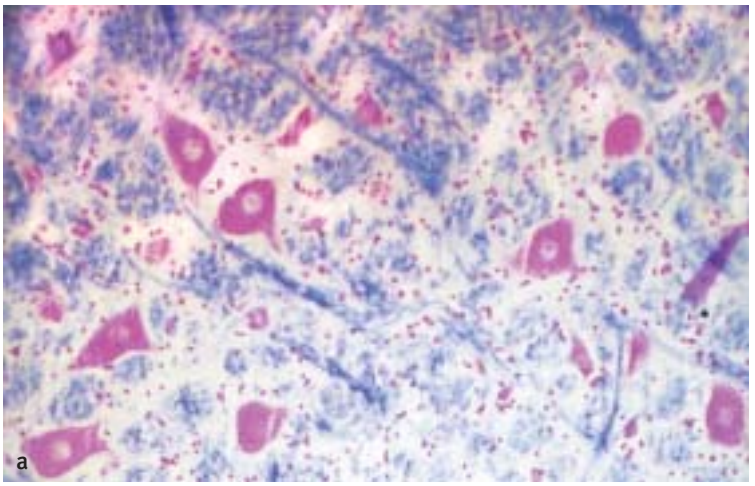
The histology of the nucleus ambiguus was studied in 4 Bouviers without laryngeal paralysis and 20 Bouviers with laryngeal paralysis. One of those without laryngeal paralysis and all 20 with laryngeal paralysis are presented in the pedigree showing the results of mating Bouviers with laryngeal paralysis (Figure 4.24). The most striking finding in the nucleus ambiguus in the Bouviers with laryngeal paralysis was the distinctly smaller number of motor neurons in comparison with the number in Bouviers without laryngeal paralysis (Figure 4.25). In each dog with laryngeal paralysis the nucleus ambiguus also contained motor neurons in a state of degeneration, these not being found in the dogs without laryngeal paralysis. In addition, oligodendroglial cells in clusters over the outlines of the degenerated large motor neurons were found exclusively in dogs with laryngeal paralysis (Figures 4.26 a–d). However, it has never been demonstrated conclusively that these changes invariably lead to definite loss of the degenerated neuron. The signs of degeneration of motor neurons observed in Bouviers with laryngeal paralysis differ from the alterations which have been described as chromatolysis, the basic reaction of the neuron to injury.[14] Chromatolysis was not observed in the nucleus ambiguus of the Bouviers with laryngeal paralysis.[62] These findings together with the finding of wallerian degeneration in the recurrent laryngeal nerves, the neurogenic atrophy in the intrinsic laryngeal muscles, and the denervation potentials in the electromyograms of the intrinsic laryngeal muscles of the Bouviers with laryngeal paralysis provide clear evidence of a *loss* of motor neurons. Although the fibers of the recurrent laryngeal nerve could not be identified separately in their common course with the vagus nerve, the evidence points most strongly to the cell bodies of the nucleus ambiguus as the site of origin of the disease. This evidence includes the bilateral occurrence of the lesion, the progressive increase in the number of affected laryngeal muscles between 8 and 12 weeks of age, the progressive degeneration of the recurrent laryngeal nerves (Figures 4.27 a–c), and the type of degenerative changes in the perikaryon.[62]

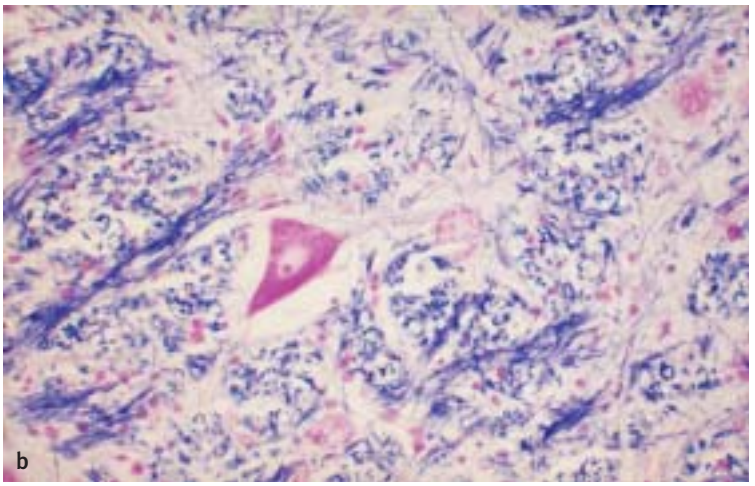
In summary, it was concluded that laryngeal paralysis in Bouviers could be characterized as a neuronal degenerative disease because: (1) it affects one or more »systems« of neurons in a more or less symmetrical pattern, (2) it is progressive, and (3) it has a genetic basis.

This study in Bouviers could be a model study for families of dogs with laryngeal paralysis.[62] The criteria for neuronal degenerative disease may vary in their clinical and pathological features and one of the variations may be that the clinical signs appear later in life than in these Bouviers.[72] One example of this may be the Husky and Husky-crossbred family in which the clinical and pathological findings were indicative of neuronal degenerative disease but in which the clinical signs appeared both in young dogs and later in life.[41] Early recognition of a hereditary basis can prevent further spreading of the disease.

Laryngeal paralysis as one sign of polyneuropathy

Some Bouviers with laryngeal paralysis also had cranial tibial muscle paralysis, and the same combination was found in Leonbergers.[53] In humans there have also been cases of familiar laryngeal paralysis together with peroneal paralysis, muscular pareses in the upper extremities, and sensory loss.[12, 18] The occurrence of laryngeal paralysis as one sign of polyneuropathy was well documented in 6 dogs, comprising 3 young Dalmatians, one young





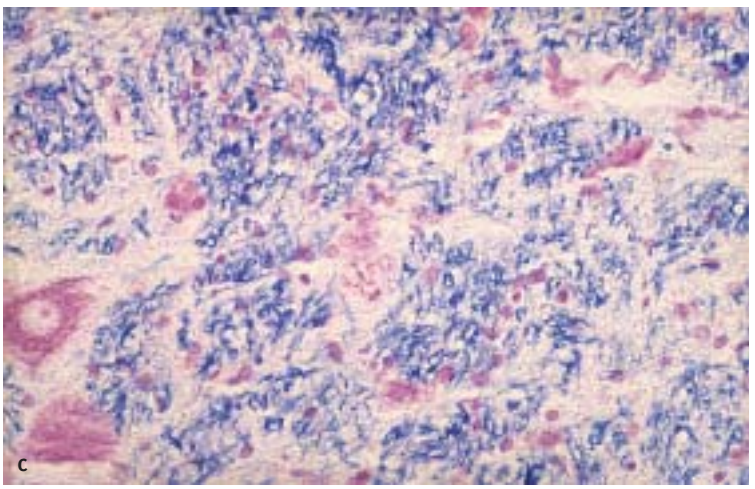


Figure 4.26 a–d:
(a) Elongated multipolar motor neurons in the nucleus ambiguus of a dog without laryngeal paralysis (Klüver-Barrera stain). (b) Normal motor neuron (just left of center) and degenerated motor neuron (just right of center) in the nucleus ambiguus of a Bouvier with laryngeal paralysis (Klüver-Barrera stain). (c) Glial reaction around a large motor neuron (center) in the nucleus ambiguus of a Bouvier with laryngeal paralysis (Klüver-Barrera stain).
(d) Longest linear dimension of the cells of the nucleus ambiguus (N.A.), measured in one of every five serial sections of the entire brain stem, in 4 Bouviers without laryngeal paralysis and 15 Bouviers with laryngeal paralysis (for identification see Figure 4.25). (From: Venker-van Haagen AJ. Investigations on the pathogenesis of hereditary laryngeal paralysis in the Bouvier, Thesis 1980, Utrecht University, Fig. 26).

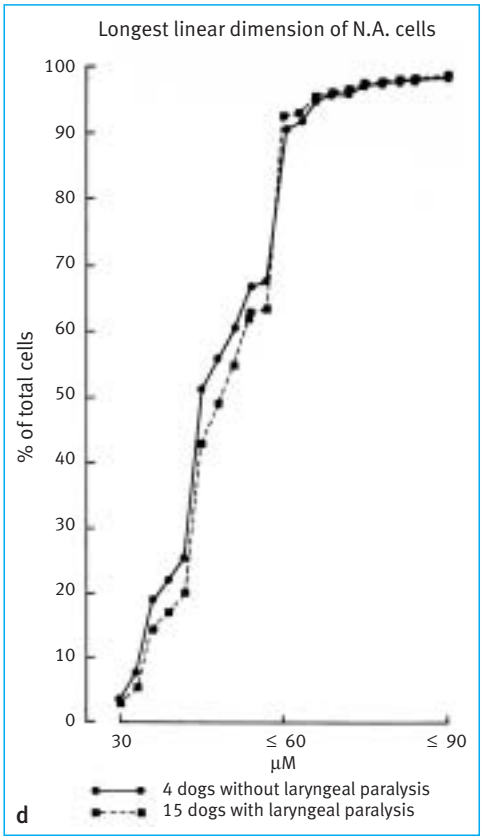
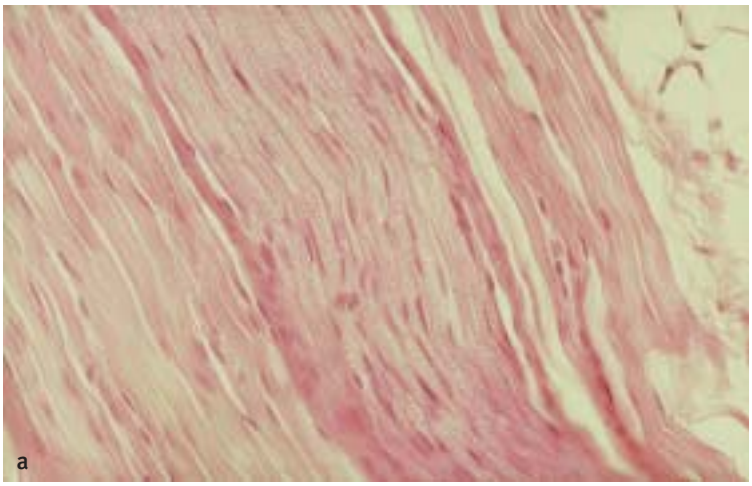
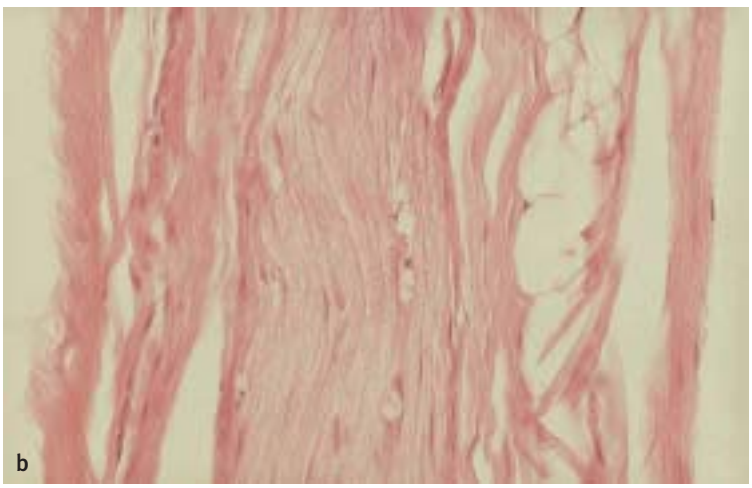
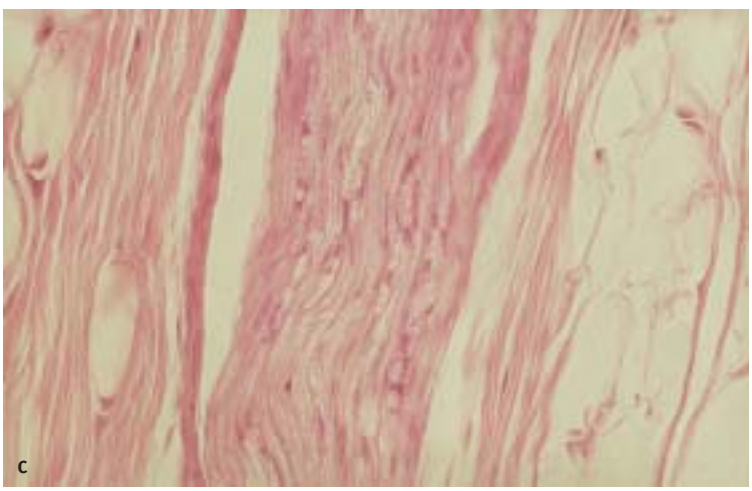


Figure 4.27 a–c:
(a) Recurrent laryngeal nerve of a 14-week-old crossbred Bouvier with laryngeal paralysis (Figure 4.24, F3). There are scattered small digestive chambers in the myelin sheaths (H.E. stain). (b) Recurrent laryngeal nerve of a 20-week-old crossbred Bouvier with laryngeal paralysis (Figure 4.24, F3). There are chains of digestive chambers in the myelin sheaths (H.E. stain). (c) Recurrent laryngeal nerve of a 24-week-old crossbred Bouvier with laryngeal paralysis (Figure 4.24, F3). There are numerous digestive chambers in the myelin sheaths (H.E. stain).







Bouvier, an 11-year-old Great Pyrenees, and a 12-year-old Irish setter. The pattern of distal limb muscle paresis and occurrence of denervation potentials in electromyographic recordings varied from dog to dog.[4] In a later report the same author described findings in 14 young Dalmatians with bilateral laryngeal paralysis and polyneuropathy. Denervation potentials were found not only in the muscles distal to the stifle and elbow joints, but also in the esophagus and facial muscles, and there was an indication of diminished sensory nerve conduction velocity.[5]

4.8.2 Laryngeal spasm

Laryngeal spasm is the acute inappropriate closing of the laryngeal opening by adduction of the vocal folds long enough to result in extreme dyspnea and forced inspiration. Functional laryngeal spasm is the term used when neither structural abnormalities nor peripheral nerve injuries are found to account for the abnormalities in laryngeal function. The disorder occurs most often in dogs and only under specific conditions when the dog is alert. The term »functional« is taken from similar disorders in humans known as »functional voice disorders«. These disorders are thought to be due to abnormalities in central nervous system control or behavior. Functional voice disorders may be idiopathic, attributable to misuse of the larynx for voice production, or psychogenic, owing to psychological difficulties that interfere with normal voice production. Most of these disorders have a distinctive pattern, allowing them to be recognized as separate disorders.[27] A recognized example of functional laryngeal spasm is that which occurs in dogs undergoing severe training. It occurs in both experienced and inexperienced dogs and always when they are being trained to attack a man. This is usually a part of the training session in which the excitement of the dogs is maximal. Fierce barking is part of the expected behavior and laryngeal spasm occurs during the actual attack, causing the dog to halt abruptly, in severe dyspnea. The spasm can last for several agonizing minutes and if it persists too long the veterinarian will be confronted with a barely conscious dog with cyanosis. Sedation, endotracheal intubation, and administration of oxygen will bring recovery, and homeostasis will be restored within 24 hours. In most cases the dog recovers quickly and training is usually resumed after a week. However, it is characteristic of this disorder that during all subsequent training sessions the spasm recurs when the dog is placed in the same situation, and always during the attack on the man. Clinical examination of a dog with such a history reveals a healthy animal which has a good record for exertion and the ability to continue trotting over long distances. Radiographs of the lungs, EEG, and cardiac ultrasonography reveal no abnormalities. Laryngoscopy reveals normal functioning of the larynx and normal laryngeal mucosa, although there may be temporary redness soon after a spasm has occurred. Electromyography of the laryngeal muscles reveals normal action potentials during inspiration and expiration. When no abnormalities are found, functional laryngeal spasm is the most probable diagnosis. The outlook for training of these dogs is poor, for once an episode has occurred, no treatment—such as avoiding training for 3 months, tranquilizers, etc.—has been found successful. Humans afflicted with laryngeal spasm are attended by a team that includes an otolaryngologist, a speech pathologist, and a psychologist. Many experienced dog trainers have been disappointed in their hopes that with tact, care for the dog, and understanding of the disorder, it might be overcome. The advice that should be given is to stop the attack training and keep the dog for other work.

Laryngeal spasm is also frequently found to be associated with laryngeal paralysis in dogs and sometimes in cats. It occurs at moments of excitement or stress and since dyspnea caused

by laryngeal paralysis is stressful, the spasms become more frequent as the disease progresses. Laryngeal spasm occurring during acute stress should be managed by sedation, endotracheal intubation, and restoration of homeostasis, followed by tracheostomy and surgical treatment for laryngeal paralysis, namely, lateralization of the arytenoid cartilage. If laryngeal spasm recurs when the tracheal cannula is removed, replacing the cannula for another day or two is usually sufficient. However, if the laryngeal spasm is refractory, bilateral resection of the recurrent laryngeal nerves in their course inside the larynx, together with lateralization of the second arytenoid cartilage, usually solves the problem.

Laryngeal spasm occurs in cats when stimulation of the cranial nerve receptors leads to a prolonged adductor response, continuing well after the initiating stimulus is removed. It can be provoked by inserting or removing an endotracheal tube or by touching the laryngeal mucosa while aspirating mucus from the laryngeal inlet. It can be prevented by spraying 10 % lidocaine on the mucosa before such procedures.

4.8.3 Paradoxical vocal fold movement

Paradoxical vocal fold movement is the adduction of the vocal folds during inspiration and abduction during expiration. It can affect one or both vocal folds. Adduction during inspiration causes stridorous breathing but abduction during expiration may cause no clinical signs. In cats paradoxical vocal fold movement is often observed together with signs of laryngitis during laryngoscopy. The laryngoscopy is usually motivated by a history of unpredictable episodes of stridorous breathing or dyspnea, alternating with periods of normal breathing. The dyspnea or stridor occurs in short periods and is usually not life-threatening. The underlying cause is usually laryngitis and treatment for laryngitis is accompanied by disappearance of the signs. The phenomenon may also be observed in dogs which subsequently develop signs of bilateral progressive laryngeal paralysis. The paradoxical vocal fold movements are seen during laryngoscopy and then electromyography of the intrinsic laryngeal muscles usually reveals denervation potentials together with normal motor unit potentials during both inspiration and expiration. The clinical signs are transitory and laryngeal surgery is not yet necessary, but in most cases it will be needed within a year.

4.8.4 Sensory laryngeal paralysis and laryngeal dysfunction

The mucosal receptors in the glottic area are those of the cranial laryngeal nerves. They respond to stimuli such as touch and contact with liquids. The reflex following stimulation of these receptors closes the glottis. Electrical stimulation of the cranial laryngeal nerve (SLN) with a single pulse causes visible contraction of the muscles involved in swallowing but does not activate the swallowing action.[67] This closure reflex is thought to represent a defense mechanism to protect the lower respiratory tract. Stimulating the SLN with a train of electrical pulses evokes the swallowing action.[68] After unilateral SLN transection in 5 dogs, all continued to eat normally and none developed signs of aspiration, although electromyography revealed irregularities in the swallowing action. After bilateral SLN transection in 5 other dogs, only one continued to eat normally and the other 4 aspirated water and saliva and scarcely ate at all.[69] Spontaneous loss of function of the cranial laryngeal nerves has been described in polyneuropathies. Since in these diseases the neuropathy is bilateral, aspiration may occur. When vocal fold paralysis causes dyspnea but there is also a history of dysphagia or aspiration, lateralization of the arytenoid cartilage

raises a dilemma because it may increase the risk of aspiration considerably. Lateralization of the arytenoid cartilage may be successful in a dog with vocal fold paralysis due to polyneuropathy without dysphagia or aspiration, but since polyneuropathy is usually progressive, cranial laryngeal nerve paralysis may develop later in life and dysphagia and aspiration may then occur.

Laryngeal sensory nerve paralysis may result from surgery to lateralize the arytenoid cartilage or other surgical procedures in which the cranial laryngeal nerve or the internal branch of the cranial laryngeal nerve is involved. Unilateral laryngeal sensory nerve paralysis following experimental transection of the SLN did not result in aspiration,[69] but in bilateral laryngeal surgery and bilateral cranial laryngeal nerve involvement severe dysphagia and aspiration is to be expected.

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5 The Trachea and Bronchi

5.1 Functional considerations

The trachea begins at the caudal border of the laryngeal cricoid cartilage, the only complete cartilaginous ring in the airway. In dogs the trachea is composed of approximately 35 C-shaped »rings« of hyaline cartilage, providing a noncollapsible tube extending from the larynx to the bifurcation that gives rise to the main stem bronchi, dorsal to the base of the heart. The space between the ends of all of the C-shaped rings is on the dorsal side of the trachea and is bridged by fibers of the smooth, transversely-running tracheal muscle and connective tissue. The rings are united in the longitudinal direction by bands of fibroelastic tissue, the annular ligaments of the trachea. They allow considerable intrinsic movement in the trachea.[20, 44]

5.1.1 Trachea and bronchi facilitate the respiratory airflow

The construction of the trachea prevents it from collapsing, while at the same time allowing it to change in length when the neck is extended and when the diaphragm contracts. Variations in diameter are effected by the tracheal muscle. The structure of the large bronchi is the same as that of the trachea. In the smaller bronchi the rings of cartilage are gradually replaced by irregular cartilaginous plaques and the eventual cessation of these marks the transition to bronchioles. Variations in diameter of the bronchi and bronchioles are relatively greater and more significant than those of the trachea.[15]

5.1.2 Trachea and bronchi condition the respiratory air

The trachea and bronchi are lined with pseudostratified, ciliated, columnar epithelium interspersed with mucus-secreting cells. Enlargement of the lumen during inspiration may lower the velocity of the airflow at the periphery of the air column sufficiently so as not to hamper the normal egression of the mucociliary blanket[55] or harm the alveoli.

The inspired air is warmed or cooled as it passes through the nose by radiation from or absorption of heat by the mucosal blood vessels, and in the same way the warming or cooling is continued in the trachea and bronchi. Humidification occurs by evaporation from the blanket of mucus covering the mucosa and the serous fluid from the tracheal and bronchial glands. The conditioning of the inspired air by the nose, trachea, and bronchi is a very important function for protection of the alveoli and it occurs even under extremely dry and cold conditions, even then providing the bronchi with air at body temperature and close to 100 % humidity.[4]

Mucosal cleaning. The pseudostratified epithelium lining the trachea and bronchi consists of ciliated, intermediate, basal, and goblet cells. They rest on a well-defined basement membrane supported by a deep, loose lamina propria containing small blood vessels, venous plexus, ducts of mucous and serous glands, sensory nerves, and blood cells. The tall ciliated cell is the predominant type and it extends from the basement membrane to the luminal surface, where there are cilia admixed with microvilli.[4] As in the nose, the cilia in the trachea and bronchi actively move the overlying blanket of mucus by a to-and-fro movement called the ciliary beat. The nasopharyngeal and the tracheobronchial parts of the ciliated airways transport mucus toward the oropharynx, where it can be eliminated by swallowing. Hence in the trachea

and bronchi the blanket of mucus is moved from caudal to cranial. The cilia make a forceful forward movement and a less forceful recovery. In the recovery stroke the shaft of the cilium curls back on itself so that it does not reach the layer of mucus. The forward movement transports the mucus blanket toward the laryngopharynx and the esophagus. The bilayered blanket of mucus, produced mostly by the serous and goblet cells, is sticky and tenacious. The outer layer is more viscid than the deeper or periciliary layer. The blanket functions as a lubricant, protects against desiccation, and traps insoluble particles and soluble gases.[4] Allergens and bacteria caught on the outer layer are thus carried to the esophagus. Soluble material reaches the periciliary layer and is removed with it. The motion of the cilium is caused by the sliding of one axonemal tubule against an adjacent one, creating a shearing force that induces bending and contact with the mucus, and may initiate the coordinated metachronous movement. The energy for this work is derived from adenosine triphosphate in the dynein arms of the axonemal tubules.[4] The tracheal mucociliary transport rate was measured in 11 conscious dogs which were conditioned to be restrained in dorsal recumbency. Boluses of ^{99m}technetium-labeled macroaggregated albumin were injected into the trachea and their movement was observed with a gamma camera positioned lateral to the dog's head and neck. The distance traveled by each bolus was measured relative to external markers and the observed tracheal mucociliary transport rate was 35.3 ± 15.9 mm/min (mean $\pm$ SD).[6] Coughing provoked by the injection could have increased the transport velocity, but coughing was infrequent and the observed effect in the dogs that coughed was inconsistent.[6]

Coughing is a violent expulsion of bronchial and tracheal air, and is a reflex activity following stimulation of »cough receptors« in the mucosa of the larynx, trachea, carina, and bronchi. The reflex is principally a defense mechanism that protects the lower respiratory tract and the alveoli. Inhaled irritants, mucus, and both mechanical and positional changes in the lung stimulate the cough receptors.

A single cough consists of a rapid, deep inspiration followed by a forced expiration against a closed glottis, and then sudden opening of the glottis with continued expiratory effort to expel air and any substance stimulating the cough. This basic pattern can have many variants in both man and animals, and it may be repeated to produce a sudden outburst of coughing. Afferent nerves from the receptors pass via the vagus nerve to the medulla oblongata, from which efferents are distributed to the respiratory and laryngeal muscles. Repeated coughing and paroxysmal coughing are signs of continuous irritation of the receptors by repeated or continuous mechanical or chemical irritants, and this continuous irritation may damage the mucosa. Many of the stimuli that cause coughing also activate other receptors and the ultimate breathing response is an interaction of reflexes. In this way the coughing reflex may be suppressed. Continuous coughing is the sign that indicates a threat to the upper airways.

5.2 History and clinical signs

5.2.1 History

The medical history in diseases of the trachea and bronchi usually reveals specific problems such as coughing, dyspnea, or stridorous breathing. Since all three of these signs may originate from parts of the airway other than the trachea and bronchi, as well as from the circulatory system, additional questions are asked about the animal's general condition, appetite, drinking, activity, and endurance, and about changes in its habits. The answers to these questions together with the results of a general

physical examination will point the way to the diagnosis.

5.2.2 Clinical signs

Coughing. Coughing is a reflex for the protection of the respiratory mucosa. The reflex is activated by stimulation of the »cough receptors« or is evoked voluntarily. Cough receptors are sensory receptors located in the mucosa of the larynx, trachea, carina, and bronchi. They respond to airway pressure, vocal fold motion, tactile stimuli, and chemical stimuli. The threshold for each of these stimuli is variable, according to the variable condition of the mucosa. For example, in laryngitis coughing is evoked by barking. The afferents from the receptors run with the cranial laryngeal and vagus nerves to the solitary tract and nucleus. The interneuronal network of the reticular formation activates the respiratory center and the motor nucleus of the vagus nerve and the nucleus ambiguus, inducing the cough reflex. The cough reflex begins with a deep inspiration with the glottis wide open. This is followed by closure of the glottis and strong contraction of the expiratory muscles, which builds up air pressure against the closed glottis. Then sudden opening of the glottis allows the air and any material that is present to be expelled forcefully. This basic pattern can have many variations. It may be repeated to produce a paroxysm of coughing. It may be fragmented by the repeated opening and closing of the glottis during a single expiratory effort. The relative contributions of inspiration and expiration may vary.[52]

The character of the cough may also vary. The cough in laryngitis and tracheitis is usually hard and dry. When the irritation is severe, paroxysmal coughing often leads to gagging. The dog's attempt to bark, or the cat's to purr, may elicit the characteristic dry cough. Laryngitis and tracheitis are often present at the same time and distinguishing between them is difficult in infectious disease. Collapse of the trachea is characterized by a honking cough, during each expiration in severe cases, or during exertion and excitement. The production of mucus in the trachea alone is not enough to produce a wet cough. Instead, a wet cough indicates chronic bronchitis or chronic bronchopneumonia. The production of mucus is not yet excessive in the acute stage of bronchitis and bronchopneumonia and thus initially the cough is painful and nonproductive. Otherwise in pneumonia the cough is soft and in pleuritis it can be soft and painful.

Coughing may also be due to heart failure. When the heart is enlarged the lumen of the intrathoracic trachea can be compromised by the increased cardiac volume, and the recurring stenosis of the trachea causes coughing. Coughing caused by stimulation of the vagus does not have a definite pattern. It may occur with each cardiac contraction or, for example, during cleaning of the ear canal. Many other examples have been reported.

Dyspnea. Dyspnea can be caused by obstruction of the trachea, as occurs in tracheal collapse in small breeds of dogs or tracheal neoplasia in both dogs and cats. In bronchitis and bronchopneumonia mucopurulent exudate may obstruct the airway during both inspiration and expiration. In asthma-like diseases in cats, constriction of the smaller bronchi and bronchioles may obstruct communication with the alveoli. These diseases must be distinguished from diseases of the lung parenchyma, but in both diseases of the trachea and bronchi and those of the lung parenchyma, the dyspnea is predominantly expiratory.

Stridorous breathing. Partial obstruction of the trachea may result in a particular extra sound during the respiratory cycle. It occurs during expiration and differs from coughing in being a softer, wheezing sound. It must be distinguished from the various stridors caused by

upper airway disease and from the soft vocalization that is sometimes heard when there is pain in the thorax.

5.2.3 Physical examination

Larynx. The larynx is included in the physical examination of the cervical trachea.[14] The examination consists of inspection and palpation, inspection giving attention to possible deformities in the throat or neck region. Palpation serves to detect deformities overlooked during inspection, to determine the firmness of a swelling or a mass, and to detect sensitivity to gentle pressure. Under normal conditions the larynx is palpable and the transition from larynx to trachea is marked by an abrupt decrease in diameter. The cervical part of the trachea can be followed to the thoracic inlet. The thoracic part of the trachea is inaccessible to inspection or palpation.

The throat and neck are inspected with the neck extended slightly forward and upward. In this position the larynx can be palpated by placing one hand—in cats and smaller dogs, just the tips of the thumb and index finger—over the larynx and moving the hand caudally. The dorsal part of the larynx is not often accessible but forceful palpation should be avoided. In larger dogs soft pressure can be used to alter the sound of a laryngeal stridor in order to confirm its source. In small dogs and cats just the touch of palpation has a similar effect.

Trachea. The sensitivity of the tracheal mucosa can be tested by pressing softly on the cervical trachea at three locations: the first few tracheal rings, the midpoint of the cervical trachea, and just before the thoracic inlet. If a cough is elicited the threshold for activation of the cough reflex is lower than normal. A cough may be delayed until the next respiratory cycle and thus there should be a pause before the next palpation. When a tracheal stridor is suspected, brief and light pressure can be applied at several sites along the cervical trachea; a change in the tone of the stridor may indicate the location of the causative obstruction.

Physical examination of the thoracic part of the trachea and the bronchi is included in the physical examination of the thorax.[14] The objectives of the examination of the thorax are to observe the respiratory movements, to detect abnormalities of the thoracic wall, and to detect abnormalities in the structure and function of the tracheobronchial tree, the lung parenchyma, and the pleura.

Respiratory movements. In dogs and cats both the respiratory muscles (chiefly the internal and external intercostal muscles) and the diaphragm play important roles in the respiratory movements which are collectively called costoabdominal respiration.[42] During inspiration both the lateral and the dorsoventral dimensions of the thorax are increased by contraction of the respiratory muscles—pulling the ribs forward, laterally, and ventrally—and by contraction and thereby flattening of the diaphragm, all of which increases the thoracic volume.[42] During expiration the dimensions and the volume of the thorax are decreased by the activity of the transverse thoracic and/or triangular sternal muscles, as well as the abdominal external oblique muscles.[13, 31] The duration of inspiration:expiration is approximately 1 : 1.3.

Examples of changes in the respiratory movements include the predominance of expiration in lung function failure, rapid superficial movements when inspiration is restricted or respiratory movements are painful, and the predominance of inspiration when there is extrathoracic restriction or paralysis of the diaphragm.

Thoracic wall. Abnormalities of the thoracic wall are detected by observing the shape and symmetry of the thorax. In dogs the shape varies greatly, from being deep dorsoventrally in rac-

ing and hunting breeds to barrel-shaped or even dorsoventrally flattened in the English bulldog. The dorsoventrally deep thorax of the cat yields considerably to external laterolateral pressure.[16] The thoracic wall should be palpated superficially at first, with one hand on each side of the thorax, and then deeper to determine whether there are any areas of pain.

Bronchi, lungs, and pleura. The bronchi, lungs, and pleura are examined by auscultation and percussion. Respiratory sounds are audible over the trachea throughout the respiratory cycle and also over the cranial part of the thoracic wall, certainly in smaller animals. These sounds are called bronchial respiratory sounds. But as the stethoscope is moved caudally along the thoracic wall, the expiratory sound in particular becomes softer and sometimes falls away completely. A fairly constant respiratory sound heard during inspiration but dying away during expiration is called the normal respiratory sound.[14] Under pathological conditions auscultation may reveal additional sounds. Musical rhonchi are sounds with a wheezing or peeping character. They occur in patients with obstructive lung and/or bronchial diseases that result in active expiration. Nonmusical rhonchi are short, crackling sounds at the end of inspiration, sometimes continuing to the beginning of expiration. They occur in areas that are not adequately filled with respiratory gases, being infiltrated with fluid.[14]

Percussion of the thorax is intended to determine the borders of the lungs and to obtain an indication of whether the amount of gas in the underlying structures is increased or decreased. It completes the examination of the thorax, but rarely adds to the diagnosis of bronchial diseases.

5.3 Special diagnostic techniques

Special techniques are indispensable for diagnosis of most diseases of the tracheobronchial tree. Although kennel cough can be diagnosed by the association of the history and the clinical signs, chronic coughing and dyspnea require further investigation. Radiographs of the cervical trachea and the thorax can reveal changes in the cartilages of the tracheobronchial tree or the mucosa when the lumen of the trachea and bronchi is altered by the disease process. Radiographs always precede inspection of the lumen of the tracheobronchial tree by bronchoscopy. Bronchoscopy provides visual confirmation of the changes in the tracheobronchial tree, such as anomalies in the shape of the tracheal or bronchial cartilaginous rings or obstruction of the lumen by foreign bodies or masses. When the mucosal lining is reddened by hypervascularization or when mucopurulent material is found covering the mucosa, bronchial and bronchoalveolar lavage with subsequent culture and cytological examination of the material obtained will often lead to the diagnosis or at least an indication of the underlying disease.

Bronchoscopy is ideal for obtaining information about anomalies in the lumen of the tracheobronchial tree, but in dogs and cats it necessitates anesthesia. In bronchopulmonary disease with reduced lung function, culture of the causative organisms may be necessary, while the risk imposed by anesthesia is high. In this situation cricothyroidotomy may be a better diagnostic approach. It simply involves the creation of a small opening in the cricothyroid ligament, through which a catheter can be inserted. The catheter is connected to a flush and suction device, so that saline can be introduced and recovered. After removal of the catheter, applying pressure over the wound for 5 minutes usually seals it sufficiently to prevent the occurrence of subcutaneous emphysema. The procedure requires only disinfection and local anes-

thesia of the area over the cricothyroid junction. The same approach is sometimes used to obtain an emergency surgical airway by placing a small stenting tube in the opening in the cricothyroid ligament.[44]

5.3.1 Diagnostic imaging

Radiography is a very important technique for the diagnosis of tracheal and bronchial diseases. Radiographic imaging of the changes in the pulmonary parenchyma is of additional importance because bronchial diseases may be associated with bronchopneumonia. Radiographic sensitivity can be improved by the use of high-quality equipment—including screens, grids, etc.—and correct positioning, effective restraint, appropriate exposure, and good development procedures.[10]

The lateral view is the most important in radiographic examination of the cervical trachea. The dorsoventral view is wanted occasionally, especially to evaluate dislocation of this part of the trachea. The lateral radiographic examination must be performed with careful positioning of the head and neck in relation to the thorax. Excessive flexion of the occipitoatlantal joint or the neck may cause an undesirable change in the lumen of the trachea, which could be mistakenly interpreted as an abnormality. The thoracic part of the trachea should be examined on radiographs of the thorax. The tracheal images should be surveyed for abnormalities in the diameter of the luminal air column, the continuity of the mucosa, and the contrast of the tracheal rings. In order to detect possible external changes affecting the trachea, the position of the trachea relative to the cervical and thoracic surroundings should be examined. The normal diameter of the trachea is difficult to define, for the diameter decreases slightly from cranial to caudal, but as a rule of thumb the diameter of the trachea at the level the third rib should be approximately three times the width of the rib at the level of the trachea.[45]

Videofluoroscopic examination of the trachea is practically indispensable for the functional evaluation of the collapsing trachea or a partially stenosed trachea. The animal should be conscious and in lateral recumbency while the entire trachea is visualized on the monitor. Playback of the observations in slow motion can reveal functional features missed during the initial examination.[19] When recording equipment is not available, comparing an end inspiratory and end expiratory radiograph will give some indication of the functional consequences of the anomaly.

On radiographs of the normal lungs the bronchial tree is mainly visible in the central area. When the lung parenchyma is adequately inflated only a slight density indicates the lung contours. The bronchi are more apparent in older dogs due to calcification of the bronchial walls. Visible thickening of bronchi, which suggests pathologic changes, gives them the appearance of ring-like structures like doughnuts and parallel lines like tram lines. The presence of air bronchograms indicates that there is consolidation of the lung tissue adjacent to the bronchi. Interstitial fibrosis, often difficult to identify, has been aptly described as a »linear density giving a hazy appearance to the lung field«.[10] Specific radiographic changes in tracheal and bronchial disease are discussed with the diagnostic procedures for the diseases.

5.3.2 Bronchoscopy

The usefulness of bronchoscopy in the diagnosis of diseases of the tracheobronchial tree in the dog and the cat is well recognized. The equipment has been described and bronchoscopic findings in the dog and cat have been illustrated.[2, 41, 48] Although the cost of good-quality equipment is substantial, it is a worth-

while investment for the more specialized small animal practitioner. Videoendoscopy is ideal for teaching, since a group of students can follow the bronchoscopic examination in real time. Bronchoscopic technique differs according to whether a flexible or rigid bronchoscope is used. Rigid systems are less expensive and much more durable. Several sizes of high-quality rigid bronchoscopes can be purchased for the price of just one size of a good-quality flexible bronchoscope. The advantage of the flexible bronchoscope in human medicine is that it makes bronchoscopy possible without anesthesia, but this is in any case never possible in dogs and cats. On the other hand, the thorax is narrower laterally in dogs and cats than in humans, facilitating access to all bronchial divisions using rigid bronchoscopes. The details of bronchoscopy using rigid instruments are therefore described in what follows.

Bronchoscopy using rigid instruments. Bronchoscopy is used to inspect the larynx, the trachea, and the bronchi. Rigid bronchoscopes are available both in small sizes for use in humans and in equipment specifically designed for dogs and cats. For dogs we use bronchoscopes in four sizes: 30 cm × 4 mm, 30 cm × 6 mm, 40 cm × 9 mm, and 50 cm × 9 mm. Each is provided with both a 180°-vision and a 90°-vision telescope, except the longest bronchoscope, which was developed as an esophagoscope for humans. For cats there is a 35 cm × 3.6 mm bronchoscope with a 2.9 mm telescope. Foreign body forceps and biopsy forceps are available for each size of bronchoscope. Long flexible catheters and rigid cannulas for suction, together with a flush and suction system for 10 cc saline solution, are used to obtain material for culture and cytological examination. An adjustable headrest for the animal is a must when rigid endoscopes are used. Illumination can be provided by a simple light source, a flash generator for photography, or a light source for video equipment.

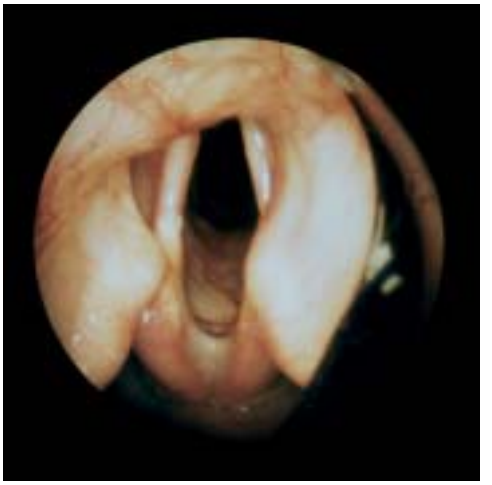
Anesthetic risks. Bronchoscopy differs entirely from rhinoscopy in that the anesthetic risks are often high. The reason for performing bronchoscopy is usually deficient lung function associated with severe bronchitis or obstructive disease. Oxygen administration is therefore necessary before, during, and after bronchoscopy. It is also essential that the ECG, the pulse, and the adequacy of oxygenation be monitored by an experienced anesthesiologist, who is able to anticipate changes in ventilation and perfusion of the lung and changes in heart action.

Visual recognition of the lesions. Diagnosis often depends not only on the visual recognition of lesions in the trachea and bronchi but also on acquisition of material under visual control for cytological examination and culture. Fluid obtained by flushing the main stem bronchi with physiological saline solution at body temperature usually provides adequate material for diagnosis of bronchial diseases. When alveolar washing is required for measurement of specific surfactant, the bronchoscope is wedged into a more peripheral bronchus before flushing with saline solution.[9] This technique is not used routinely because the results are of no consequence in the diagnosis of common bronchopneumonias.

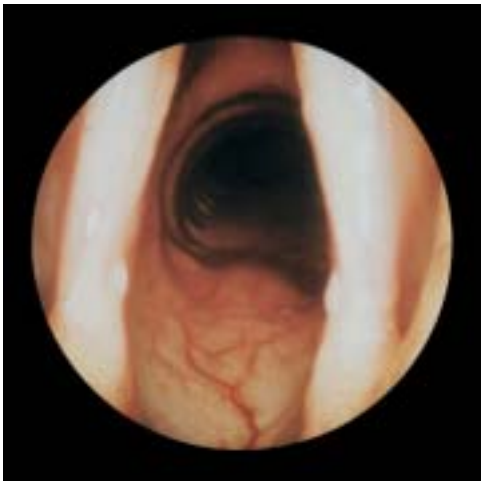
Procedure. Bronchoscopy is always preceded by radiographic examination of the trachea and thorax. In dogs, anesthesia is then induced with medetomidine and propofol in doses according to the risk status of the dog. In cats anesthesia is induced with acepromazine, ketamine, and atropine. The cat is very sensitive to vagal stimulation during bronchoscopy and if not prevented, this causes a massive production of fluid in the bronchi and sometimes death due to acute heart failure. Induction of anesthesia in both dogs and cats is completed with endotracheal intubation and instrumentation of the patient for monitoring. The patient is

then placed in dorsal recumbency with its head on the headrest. When the patient is stabile the endotracheal tube is removed and the bronchoscope, through which oxygen is administered, is introduced with the right hand while the epiglottis is lifted with the laryngoscope in the left hand. After inspection of the larynx the bronchoscope is introduced into the trachea and the telescope is fitted into the bronchoscope. After inspection of the cervical trachea, the headrest is elevated and the bronchoscope, with the telescope, is introduced into the thoracic part of the trachea, bringing the carina into view. The bronchoscope is carefully moved caudally to pass the carina into the right main stem bronchus (in dorsal recumbency, the patient's left and right correspond to the bronchoscopist's left and right). By moving the head on the headrest slightly to the left, the orifices of the right cranial bronchi are inspected. For the inspection of the intermediate bronchus the headrest and head are moved to the right. The bronchi of the caudal lobe are straight ahead, while for deeper parts of the cranial and cardiac bronchi the 90°-angle vision telescope is used. When the inspection of the right bronchi is completed, the bronchoscope is retracted and then introduced past the carina into the left main stem bronchus. The headrest and the head are moved to the right until there is a clear view of the entire left main stem bronchus. The orifices of the lingula (the combined orifice of the left cranial and cardiac bronchi) and the bronchi of the left caudal lobe are inspected. For deeper inspection of the bronchi of the left cranial and left caudal lobes the 90°-angle vision bronchoscope can be used (Figures 5.1 a–q). The bronchoscope is then carefully retracted. Bronchial material is routinely obtained for culture and cytology by flushing and retrieving the fluid, and biopsies are obtained for histological examination if specifically indicated.

Among the most significant findings are anomalies of the trachea, *Filaroides osleri*, foreign bodies, eosinophilic tracheitis and/or bronchitis, and the location of tumors. Bronchoscopic findings are less conclusive in lung fibrosis and other diseases of the lung parenchyma, although extreme expiratory activity will be recognized (Figures 5.2 a, b).



(a) The larynx. The epiglottis is lifted with the tip of the bronchoscope to reveal the cranial aspect of the laryngeal entrance, the aryepiglottic folds, and the vocal folds.



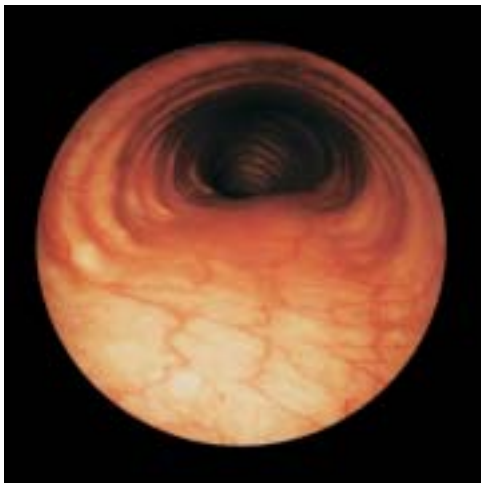
(b) The vocal folds are in the field of vision and the tip of the bronchoscope is just inside the glottis.

Figure 5.1 a–q: Bronchoscopic examination begins with inspection of the larynx and the vocal folds. It then proceeds to the trachea, the carina, the right side of the bronchial tree, and finally the left side of the bronchial tree. Accompanying the pictures of the normal bronchial tree are reference diagrams in which red lines indicate the bronchial walls seen in each picture. The dog is in dorsal recumbency for bronchoscopic examination and hence the right side of the picture corresponds to the dog's right side.

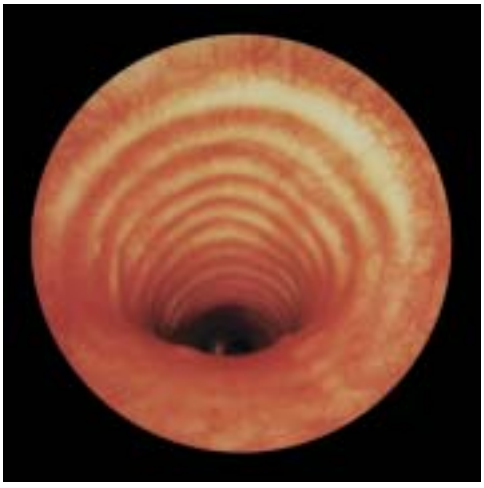
5.4 Congenital diseases of the trachea and the bronchi

5.4.1 Hypoplasia of the trachea

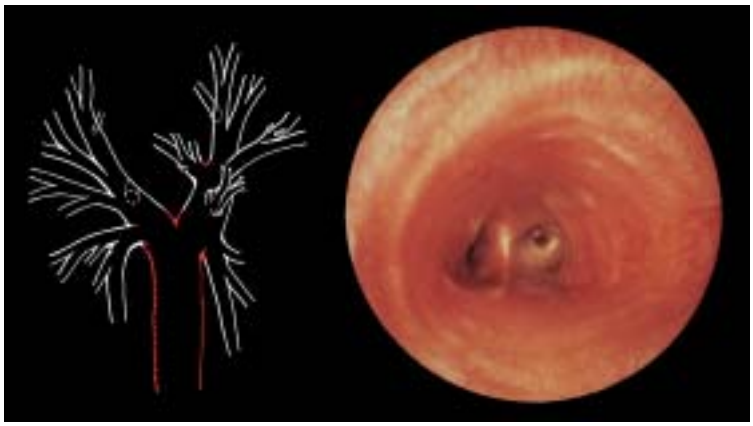
Hypoplasia is incomplete development or underdevelopment of an organ or tissue.[1] Tracheal hypoplasia results in a lumen that is too small for the passage of sufficient air for adequate ventilation. In one form of hypoplasia, the tracheal rings are thicker and smaller than normal for the size of the dog (this defect is uncommon in cats) and their ends meet, leaving no space for a functional tracheal muscle. The muscular and fibrous dorsal ligament is found inside the lumen, ventral to the conjunction of the ends of the tracheal rings. This form of hypoplasia of the trachea occurs frequently in the English bulldog. The diameter of the trachea varies from dog to dog and determines the resulting dyspnea. In some other breeds tracheal hypoplasia results in rings that are very small in diameter but otherwise normal in construction. This form of hypoplasia may also involve only part of the trachea but causes similarly severe dyspnea. This congenital defect is recognized in several



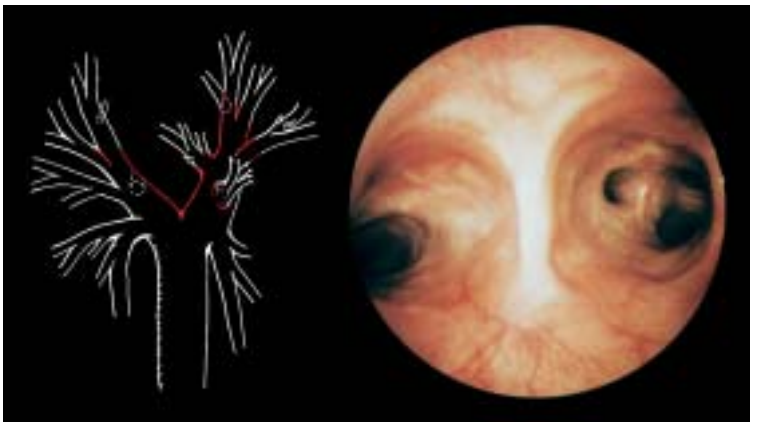
(c) The cranial part of the normal trachea. A network of fine vessels is normally seen in the mucosa. The flat surface of the dorsal wall is the lining of the dorsal ligament of the trachea.



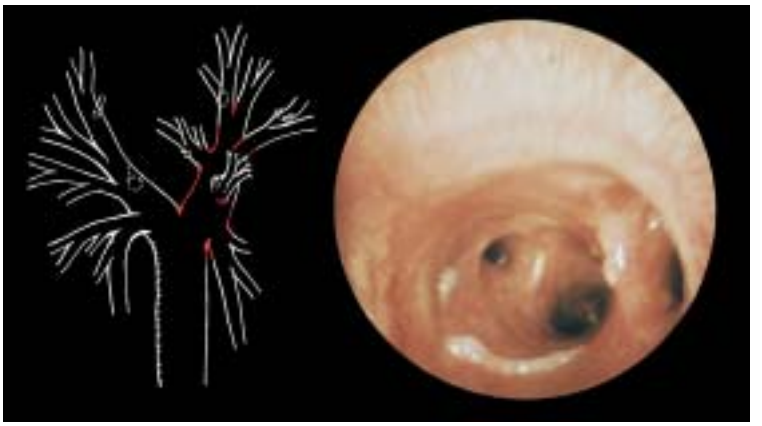
(d) The middle part of the normal trachea. The bronchoscope should be lifted, or the headrest should be raised, to continue the inspection of the thoracic part of the tracheobronchial tree.



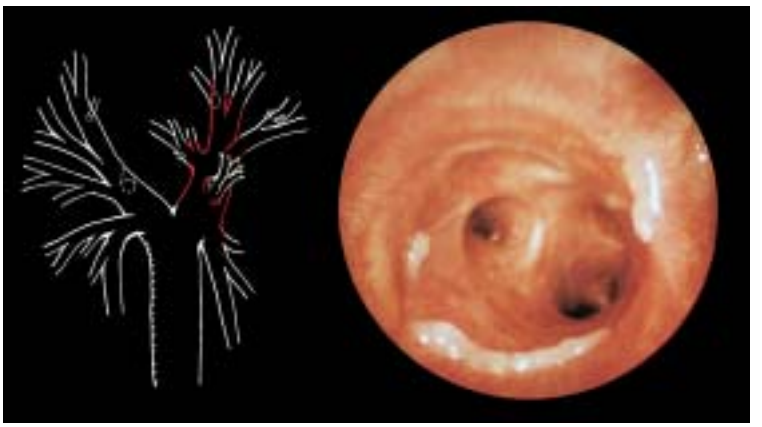
(e) The thoracic part of the trachea, during inspiration. The dorsal ligament is distinct but not prominent. The carina and the orifices of the right and left main stem bronchi can be seen in the distance.



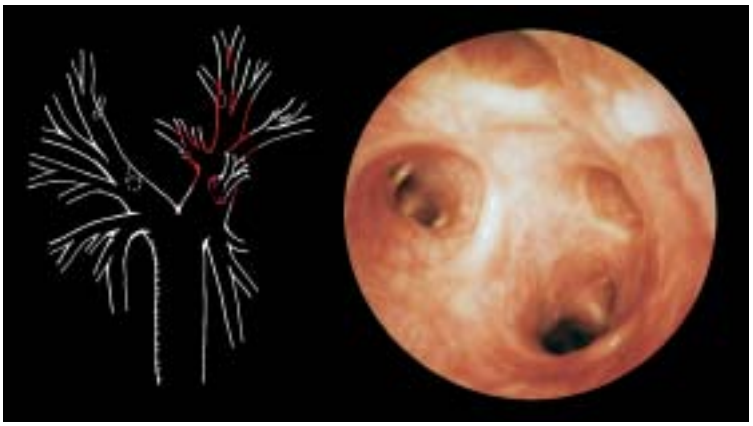
(f) Normal carina. The edge of the carina is sharp. In the right main stem bronchus the orifices of the middle, intermediate, and diaphragmatic bronchi are clearly seen. In the left main stem bronchus the divisions of the left diaphragmatic bronchi are vaguely visible.



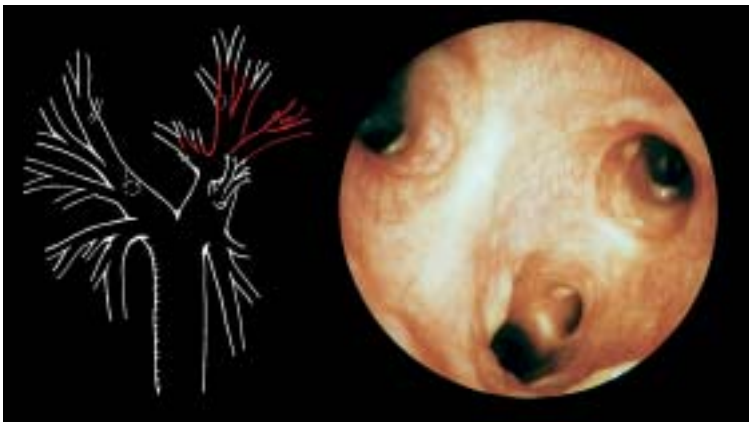
(g) Normal right main stem bronchus. In the left foreground the ventral part of the carina is just visible. On the right the caudal part the right upper bronchial orifice can be seen. A small amount of mucus is present in the right main stem bronchus. In the distance the intermediate bronchus is directed medially and the divisions of the right ventral and dorsal diaphragmatic bronchi are directed caudally.



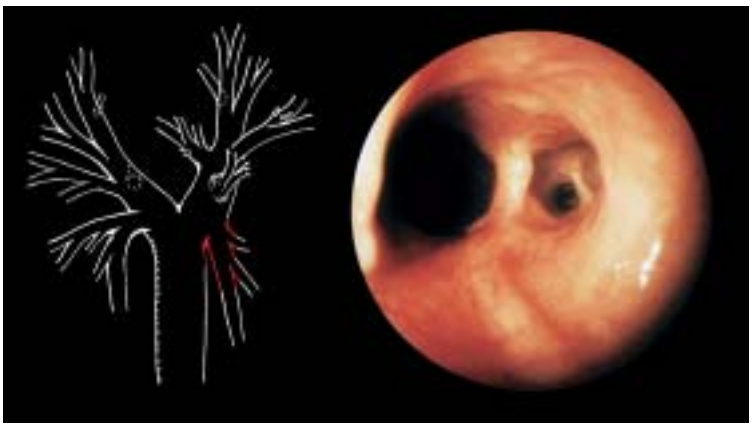
(h) The bronchoscope is situated in the right main bronchus. In the ventral wall the orifice of the middle bronchus is distinct, and beyond it the intermediate and diaphragmatic divisions are seen.



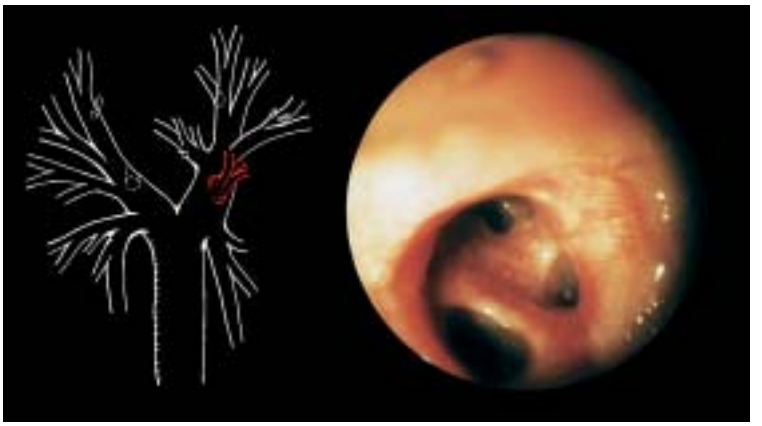
(i) The bronchoscope points almost straight into the lumen of the intermediate bronchus, on the left. The ventral and dorsal divisions of the diaphragmatic bronchus are visible, as well as part of the lateral division.



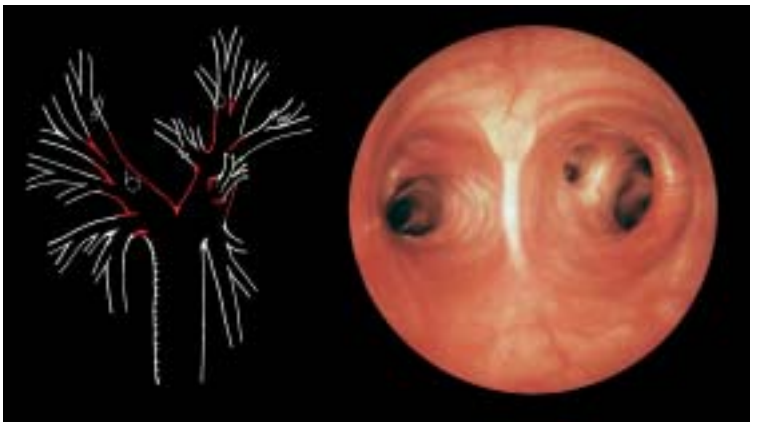
(j) With the bronchoscope in a more lateral position, a deeper view into the diaphragmatic bronchus is obtained. The ventral and dorsal divisions of the diaphragmatic bronchus are also clearly visible.



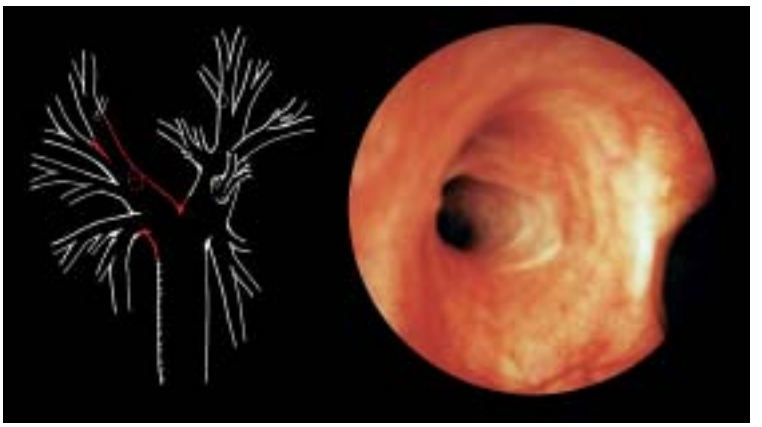
(k) Normal right upper bronchial orifice. The 90°-vision telescope is inserted in a lateral viewing position. Division into the ventral right upper bronchus and dorsal and apical bronchi can be seen.



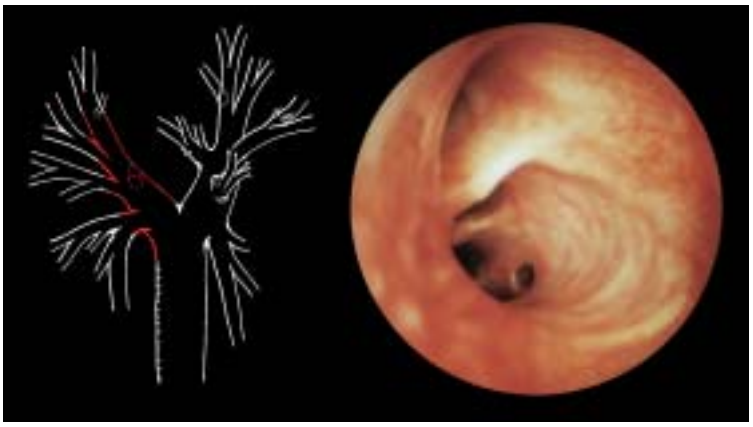
(l) Normal middle (cardial) bronchial orifice. The 90°-vision telescope is now inserted in a ventral viewing position. The triple division of the middle bronchus is well seen here.



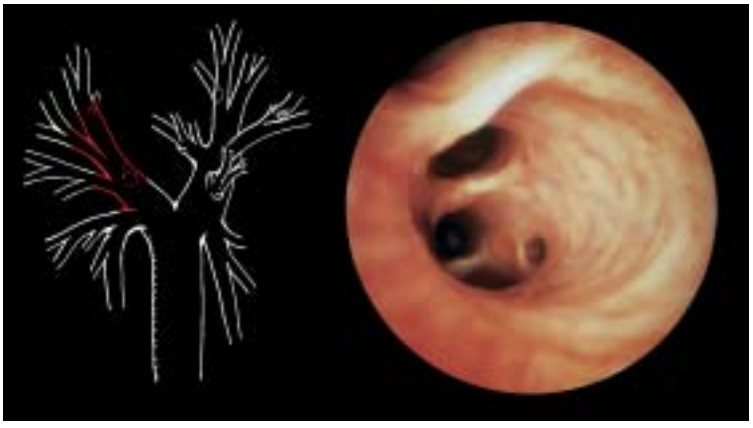
(m) The bronchoscope has been retracted back into the caudal part of the trachea and is ready to be inserted into the left main bronchus.



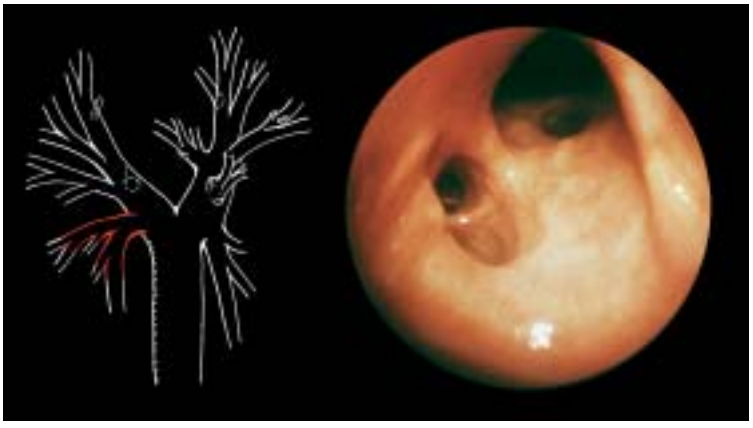
(n) Normal left main bronchus. In the right foreground the left side of the carina is still visible.



(o) The bronchoscope is situated in the left main bronchus. The orifice of the left upper bronchus is seen ventrolaterally and the medial wall and divisions of the normal left diaphragmatic bronchus are in view caudally.



(p) As the bronchoscope passes the orifice of the upper bronchus, the lateral, dorsal, and ventral divisions are seen directly ahead and the apical lower division of the left diaphragmatic bronchus is medial. In most dogs the latter is more ventral.



(q) Normal left upper bronchial orifice. The 90°-vision telescope is inserted in a ventrolateral viewing position. The orifice of the left cardial bronchus and the divisions of the upper division bronchus are seen. (From: Venker-van Haagen AJ. Bronchoscopy of the normal and abnormal canine. J Am Anim Hosp Assoc 1979; 15: 397–410, Figs. 3-19).

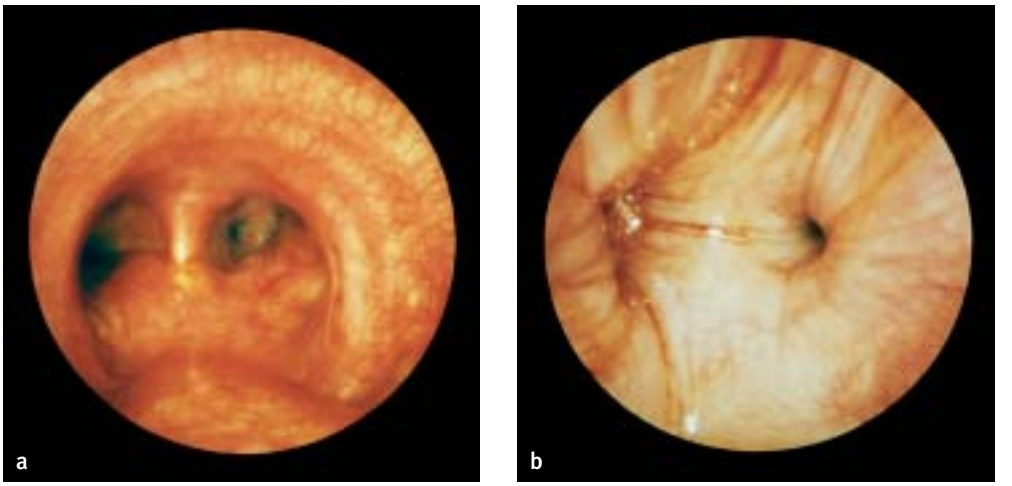


Figure 5.2 a, b: Bronchoscopic views in a dog with lung fibrosis, (a) at the beginning of expiration, and (b) during the last phase of expiration, showing the remarkable intrusion of the dorsal ligament of the trachea and main stem bronchi. The bronchial lumina are almost closed, the mucosa is white and edematous, and there is serosanguinous fluid in the mucosal folds. (From: Venker-van Haagen AJ. Bronchoscopy of the normal and abnormal canine. J Am Anim Hosp Assoc 1979; 15: 397–410, Figs. 33 and 34).

large breeds, including the Shar-Pei, Rottweiler, and Bouvier. Inbreeding was shown to be common among 168 Bouviers in the patient records of the Utrecht University Companion Animal Clinic, in a study published in 1992, and it was concluded that in this population of Bouviers inbreeding increased the risk of tracheal hypoplasia.[47]

History and clinical signs. The history and clinical signs of tracheal hypoplasia are characterized by dyspnea in the young dog. When severe, the dyspnea occurs in the very first months of life and is characterized by rapid respiration, moist respiratory sounds, and frequent coughing. There may be intermittent periods of bronchopneumonia. In less severe cases the signs develop later, around one or two years of age, and are then characterized by dyspnea on exertion and prolonged panting after exercise, but no stridor.

Clinical findings in dogs with a very narrow trachea include rapid respiration, an elevated body temperature, and wet sounds heard during auscultation of the trachea and the lungs. During the examination the dyspnea should be monitored continuously and the dog should be given a rest when needed. Radiographic examination of the cervical trachea and the thorax of the English bulldog reveals thick tracheal rings and a very narrow lumen. The diameter of the trachea at the level of the third rib on the lateral view is less than three times the diameter of the rib, but it is the narrowness of the lumen that is most conspicuous. In such cases bronchoscopy is not needed and should be avoided because of the high risk associated with anesthesia in these dogs.

Clinical findings in cases of less severe narrowing of the tracheal lumen are often unremarkable. Radiographic examination of the trachea often fails to indicate hypoplasia but there is sometimes a slight thickening of the bronchial mucosa, presumably caused by frequent panting. Bronchoscopy reveals a narrow tracheal lumen, redness of the mucosa, and small patches of mucus throughout the length of the trachea (Figure 5.3). Although the normal size of the trachea for each size of dog has not been established, these bronchoscopic findings together with the history of unsatisfactory staying power and prolonged panting after exercise, without stridor, are usually sufficient for the diagnosis.

Therapy. Hypoplasia of the trachea cannot be corrected. In severe cases in very young dogs with almost continuous dyspnea and recurrent pneumonia, euthanasia is an acceptable alternative to any predictably unsuccessful treat-

ment. In less severe cases restriction of exercise, complete avoidance of exercise during warm weather, and immediate cooling when the dog's temperature rises may help to keep the animal alive with little discomfort. But these restrictions apply lifelong.

5.4.2. Collapse of the trachea

This congenital defect affects the shape of the tracheal rings differently: they are wide in the laterolateral direction and flattened in the dorsoventral direction. The space between the ends of the rings is abnormally wide, resulting in a broad tracheal muscle and ligament. The abnormal shape sooner or later causes dyspnea because the trachea collapses during expiration and in severe cases also during inspiration. The defect is commonly diagnosed in the Yorkshire terrier and in the United States also in other popular miniature breeds such as the Chihuahua, Pomeranian, Toy poodle, Shih Tzu, and Lhasa apso.[19] It has also been reported in two cats.[30] In dogs the anomaly may affect not only the trachea but also the bronchi. The disease varies in severity and thus also does the dyspnea.

This congenital defect is commonly called tracheal collapse and while its etiology is unknown, its breed predisposition raises the conjecture that it may be inherited. Histochemical studies of tracheal cartilages from 15 toy-breed dogs with normal tracheas and 6 with collapsed tracheas revealed that the latter had areas of apparent hypocellularity, some areas resembling fibrocartilage or fibrous tissue, and contained less chondroitin sulfate and calcium than did the normal tracheas.[12] The rings may lose their ability to remain firm and over time the dorsal membrane and tracheal muscle apparently become stretched. When the tracheal tube loses its resistance to the negative pressure resulting from the venturi effect, the increased velocity of the expired air may cause collapse of the dorsal membrane, and in very severe cases even collapse of the tracheal rings. In most cases, however, it is only the wide dorsal membrane bridging the abnormally wide gap between the ends of the tracheal rings that is insufficiently rigid. With increasing age, it becomes further weakened and thus more and more drawn into the lumen. At first this occurs only during expiration, for the velocity of air flow is higher during expiration than during inspiration. But as the tracheal lumen decreases and the respiratory distress progresses, collapse also begins to occur during inspiration. This hypothesis provides an explanation for the progression of clinical signs with aging in dogs with this deformity of the tracheal rings.

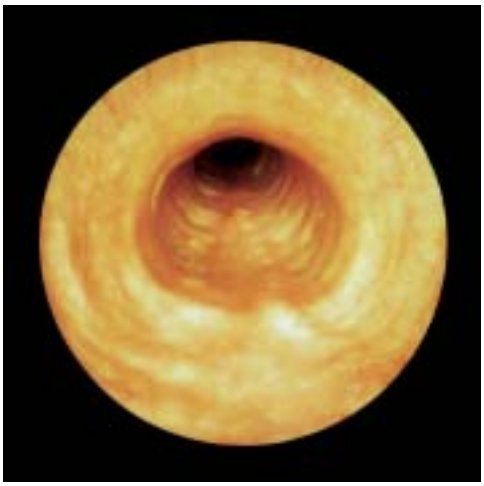


Figure 5.3:
The cranial cervical part of this dog's trachea is of normal size, but with an abrupt narrowing the remainder of the trachea is hypoplastic.

History and clinical signs. The history and clinical signs in patients with tracheal collapse are characterized by a honking cough and progressive dyspnea. If the trachea collapses only during expiration, the stridor is heard only during expiration, but when the trachea also collapses during inspiration, the stridor is heard almost continuously and there is continuous dyspnea. The anxiety of the dog increases its respiratory distress and a very excited or nervous dog with collapse of the trachea may all at once be in a state of severe distress although appearing nearly normal only a few minutes before. Clini-

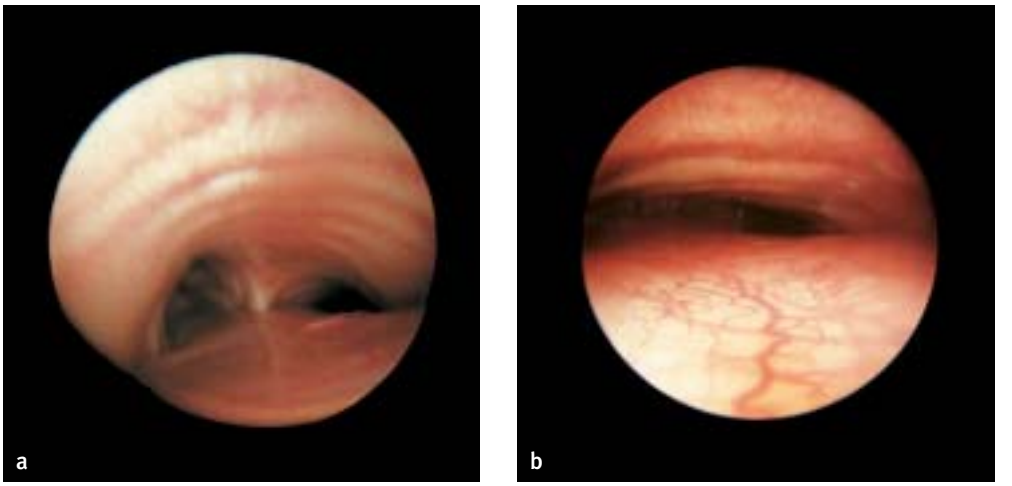


Figure 5.4 a, b: Endoscopic appearance of a collapsed trachea in a dog during inspiration (a) and during expiration (b). In both views note the remarkable width of the dorsal ligament of the trachea.

cal examination is best delayed until the dog ceases coughing. Auscultation of the thorax should be performed before the trachea is touched to elicit a cough or for palpation. After a period of respiratory distress the body temperature may be elevated. Further abnormal findings are not expected, but concurrent diseases may of course be found during the examination.

Imaging of tracheal collapse. Radiographs of both the cervical and thoracic parts of the trachea are made in both the laterolateral and dorsoventral projections of the neck and the thorax. The position of the neck is important for evaluation of the dorsoventral dimension of the trachea. The thoracic and cervical parts are usually not in the same phase of inspiration or expiration, which should be taken into account because the volume of air passing during the respiratory cycle differs for each period. The dynamics of the collapsing trachea can best be observed by videofluoroscopic examination during the respiratory cycle. The dog should be conscious and calm enough to have ceased panting, and should be placed in lateral recumbency while the entire trachea is visualized on the monitor. Playback in slow motion helps to define the locations of the collapse. The videofluoroscopic diagnosis of tracheal collapse is sufficient and bronchoscopy does not add to the conclusions, although it does reveal how the tracheal lumen collapses (Figures 5.4 a, b). Bronchoscopy causes additional irritation of the tracheal mucosa and thus more coughing and dyspnea, and should be avoided unless absolutely necessary.

Therapy of tracheal collapse. Surgical treatment of tracheal collapse has been devised with the aim of constructing a more rigid tube to withstand the negative pressure on the flaccid tracheal wall during ventilation. Dorsal tracheal membrane plication has been used when the trachea has a wide dorsal membrane but firm C-shaped rings. The width of the flaccid membrane is reduced by plication with interrupted horizontal mattress sutures of monofilament nonabsorbable material. This prevents the dorsal membrane from being drawn into the lumen but it also usually results in severe narrowing of the trachea, leading to continuous dyspnea and coughing. The technique has been described in detail.[40]

Providing support to a weak trachea by insertion of an internal stent is widely used. In the long term this has been less successful because of such complications as intermittent coughing and the formation of granulation tissue at the ends of the stent. Once they have been placed,

fixed wire stents cannot be removed and serious complications such as necrosis of the trachea have been reported, although there have also been reports of successful use. A thermal shape-memory titanium-nickel alloy airway stent was placed in two children with tracheobronchomalacia. A 5 mm × 22 mm stent was placed in the left main stem bronchus in one child and a 5 mm × 25 mm stent was placed in the left main stem bronchus of the other (and was removed bronchoscopically 2 years later). This stent can easily be inserted and removed bronchoscopically, has good tissue compatibility, and causes little interference with mucociliary function.[46] The technique has not been tested in tracheal collapse in dogs.

Externally supporting the collapsed trachea by means of partly open rings is the most suitable method for long-term use. Support rings are sutured on the outside of the trachea, 2 to 3 tracheal rings apart to preserve tracheal flexibility. The formation of granulation tissue in the lumen and trauma to the recurrent laryngeal nerves are possible complications. The technique has been described in detail elsewhere.[40]

Comparing results of therapy. In a survey of 100 dogs with tracheal collapse which had been managed by medical or conservative treatment, the signs in 71 cases were reduced to only intermittent dyspnea and coughing. Eleven dogs were considered refractory to medication and underwent tracheal reconstructive surgery but only 5 of these were asymptomatic one year later. The other dogs underwent upper airway surgery for the relief of other obstructive diseases or had coexisting diseases that made them ineligible for surgery. It was concluded that medical and conservative treatment has a prominent place in managing the clinical signs of tracheal collapse in dogs and that surgery should be reserved for those refractory to medical management.[50] A variety of medications can be used. Short-term treatment with glucocorticoids and prolonged treatment with atropine sulfate, diphenoxylate hydrochloride, and phenobarbital are reported to have been successful. Successful treatment using an inhaler delivering a glucocorticoid and a bronchodilator has also been reported.[11] All of these should be combined with advice on improving the general constitution of the dog, weight reduction for obese dogs, and a smoke-free environment.

5.4.3 Segmental tracheal stenosis

Segmental tracheal stenosis is an uncommon congenital disease in dogs and cats and is not known to have a breed predisposition. The abnormal tracheal rings are simply smaller, abnormally shaped, or flattened dorsoventrally or laterally. The number of affected rings varies, as does the location of the abnormal rings along the trachea.

Depending on the severity of the obstruction or discontinuity of the tracheal lumen, the clinical signs are moderate to severe dyspnea and intermittent bouts of coughing. Radiography reveals the location of the abnormal segment in the trachea, videofluoroscopy provides functional information, and bronchoscopy reveals the severity and extent of the anomaly. Surgical resection has the best prognosis if the abnormal segment is located in the cervical trachea. If it is in the thorax, stenting can be considered.

5.4.4 Congenital ciliary dysfunction

Congenitally ciliary dysfunction has been documented in various breeds of dogs.[17] Primary ciliary dyskinesia is a disorder in which ciliary function is ineffective and uncoordinated. As described for congenital ciliary dysfunction in the nose and frontal sinuses (Chapter 2.4.5), cilia are complex structures lining various organs, including the upper and lower respira-

tory tract, auditory tubes, cerebral ventricles, spinal canal, oviducts, and efferent ducts of the testis. The combination of ciliary dysfunction (sinusitis, bronchiectasis) and situs inversus is known as Kartagener's syndrome. Cilia are thin, longitudinal extensions from the free surface of the cell, encased by the cell membrane.[17] During their arc-like effective stroke, they move in an extended planar fashion, contacting, propelling, and then dropping below the overlying mucus layer into the periciliary fluid. The recovery stroke occurs within the periciliary fluid in a side-arm fashion. Coordinated beating of adjacent cilia moves the mucus blanket in the respiratory tract and auditory tubes toward the pharynx. The ultrastructural lesions in the respiratory cilia and sperm flagella in persons with Kartagener's syndrome have been described as defects in the dynein arms, based on electron micrographs of transverse sections of respiratory cilia. The diagnostic significance of these lesions has been contested on the basis of the difficulties inherent in electron microscopy of cilia, the presence of ciliary lesions in clinically normal persons, and the production of ciliary lesions by other disorders such as bacterial or viral infections, smoking, and asthma. Despite the controversy, several distinct ultrastructural abnormalities, when present in a sufficient number of cilia, are believed to be diagnostic for primary ciliary dyskinesia.[17]

Prognosis in mucociliary dysfunction. Mucociliary clearance in the dog's trachea and bronchial tree has been described under functional considerations earlier in this chapter. Disease of the lower airways of dogs with primary dyskinesia varies from mild bronchitis and bronchiolitis to severe bronchopneumonia with bronchiectasis and ventral lung lobe consolidation.[17] The prognosis is reserved. Affected dogs that develop severe recurrent pneumonia eventually die of this, although continuous treatment with broad-spectrum antibiotics can prolong survival. Cultures should be repeated in order to change antibiotics on the basis of sensitivities of the bacteria involved.

5.5 Tracheitis and bronchitis

Tracheitis is a common disease in dogs but unusual in cats. It is characterized by reddening of the tracheal mucosa due to dilatation of the capillaries and infiltration of leukocytes, and sometimes by slightly increased mucus production. The symptoms are dry and harsh coughing that is paroxysmal and often terminates in gagging. The disease may be caused by infection (usually viral), congenital or acquired deformity of the trachea, trauma, foreign body, or allergy, or it may be idiopathic. Tracheitis is usually accompanied by laryngitis, especially in infectious disease, and may develop into tracheobronchitis. In dogs infectious laryngitis and tracheitis is known as kennel cough, mainly caused by canine adenovirus 2 and canine parainfluenza virus. In cats infectious upper respiratory disease caused by feline herpesvirus 1 and calicivirus also produces laryngitis but tracheitis is rarely diagnosed. The symptoms of chronic tracheitis are usually accompanied by those of chronic bronchitis and are characterized by intermittent productive coughing and, in longstanding illness, by decreased endurance.

Therapy for tracheitis should be based on the underlying cause(s), which should therefore be identified. The medical history is important in doing this correctly. Questions should be concerned with the onset and the duration of the clinical signs, recent contact with a kennel, exposure to caustic agents such as smoke in the house, and, in dogs, the use of a choke collar or chain during training sessions, and recent surgery (possible irritation of the tracheal mucosa by endotracheal intubation).

5.5.1 Infectious tracheobronchitis in dogs

Viral tracheobronchitis. The most important viruses causing tracheobronchitis in dogs are canine adenovirus type 2 and canine parainfluenza virus. These viruses may damage the respiratory epithelium to such an extent that bacteria and mycoplasmas are able to cause secondary disease. Other viral infections are caused by canine adenovirus types 1, 2, and 3, and canine herpesvirus.[32] Canine adenovirus type 2 and canine parainfluenza virus are transmitted by aerosol droplets. Canine adenovirus type 2 is moderately resistant and can survive for months in the environment, while canine parainfluenza virus is relatively labile, but quaternary ammonium disinfectants are effective against both.[32]

The history and clinical signs of viral tracheitis without bronchitis differ from those of tracheobronchitis. Viral tracheitis is characterized by a history of contact with coughing dogs or a stay in a kennel or visit to another place where dogs are brought together and where viruses may survive. The harsh, dry cough develops 3 to 5 days after initial exposure.[19] In most cases the infection is self-limiting in about 3 weeks. Palpation of the larynx and trachea elicits a cough or paroxysmal coughing. In mild infections there is no fever and the dog is otherwise healthy. The coughing may continue during the night and may also be stimulated by barking and by drinking. It may even cause the dog to avoid barking and drinking.

In viral tracheobronchitis a productive cough is to be expected, for the production of mucus is more abundant in the bronchial tree than in the trachea. The history is usually suggestive of viral tracheitis at the onset of the clinical signs, but it is the change from a dry cough to a productive cough that is the alarming sign. The dog can still appear to be otherwise healthy, but the presence of a productive cough should direct attention to the possible development of bronchopneumonia, for *Bordetella bronchiseptica* infection is commonly associated with tracheobronchitis. This condition is also self-limiting, but its natural course may require 3 months.[19] The diagnosis can rest on the history and clinical signs alone, unless they include evidence of complications, such as malaise and fever. Then further clinical examination, laboratory tests, and radiographs of the thorax are needed to estimate the seriousness of the disease. If no complications develop but the disease is not resolved after 3 months of care as described below, radiographic examination and bronchoscopy with bronchial lavage should be performed. An underlying disease such as tracheal hypoplasia or allergic tracheobronchitis may be revealed as the reason for the delay in spontaneous resolution of the tracheobronchitis.

Treatment of viral tracheitis in its usual mild form of laryngotracheitis without fever is symptomatic. House or kennel rest with only short walks and avoidance of excitement is important so long as the dog is coughing. Daily monitoring of body temperature is helpful in following the progress of the disease. The coughing will diminish if the dog is kept calm. Leading the dog on a leash will provoke coughing and should be avoided. When drinking provokes coughing, dogs tend to avoid the water pan. However, a drink of water activates the glands that moisten the laryngeal mucosa, which diminishes the irritation, and thus water should be given orally (20 cc for a dog of 15 kg) several times daily according the frequency of the cough. Excessive coughing may be treated with sedatives, especially during the night. Phenobarbital is satisfactory in a dose of 2 mg/kg once or twice daily, depending on the effect. Most dogs recover from kennel cough without complications, but when it becomes a serious problem in a kennel, intranasal vaccines are recommended for puppies as young as 2 to 4 weeks of age. Intranasal vaccines may contain attenuated canine parainfluenza virus together with attenuated *Bordetella bronchiseptica*.

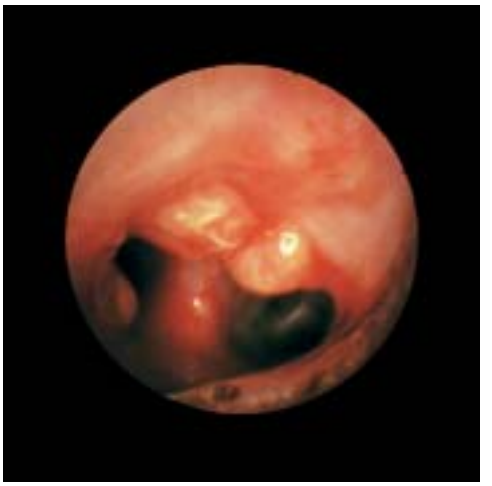


Figure 5.5: Larvae of Filaroides osleri are visible through the mucosa covering the nodules, which are mainly located around the carina, in the caudal part of the trachea, and the cranial part of the main stem bronchi.

Attenuated canine adenovirus 2 vaccines are also available (Chapter 4.5). Treatment of tracheobronchitis without fever can be similar to that of uncomplicated viral tracheitis.

Antibiotic treatment with broad-spectrum antibiotics is indicated when fever, malaise, or systemic disease complicate the infection. Further therapy should be guided by the results of special diagnostic procedures. Antitussives should not be used if the cough is predominately productive, since coughing removes obstructive mucus from the bronchial tree. Water should be given orally (20 cc for a dog of 15 kg) several times daily, to prevent desiccation of bronchial mucus or mucopurulent material.

Bacterial tracheobronchitis. Primary tracheobronchitis is not common in dogs, although pure cultures of *Bordetella bronchiseptica* have been obtained in the absence of respiratory viruses.[53] Positive cultures have been obtained from the trachea and bronchi of dogs with viral tracheobronchitis, eosinophilic bronchitis, tracheal hypoplasia, ciliary dyskinesia, foreign bodies, and bronchopneumonia. The organisms identified have included *Bordetella bronchiseptica, Escherichia coli, Pseudomonas, Klebsiella, Streptococcus, Staphylococcus,* and sometimes mycoplasmas.[19, 26]

Fungal tracheobronchitis. Fungal tracheobronchitis is uncommon in dogs. When nasal aspergillosis was first recognized, we made several attempts to culture *Aspergillus* from the tracheobronchial tree of affected dogs but never obtained a positive culture. Aspergillomas have not been found in the lungs in dogs, as they are in humans, but the possibility of their occurrence should not be disregarded, in view of the wider use of immunosuppressive drugs in dogs. As in humans, these drugs may become a cause of aspergillosis in the lungs of dogs being treated for neoplasms.

Parasitic tracheobronchitis. In young dogs with increasing coughing and dyspnea, the nematode parasite *Filaroides osleri*—also called *Oslerus osleri*—may be found during bronchoscopic examination of the trachea and bronchi. The infection is manifest as multiple pink to cream-colored submucosal nodules in the caudal part of the trachea and the cranial parts of the main stem bronchi. The identifying characteristic of these nodules is the presence of the short, white, curved parasite larvae visible through the covering mucosa (Figure 5.5). The number and size of the nodules determine the extent of the airway obstruction which they cause and hence the severity of the clinical signs. In addition, tracheitis is caused by the irritating effect of the nodules and sometimes free parasites.

Pups may become infected via the saliva of the dam. In the pup, ingested larvae pass from the gastrointestinal tract via the lymphatic and portal systems to the heart. They enter the pulmonary arteries and are transported to the lungs, from which they enter the primary bronchi and trachea. In the wall of the trachea, adult worms produce larvae, leading to the formation of nodules. The clinical signs become manifest from around the age of 6 months.[8]

The diagnosis of the infection is usually made by bronchoscopic examination. The nodules are so distinctive that biopsies are not

needed. Radiographs do not always reveal the nodules. Fecal examination is generally considered to be relatively insensitive and a negative result is not conclusive.[8]

Treatment of *Filaroides osleri* infection with levamisole is effective. When the dog is very dyspneic at the initial examination it may be helpful to remove the most obstructive nodules via the bronchoscope before starting the levamisole treatment. Most of the parasites in the nodules are dead after 14 days of oral treatment using a daily dose of 7 mg/kg body weight, but the treatment is usually continued for 6 weeks. The nodules shrink, coughing stops, and respiration becomes easier or normal. Six weeks after the beginning of treatment bronchoscopy still reveals many nodules, but they are smaller in size and lighter in color. If the clinical signs are not completely resolved, removal of the shrunken nodules via the bronchoscope may be helpful.

To prevent further infection of the pups the dams should be examined for the presence of nodules in the tracheal bronchial tree, but not all breeders are willing to submit their dogs to bronchoscopic examination.

5.5.2 Infectious tracheobronchitis in cats

Infectious tracheobronchitis, whether viral, bacterial, fungal, or parasitic, is uncommon in cats. Even the common feline herpesvirus type 1 and the feline calicivirus do not cause tracheitis or tracheobronchitis, at least not on clinical evidence.

5.5.3 Noninfectious tracheobronchitis

Eosinophilic tracheobronchitis in dogs. Eosinophilic tracheobronchitis in dogs is characterized by the presence of eosinophils among the inflammatory cells in material obtained by bronchial lavage, mucosal brushing, or mucosal biopsy. The history and clinical signs are those of chronic, frequent coughing that is refractory to antibiotic therapy. There is often a history of presumptive kennel cough at the outset, but the coughing will have continued for months, unaffected by repeated antibiotic therapy. During clinical examination coughing is elicited by palpation of the trachea. The cough may be dry and paroxysmal, but may also be productive, i.e., even though it does not always produce visible mucus, the cough sounds wet and the dog swallows after coughing. Further clinical examination usually reveals no other abnormalities, although auscultation of the thorax may reveal musical rhonchi.

Radiography may reveal no specific abnormalities in either the cervical or thoracic trachea, but there may be thickening of the walls of the bronchi. Bronchoscopic examination is necessary for further diagnosis. In most cases the tracheal mucosa is hyperemic and swollen, and there are small patches of mucus. The mucosa of the main stem bronchi is similar, sometimes with more abundant mucus. Material from the bronchial mucosa is obtained by bronchial lavage or brushing, and in rare cases by biopsy of elevations in the mucosa (Figures 5.6 a, b). Cytological examination of this material reveals eosinophils, sometimes entire fields of them, along with other inflammatory cells and ciliated bronchial epithelial cells. Histological examination of biopsies reveals a similar cell pattern. Culture of this material usually obtains *Pasteurella* and *Streptococcus*, sensitive to a wide range of antibiotics.

Therapy begins with a broad-spectrum antibiotic, which is continued for 10 days, and prednisolone is added as soon as the cytology is known. Prednisolone is started in a dose of 1 mg/kg body weight once daily for the first 3 days. Then the dose is then reduced to 1 mg/kg body weight on alternate days for 14 days and then to 0.5 mg/kg body weight on alternate days, lifelong. Every time prednisolone is stopped, the coughing will recur and

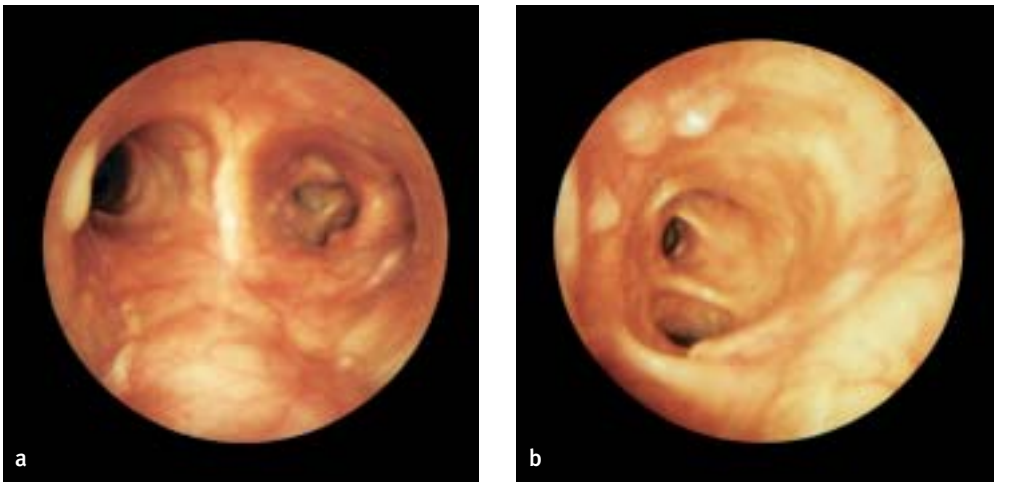


Figure 5.6 a, b: Eosinophilic tracheobronchitis in dogs is characterized by hyperemia and swelling of the mucosa, sometimes with distinct elevations (a). The elevations are easily seen in this right main stem bronchus (b). Biopsies revealed many eosinophils.

the inflammatory changes in the bronchial mucosa will increase. The prescription of lifelong prednisolone therapy necessitates that the diagnosis be correct. When all of the clinical signs strongly suggest eosinophilic tracheobronchitis but eosinophils are not found in the bronchial cytology, the possibility of previous administration of a glucocorticoid should be reexamined, even though this is checked routinely before bronchoscopy is performed. Glucocorticoids can suppress the appearance of eosinophils for up to 5 weeks after the last dose

Eosinophilic tracheobronchitis in cats. As in dogs, eosinophilic tracheobronchitis in cats is characterized by the finding of eosinophils among inflammatory cells in bronchial lavage fluid or brush material. The history and clinical signs differ from those in dogs, for cats usually have recurrent attacks of dyspnea, often sudden in onset and paroxysmal. In the intervals between attacks, the cat eats normally and may be playful. The syndrome is similar to asthma in humans in that the dyspnea is expiratory. Coughing may also be observed. In severe cases with almost continual dyspnea the cat avoids exertion. Lung emphysema may develop as a result of recurrent narrowing of the bronchi and bronchioles.

Clinical examination during an attack of dyspnea reveals tachypnea with accentuated expiration. The cat remains very quiet, shows that it dislikes being handled, and prefers sternal recumbency or a sitting position. The mucous membranes may be normal or slightly cyanotic. Palpation of the trachea may evoke a dry cough. If there is emphysema, auscultation of the lung reveals wheezing during expiration over a large part of the lung field, and if there is no emphysema, bronchial sounds may be heard over a larger than normal area. However, the diagnosis of emphysema cannot be made on the basis of clinical signs alone.

Radiographs may reveal increased radiolucency of the lung field, an increase in the size of the thorax, and extension of the lumbodiaphragmatic recesses. The bronchial vascular markings are unusually well visualized. The bronchial walls are thickened and many end-on views of bronchi appear as annular shadows. The diagnosis of vesicular emphysema or hyperinflation is strongly supported when the lung field remains hyperlucent and large on radiographs made during the expiratory phase.[26] It may prove useful to study changes in the subepithelial thickness of the bronchi by means of ultrasonography and to use the bronchial subepithelial thickness as a measure of the progress of the disease or the efficacy of the therapy.[54]

A characteristic bronchoscopic finding is severe coughing during the introduction of the bronchoscope. The main stem bronchi appear narrower than normal and the mucosa is covered with a white layer of mucus and cell detritus. Cytological examination of the bronchial lavage fluid reveals many eosinophils, sometimes in solid fields. It should be ascertained that the cat has not been treated with a glucocorticoid for 5 to 6 weeks, for glucocorticoids can suppress the eosinophils and thereby conceal the diagnosis. Bacterial culture is usually positive, predominantly *Pasteurella*, and sensitive to a wide range of antibiotics.

Therapy consists of a broad-spectrum antibiotic for 10 days with the addition of prednisolone when the cytology (eosinophils) becomes known. Prednisolone is started in a dose of 1 mg/kg body weight once daily for the first 3 days. Then the dose is reduced to 1 mg/kg body weight on alternate days for 14 days and then to 0.5 mg/kg body weight on alternate days, lifelong. Every time prednisolone is stopped, the coughing will recur and the inflammatory changes in the bronchial mucosa will increase. The prescription of lifelong prednisolone therapy necessitates that the diagnosis be correct. Beta$_2$-selective sympathomimetic agents such as terbutaline are effective as bronchodilators in cats and can be given if the cat is dyspneic before diagnostic bronchoscopy has been performed. Terbutaline can be given orally, begins to be effective in about 30 minutes, and acts for 12 hours. It does not suppress the inflammatory changes in the bronchi and therefore does not affect the cytological findings, but it also cannot be used in place of a glucocorticoid for long-term therapy.

5.5.4 Bronchiectasis

Bronchiectasis is characterized by irreversible dilation of the medium-sized bronchi. It may be focal or it may affect a large part of the bronchial tree. The clearance of material from the bronchi is seriously hampered by the severe structural alterations. The accumulation of secretions damages the epithelial surface, further delaying clearance of both mucus and bacteria and resulting in recurrent bronchitis or bronchopneumonia.[34] The disorder usually occurs as a sequel to chronic bronchitis in middle-aged dogs but is also seen in young dogs with ciliary dyskinesia. The medical history is characterized by chronic coughing and episodes of bronchopneumonia. The response to antibiotic therapy is satisfactory initially, but the recurring cough and periods of fever eventually necessitate special diagnostic techniques. In addition to the recurring productive cough, physical examination reveals hypersensitivity of the trachea and auscultation of the lung fields reveals moist crackles and expiratory wheezing.

Radiographs of the thorax show irregularly dilated bronchi, often with infiltrates characteristic of bronchopneumonia. Bronchoscopic findings include abundant mucopurulent material throughout the bronchial tree and irregularly shaped bronchi. Culture of bronchial lavage fluid will determine the predominant bacterial growth, which can guide the choice of antibiotic therapy. The prognosis is reserved, especially when the cause of the disease is ciliary dyskinesia. But since in all other cases the changes in the bronchi are irreversible, antibiotic therapy should be repeated or given almost continuously. If the bronchiectasis is focal, surgical removal of the affected lung lobe is indicated.

5.5.5 Prolapse of the dorsal ligament of trachea and main stem bronchi

This occurs in dogs with progressive lung failure associated with long-term bronchitis. They have a chronic cough and gradually increasing expiratory dyspnea. Prolapse of the dorsal ligament occurs during expiration and leads to a progres-

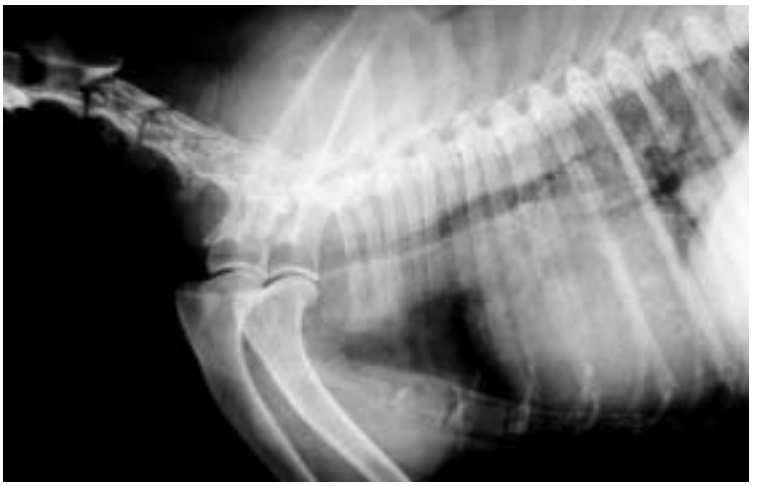


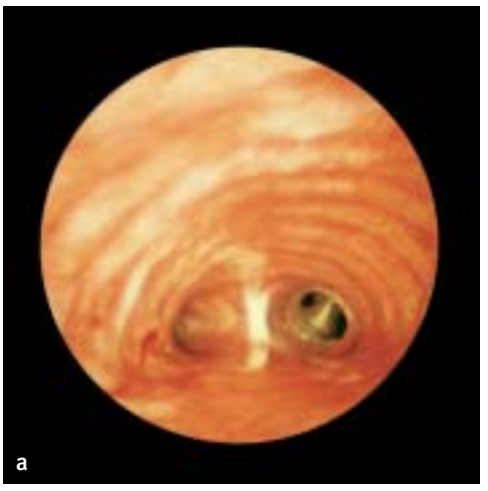
Figure 5.7: Prolapse of the dorsal ligament of the trachea and main bronchi can occur in longstanding bronchitis and progressive lung failure. Latero-lateral radiographs of the trachea show the prolapse during expiration.

sively louder and eventually almost continuous dry expiratory cough during exercise.

The history always reveals that there has been frequent coughing for many years, with progressively diminishing exercise tolerance. The prominent clinical sign in these elderly dogs is a hard, dry cough during expiration. The frequency of the cough subsides when the dog calms down. Physical examination reveals tracheal hypersensitivity to palpation, as in tracheitis. Increased bronchial sounds are heard during auscultation of the lungs and crackles are heard over some areas. The dog may be overweight and have a heart murmur, which is not unusual in older dogs.

Diagnostic studies begin with radiographs of the tracheobronchial tree, the lungs, and the heart. On radiographs made during inspiration the bronchial structures are more prominent, the density of the lung parenchyma is increased, and the trachea and bronchi are filled with air. During expiration the radiographic density of all lung parenchyma is accentuated and the prolapsed dorsal ligament of the trachea can be seen to almost touch the opposite wall of the trachea (Figure 5.7). The bronchoscopic view during inspiration reveals normal tracheal rings, with irregular redness of the mucosa indicative of tracheitis. However, during each expiration the dorsal ligament of the main stem bronchi and trachea is drawn progressively inward, obliterating the lumen (Figures 5.8 a, b). There is no collapse of the tracheal rings. The cytological findings in bronchial lavage fluid are consistent with chronic bronchitis, sometimes with eosinophils. The culture is negative or there are bacteria which are sensitive to a wide range of antibiotics.

Therapy is primarily directed at diminishing the cough. Since the loud tracheal cough dominates the clinical signs, it is worthwhile to consider its cause. Chronic bronchitis is thought to have led to progressive lung failure and loss of elasticity of the lung tissue. This can be expected to result in expiratory dyspnea and



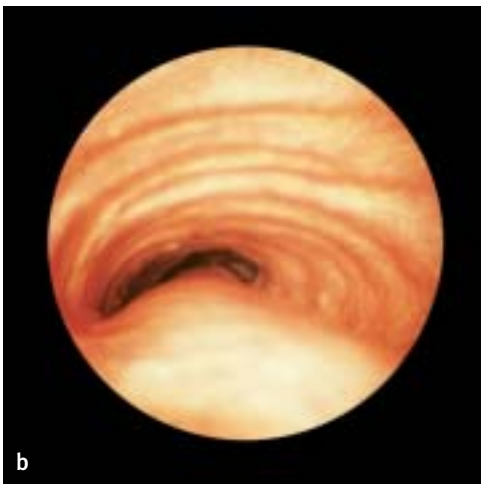


Figure 5.8 a, b: Endoscopic view of the trachea and main stem bronchi of the dog in Figure 5.7. During inspiration the trachea and main bronchi are open (a) but during expiration the dorsal ligament protrudes into the lumen (b).

increased expiratory effort. The increased velocity of the expired air may be irritating to the already hypersensitive tracheobronchial mucosa, resulting in coughing. In addition, as the velocity of the expired air increases, the pressure decreases, which in effect causes traction on the wall of the trachea and bronchi. The cartilaginous rings are normal and are not deformed but the soft tissue of the dorsal ligament thickens and begins to protrude into the lumen. The velocity of the expired air is increased and its pressure lowered by passing through the narrowed lumen (venturi effect), placing traction on the dorsal ligament which eventually leads to its prolapse (Figure 5.9). The near contact of the prolapsed dorsal ligament with the opposite tracheal wall causes a honking sound during expiration similar to that made by a dog with a collapsed trachea. As in tracheal collapse, treatment is directed at preventing excessive excitement and exertion, with the aid of mild sedation with phenobarbital. The bronchial disease may still respond to treatment with a glucocorticoid but the changes in the lung parenchyma will most likely be found to be refractory. Treatment with phenobarbital and the glucocorticoid should therefore be continued lifelong and the long-term prognosis is guarded.

5.5.6 Foreign bodies in the tracheobronchial tree in dogs

A foreign body in the tracheobronchial tree is a common problem among dogs of all ages. Most of the foreign bodies are of plant origin and these are the most dangerous because they are difficult to detect by radiography, which is far more readily available in general practice then is bronchoscopy. The history and clinical signs are very important, and especially the history should be pursued in detail when coughing of acute onset is one of the clinical signs. If the dog lives in close contact with the owner, questions should be asked about the dog's habit of playing with stones, bones, or sticks, or chewing on rags or toys. A history of the sudden onset of coughing and restlessness should immediately be associated with the likelihood

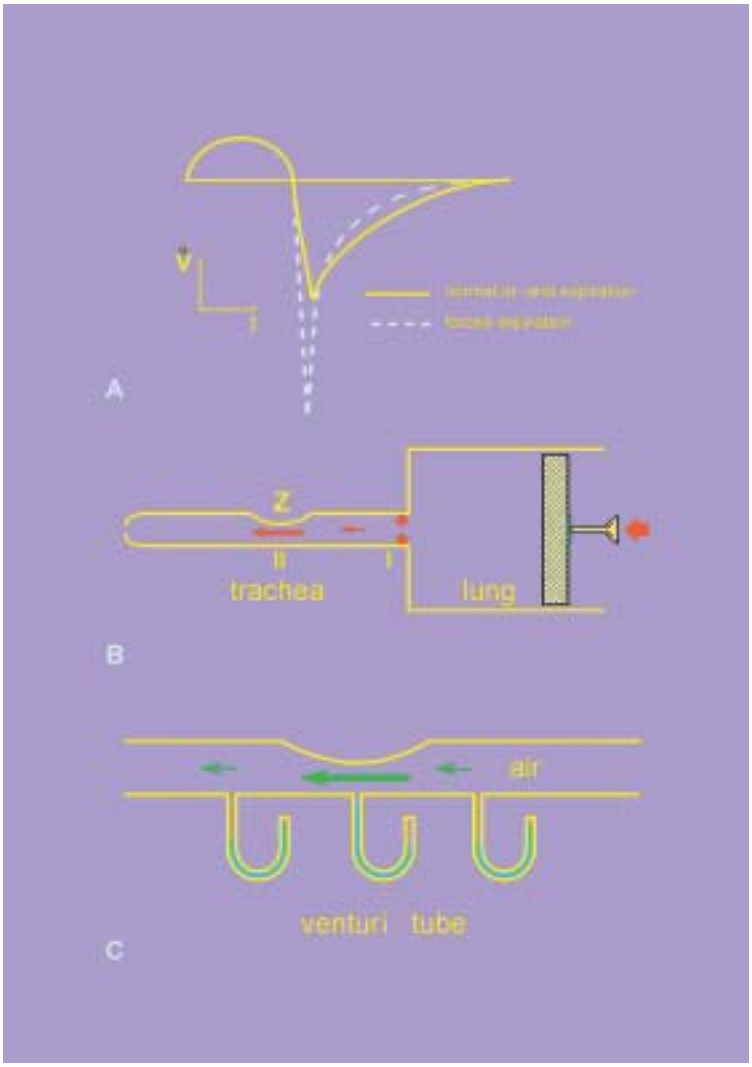


Figure 5.9:
Functional mechanisms thought to result in protrusion of the dorsal ligament during expiration following loss of elasticity of the lung parenchyma. (A) Active expiration causes shortening of the expiratory phase, the air being expelled at a higher velocity (normal = yellow line, lung failure = dotted blue line). (B) The increased velocity of expired air may lead to thickening of the bronchial and tracheal mucosa, further raising the velocity of the air passing through them. (C) The elevated velocity of air expelled through the bronchi and trachea increases the traction on their walls, as demonstrated by the venturi tube. The increase in velocity of air as it passes through the narrow part of the venturi tube causes the fluid in the corresponding manometer to rise, indicating a local lowering of pressure.

of a foreign body in the tracheobronchial tree. If radiographs do not reveal foreign material in the trachea or bronchi, bronchoscopy should still be performed. When the history is less suggestive, the dog is often referred because of a longstanding productive cough and increasing malaise. The examination then reveals signs of chronic tracheobronchitis, including tracheal hypersensitivity to palpation and nonmusical rhonchi auscultated over one or more areas of the lungs. Radiographs of the trachea may reveal focal densities, but may also indicate chronic bronchitis, sometimes bronchopneumonia and consolidation of a lung segment. If a foreign body is distinctly demonstrable in the trachea or bronchi, bronchoscopy is clearly indicated to remove it. Even if the radiographic evidence is less conclusive, bronchoscopy should still be performed, because it will provide more specific information than the radiographs.

There are several techniques for removal of foreign bodies from the trachea. A stone or marble in the trachea of a dog will cause reflex coughing when it begins to obstruct the airway at its narrowest part, just cranial to the bifurcation. The dog will then cough with its neck bent downward and will prefer to stand on all four legs rather than sitting or lying down, to prevent the stone or marble from blocking the main stem bronchi. The widest part of the airway is within the cricoid cartilage, just cranial to the entrance to the trachea. A rolling stone or marble should be tilted into that location if possible, when the dog is anesthetized. Hence the dog should be placed on a sloping table with its head downward and should remain in this position during bronchoscopy. If, after being anesthetized, a small dog is held vertically with its head downward, it is sometimes possible to dislodge a round foreign body lying within the cricoid cartilage by an abrupt and forceful laterolateral compression of the thorax.

A round foreign body with a smooth surface can usually be removed from the cranial trachea. Two long instruments that can be introduced through the larynx into the trachea, such as a rigid bronchoscope and a large curette with a long handle, are helpful to catch the foreign body and retrieve it through the glottis. Flexible wire nets have been developed for this purpose, but they may be found disappointing for use in the trachea. If in maneuvering the stone or marble it accidentally ends up near the carina, it should not be allowed to remain there. It is even preferable to move the foreign body into one of the main stem bronchi, so that one lung remains free for ventilation, rather than to risk blockage of most of both orifices to the main stem bronchi. Repositioning the dog and increasing the slope of the table may be helpful before trying again. Abrupt and forceful laterolateral compression of the thorax will dislodge the foreign body into the trachea, and it may then roll toward the larynx. If all efforts to remove it fail but the foreign body is in the cranial part of the trachea, a tracheostomy can be performed in order to insert an endotracheal tube to aid ventilation and also prevent the foreign body from moving caudally. The foreign body can then be removed via cranial extension of the tracheostoma. One of the most important organizational considerations for the endoscopist and the anesthetist is to always prepare for a long-term procedure when bronchoscopy is undertaken for removal of such foreign bodies.

Plant material can usually be removed from the trachea and larger bronchi under visual guidance by use of one of the foreign body forceps that fit over the telescope in the rigid systems. When plant material remains in the tracheobronchial lumen for a long period it becomes buried in nontransparent mucopurulent exudate and only becomes visible after the exudate has been aspirated. Broad-spectrum antibiotic treatment is given for 2 to 3 weeks after removal of the foreign body. It may be useful to culture the exudate in the event that the response to the initial antibiotic treatment is unsatisfactory.

Especially when the foreign material is of plant origin, it may happen that it can be located but not recovered from a deep bronchus. The bronchus involved should be identified and the affected segment of lung should be removed surgically.

5.5.7 Foreign bodies in the tracheobronchial tree in cats

Foreign bodies in the tracheobronchial tree in cats are usually detected because of the long-standing coughing that they cause. Coughing is such a rare clinical sign in cats that some owners may remember the date and the circumstances under which the cough was first observed. Most of the foreign bodies in the tracheobronchial tree are of plant origin, but some amazing materials are also found, such as paper and small beads. Fish bones occur much less frequently than might be expected. The clinical signs of a foreign body in cats are coughing and dyspnea. The general physical findings vary from coughing during palpation of the trachea to signs of bronchopneumonia and malaise. Radiographs of the trachea can reveal the foreign body as such if it is radiopaque. Radiolucent foreign material may be visible as a density in the lumen of the trachea. Foreign bodies in the bronchial tree have the same characteristics as those in the trachea, but may be surrounded by lung infiltrates.

When a foreign body is presumed to be present, bronchoscopy is indicated. A complete set of instruments for bronchoscopy in cats consists of a rigid tube through which the instruments can be passed and oxygen administered and foreign body forceps with a hollow stem through which the telescope can be passed, enabling removal of the foreign body under visual control. Material from the tracheobronchial tree can be collected for culture. Broad-spectrum antibiotic treatment is given for a week or more after removal of the foreign body. The treatment can be corrected when the result of the bacterial culture is known.

In cats also, it may happen that plant material can be located but not recovered from a deep bronchus. The bronchus involved should be identified and the affected lung segment should be removed surgically.

5.5.8 Tracheitis caused by aspiration

Chronic inflammation due to aspiration in the cranial trachea is seen especially in dogs. There is a history of a chronic dry cough and further questioning reveals that it occurs in the morning and is sometimes associated with drinking. Episodes of bronchopneumonia are rare. The trachea is found to be sensitive to palpation and a harsh, dry cough may be elicited. Since further physical examination reveals no abnormalities, special diagnostic techniques are necessary. The slight thickening of the cranial tracheal mucosa is not visible on radiographs of the tracheobronchial tree, but bronchoscopy reveals severe tracheitis in the cranial two-thirds of the cervical trachea, beginning at the first tracheal ring. The tracheal mucosa is swollen and reddened by hypervascularization and edema, and there is an abnormal quantity of mucus in the affected area, probably due to irritation. The cytology of the material in the tracheal lumen discloses the diagnosis when groups of large, mature squamous cells are found among much smaller neutrophils and occasional macrophages. These squamous cells originate in the mouth and some parts of the larynx, and they indicate aspiration. Further evidence of aspiration is almost always present in the form of *Simonsiella*, the large, aerobic bacteria occurring in multicellular filaments in the mouth in cats, dogs, sheep, and humans, even in the absence of oral disease.[23, 36] These findings indicate that material from the mouth is leaking into the trachea, the coughing in the morning probably being due to leakage of

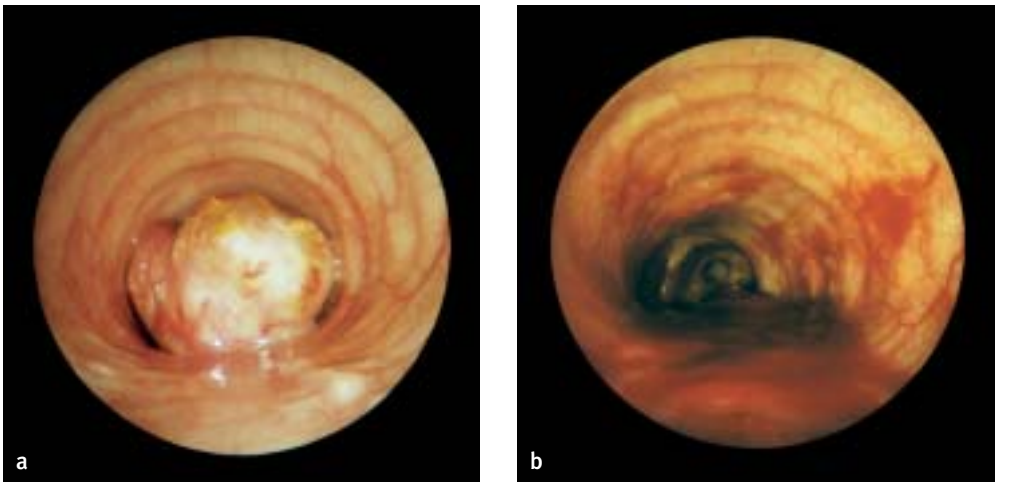


Figure 5.10 a, b: (a) Bronchoscopic appearance of a chondroma in the thoracic part of the trachea. Radiographs indicated that its attachment was small and hence it was removed under bronchoscopic vision. (b) Bleeding was moderate.

saliva during the night. The explanation for the leakage is presumed to be at least in part defective sensory innervation of the laryngeal mucosa, but with normal sensory innervation of the trachea, as demonstrated by the elicited cough and the limitation of the area affected. The clinical signs do not include dysphagia.

No effective means of preventing the leakage has been devised. The owner can only be advised to react promptly if signs of bronchopneumonia develop, so that broad-spectrum antibiotic therapy can be started. Antitussives are contraindicated.

5.6 Tumors of the trachea and bronchi

Primary tumors of the trachea are unusual in cats and dogs. Only malignant lymphoma and adenocarcinoma have been reported in cats, but the tumors reported in dogs include lipoma, chondroma, osteochondroma, chondrosarcoma, mast cell tumor, leiomyoma, and squamous cell carcinoma.[3, 5, 21, 22, 25, 33, 35] The usual history is one of increasing dyspnea and increasing inspiratory and expiratory stridor, sometimes with coughing. Cyanosis of the mucosa and gagging are observed occasionally. The predominant clinical sign is dyspnea, and auscultation of the trachea often reveals rhonchi caused by an unusual amount of mucus moving up and down around the obstruction in the trachea. The appropriate special diagnostic techniques are radiography, bronchoscopy, and biopsy with histological examination. Radiography reveals a mass in the lumen of the trachea, outlined by air. Disruption of the mucosal continuity can be seen at the base of the neoplasm. This is in contrast to the silhouette produced by an extraluminal mass, in which the interface of the luminal air and the mucosa is a continuous line passing without interruption through the mass.[18] It is important to use radiography or CT to determine the extent of infiltration by the neoplasm into the tracheal wall and beyond. A thoracic radiograph is essential to determine whether the tumor has metastasized to lymph nodes and/or the lungs.

Bronchoscopic examination of the trachea will reveal the exact location and extent of the tumor and the diameter of its base of attachment to the tracheal wall. If the tumor originates in the wall of the cervical trachea, the portion of the wall that must be removed to achieve a complete resection should be determined. Reconstruction or resection of the wall of the thoracic trachea has a poor prognosis, and bronchoscopic resection via the lumen may be the best option if the attachment of the tumor to the wall

is small (Figures 5.10 a, b). For tumors with a broad attachment, transluminal removal by laser surgery under visual control may be a worthwhile palliative measure.[43] In any case, a biopsy should be taken to determine the character of the tumor and the prognosis.

Surgical removal of the tumor. Some tumors in the cervical trachea can be removed by resecting the involved segment of the tracheal wall. A long endotracheal tube of small diameter is introduced and the cuff is inflated caudal to the site of the resection. If the size of the tumor does not permit the endotracheal tube to pass, the tube is left in the cranial end of the trachea, its cuff is inflated, and ventilation is begun. With the patient in dorsal recumbency, the skin is incised on the ventral midline and the subcutis is dissected to expose the left and right sternohyoid muscles. These muscles are separated on the midline to expose the ventral side of the trachea. Dissection along the lateral sides of the trachea is carried out cautiously in order to identify the vascular supply to the trachea and the recurrent laryngeal nerves. Circumferential dissection is carefully restricted to the segment of the trachea to be resected and only a very narrow rim of trachea cranial and caudal to this, so that the blood supply to the trachea will be intact at the level of the anastomosis.[40]

The trachea is first transected caudal to the tumor and a sterile endotracheal tube is inserted into the distal opening. The cuff is inflated and ventilation is transferred to this tube. The first endotracheal tube is left in place for a later purpose. The segment of the trachea incorporated by the tumor is then removed. Traction sutures are placed around the rings caudal and cranial to the site of resection, in order to approximate and align the two ends of the trachea. The tube in the caudal part of the trachea is removed and the tracheal rings forming the anastomosis are drawn together. The cuff of the endotracheal tube in the cranial part of the trachea is deflated and the tube is moved through the trachea until its cuff is caudal to the anastomosis. When the cuff is satisfactorily positioned, it is reinflated and ventilation is transferred to this tube. The anastomosis of the trachea is then completed with interrupted sutures of monofilament nonabsorbable material placed through or around the cartilages of the apposed tracheal rings, with the knots outside the trachea. Tension sutures can be added if there is considerable traction on the anastomosis.[40] In this technique the annular ligaments of the tracheal rings are apposed. In an alternative technique the terminal cartilage ring of each of the two severed ends of the trachea is split circumferentially to completely remove the annular ligament. Then the split rings are apposed with simple interrupted sutures, to achieve a cartilage-to-cartilage anastomosis. A study comparing the stenoses resulting from these two techniques in 20 healthy dogs found that the stenosis following the split ring technique left a wider lumen and hence it is preferred.[28]

Primary tumors of the bronchi are rare in dogs and cats. The diagnosis and treatment of pulmonary neoplasia are beyond the scope of this book, but bronchoscopy can be helpful in recognizing bronchial involvement of pulmonary tumors and the bronchoscopist may advise the thoracic surgeon about the extension of the disease in the bronchial tree.

5.7 Tracheal trauma

In dogs and cats, traumatic injury to the cervical trachea most often occurs during a fight between a dog and a cat or between two dogs. Dogs go for the throat or the ventral part of the neck when they fight and their canine teeth may penetrate or rupture the trachea. Gunshot wounds of the trachea are uncommon but they do occur in both dogs and cats. The position of the somewhat flexible cervical trachea between

the ventral neck musculature and the vertebral column provides some measure of protection from blunt trauma. Accidents in which the trachea is ruptured by traction occasionally result in an intrathoracic circumferential cleavage between two tracheal rings that nevertheless leaves the adventitia or serosa around the trachea intact. This can prevent inspired and expired air from leaking into the mediastinum, so that the dog or cat may survive.

Tracheal stenosis usually results from penetrating tracheal trauma, but sometimes from blunt trauma, a tracheostoma or other tracheal surgery, long-term endotracheal intubation associated with hypotension, or hyperinflation of the endotracheal tube cuff.

The intrathoracic trachea and bronchi can be ruptured in trauma to the thorax and can be lacerated by fractured ribs or dog bites.

5.7.1 Trauma to the cervical trachea

Management. When a dog is presented with trauma to the cervical trachea, the history usually includes adequate details about the time and circumstances of the trauma. For a free-roaming cat the history may only reveal when the cat was found after a presumed injury. The clinical signs usually include skin wounds in the neck and subcutaneous emphysema along the trachea. Sometimes the emphysema extends over the entire body. Subcutaneous emphysema in the neck area, recognized as a crackling sensation during palpation, is a clear sign of tracheal leakage of expired air. There may be dyspnea if a pneumomediastinum has developed. Dyspnea may also occur with large wounds in the trachea, when inspiration of soft tissue into the tracheal lumen obstructs the airway. The dyspneic patient must be examined quickly to determine whether there are signs of shock or hypoxia. If either of these is evident, treatment must be started immediately and without further examination. If there is a large wound in the trachea oxygen is administered without stress to the patient and without anesthesia. When oxygenation is satisfactory the patient must be examined thoroughly for wounds, luxations, fractures, or other injuries. The skin wounds in cervical tracheal trauma are usually remarkably small punctures on either side of the neck. The skin may be bruised but the coat is often intact and may obscure the injuries.

Radiographs should be taken before the surgical exploration of the trauma. Pneumomediastinum, pneumothorax, and other possible complications of cervical tracheal trauma should be evaluated to determine the anesthetic protocol. The location of the tracheal laceration is usually not to be found on the radiographs because the emphysema obscures radiographic detail. Bronchoscopy is usually not indicated because the risk of the procedure outweighs possible consequences of the findings. It should be realized that surgery is always necessary to close the laceration of the trachea and that direct visualization during surgery always provides better information about the laceration and the damage to surrounding tissues than bronchoscopy can.

The animal is anesthetized for surgery with appropriate regard for the findings of the clinical and radiographic examinations. Endotracheal intubation is accomplished with a long endotracheal tube of small diameter and without inflating the cuff. Intubation is for the purpose of administering oxygen without raising the pressure in the ruptured trachea. The animal is placed in dorsal recumbency and the neck is prepared for surgery. The skin is incised over the entire cervical trachea, from the larynx to the thoracic inlet. After ligation of lacerated vessels and midline exposure of the trachea, the site of the rupture is located. The endotracheal tube is repositioned under visual guidance so that the cuff is caudal to the laceration and then the cuff is inflated. In most cases resection of a segment of the trachea is not

needed, and the tracheal cartilage and mucosa can be débrided and then rejoined with interrupted sutures of fine monofilament nonabsorbable material. Additional sutures are placed on the outside of the trachea adjacent to the sutured wound in order to reduce tension on the closure. These sutures are placed around two tracheal rings on each side of the wound. The traction of the sutures may cause the trachea to be flexed slightly (Figure 5.11), but this has never resulted in functional disability. After resection of a segment of the trachea there is a high risk of wound dehiscence and stenosis, especially in a contaminated wound surrounded by damaged tissues. Drainage of the peritracheal area is required until the infection is controlled. During all procedures the recurrent laryngeal nerves should be avoided, for they are very susceptible to damage by handling.

When the trachea is found to be lacerated in a heavily damaged and contaminated area, and the tracheal wound is so large that resection of the affected segment is being considered, the surgeon should realize that there is a high risk of dehiscence of a tracheal resection wound. Instead, the wound and the surrounding tissue should be débrided and a T-tube should be inserted through the wound into the tracheal lumen. The technique is described below (Chapter 5.8.5). The aim of using a T-tube is to promote secondary healing of the tracheal wall while limiting stenosis. The tube selected should be wide enough to compensate for some stenosis and thus minimize the narrowing of the trachea. The T-tube remains in place for 6 to 8 weeks, during which time drainage of the wound around the trachea is maintained and antibiotic therapy is given. The state of healing in the tracheal lumen can be monitored by tracheoscopy.

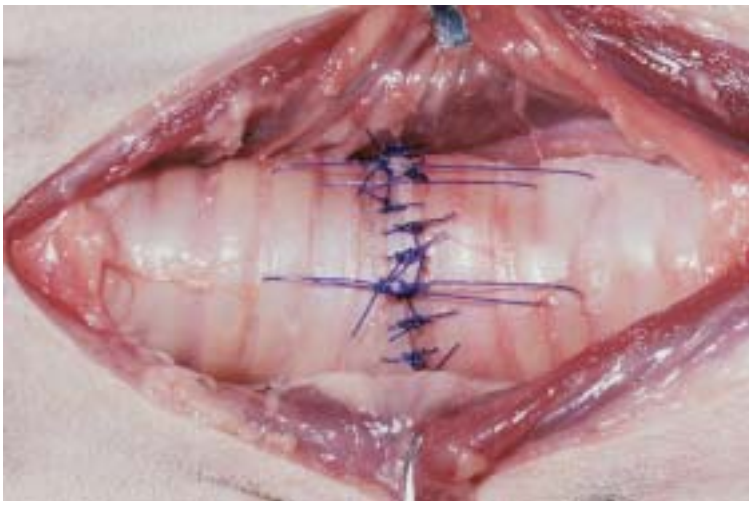


Figure 5.11:
Traction sutures are employed after resection of a segment of the trachea. Only the cartilaginous rings are removed; the dorsal ligament is not severed. After healing, the trachea may be flexed but this usually does not result in functional disability.

5.7.2 Trauma to the thoracic trachea

Intrathoracic avulsion of the trachea has been described as a posttraumatic syndrome in cats, characterized by circumferential cleavage between two tracheal rings that leaves only the tracheal adventitia or serosa intact.[51] The rupture is usually found 1 to 4 cm cranial to the bifurcation. It leads to tracheal stenosis and there may be formation of a pseudotracheal lumen or diverticulum.

In most but not all of the 12 reported cases of intrathoracic tracheal avulsion in cats, the history has included a known accident or trauma. The clinical findings are dominated by dyspnea but sometimes other injuries are the reason for consulting the veterinarian. The first special diagnostic technique is radiography of the trachea and thorax. The lateral view reveals a gas-filled dilation of the trachea, halfway between the thoracic inlet and the bifurcation, with a thin soft-tissue lining (Figure 5.12). The length of the dilated segment varies but may reach 2 cm. In three of the reported cases, endoscopic examination revealed a tracheal stenosis through which the endoscope could not be passed.[51] An uncuffed endotracheal tube, long enough to reach beyond the proposed tracheal anastomosis, was put in place

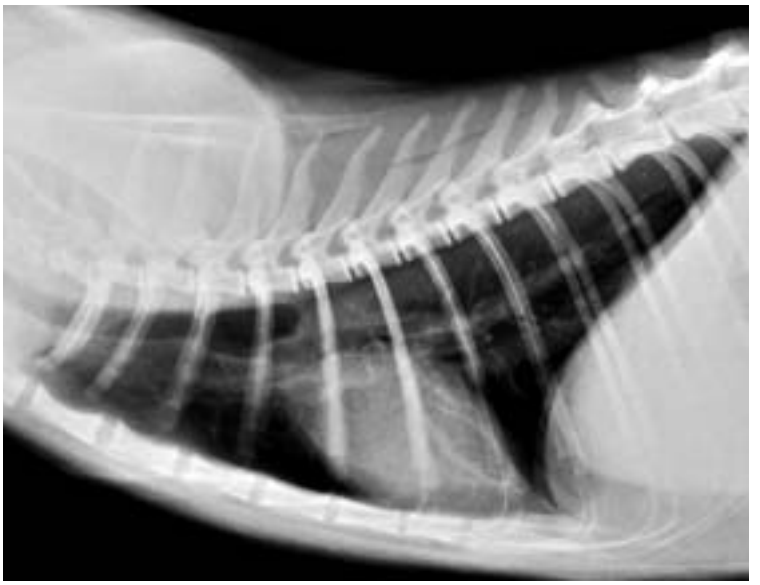


Figure 5.12: Laterolateral radiographic appearance of intrathoracic tracheal avulsion in a cat, approximately 3 cm cranial to the bifurcation. (Courtesy of G. ter Haar, Division of Surgery, ENT, Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, and Dr. W.Th.C. Wolvekamp, Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University).

cranial to the stenosis. A sterile cuffed endotracheal tube was prepared for insertion into the trachea caudal to the avulsion during surgery. The tracheal laceration was approached via a lateral thoracotomy in the third or fourth right intercostal space. The pseudoairway was opened to expose the stenotic caudal tracheal orifice. Temporary ventilation was provided by inserting the uncuffed endotracheal tube into this orifice. Because this ventilation was insufficient, the stenotic end of the distal trachea around it was rapidly resected so that the sterile cuffed endotracheal tube could be inserted to continue adequate ventilation. The stenotic end of the trachea cranial to the avulsion was then also removed and the two cleaned ends were prepared for anastomosis by placing 3 fine monofilament sutures around the terminal ring of each. The caudal endotracheal tube was removed and the cranial tube was advanced past the site of anastomosis for continuing ventilation. Additional interrupted sutures were placed to complete the anastomosis, and the thorax was closed routinely. In the reported cases respiratory function was normal after surgery and also at long-term follow-up.[51]

5.7.3 Tracheal stenosis

Tracheal stenosis usually follows an external injury that causes blunt trauma to the cartilage accompanied by disruption of the mucosa or the annular ligament and formation of a hematoma. Organization of the hematoma leads to collagen deposition and scar tissue formation. Mucosal ulceration associated with endotracheal intubation, through pressure or direct injury, also leads to healing by collagen deposition, fibrosis, and scar tissue formation that culminates in tracheal stenosis.[44] Surgical intervention in the trachea is another event that may lead to the development of stenosis.

A representative history in cases of tracheal stenosis reflects the time required for development of the stenosis; the surgical procedure or a traffic accident may have taken place a couple of weeks before the dyspnea became evident. Dyspnea is the predominant clinical sign in tracheal stenosis. Sometimes a stenosis is found fortuitously, which indicates that not all degrees of stenosis cause clinical signs, and if there are no signs, there is no call for therapy.

Although imaging of the trachea by radiography, CT, and MRI may provide information about the exact location and the length of the stenotic segment, endoscopic examination provides information about the diameter of the lumen within the stenosis and the type of tissue that forms it. In early stages of the development of a stenosis granulation tissue and edema may be recognized (Figure 5.13), while in a mature stenosis fibrosis is observed (Figures 5.14 a, b).

Surgical approaches to treatment. There are several approaches to treatment of a tracheal stenosis. When the stenosis is caused by granulation tissue and edema, dilation may be preferred to conservative treatment with an antibiotic and a glucocorticoid, because once fibrosis and scarring have begun, the natural process of contraction may reduce the diameter of the lumen even further. A T-tube can be inserted to

accomplish dilation in the cervical part of the trachea (Chapter 5.8.5). Dilatation in the thoracic trachea by insertion of a stent for several weeks may be not feasible. In humans, intraluminal removal of granulation tissue with the Nd-YAG laser is highly recommended.[44] In lieu of this, dilation of a fibrotic stenosis in the dog or cat by tracheal bougienage using bronchoscopes of increasing diameter over several sessions is worth trying. The dilation should be achieved by stretching the tissue, taking care to avoid lacerating the trachea. This procedure has proved to be useful in both the cervical and the thoracic trachea, and is the treatment of choice in the latter, but scar tissue can recur.

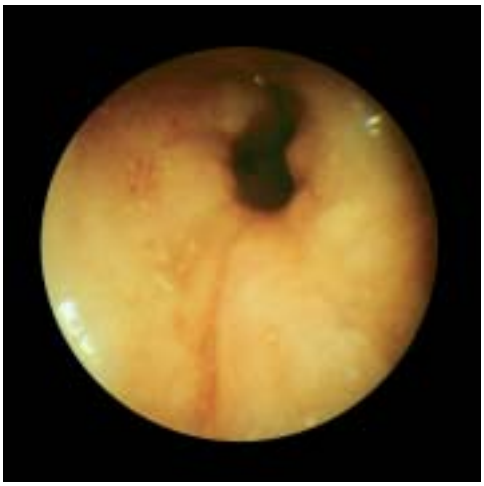
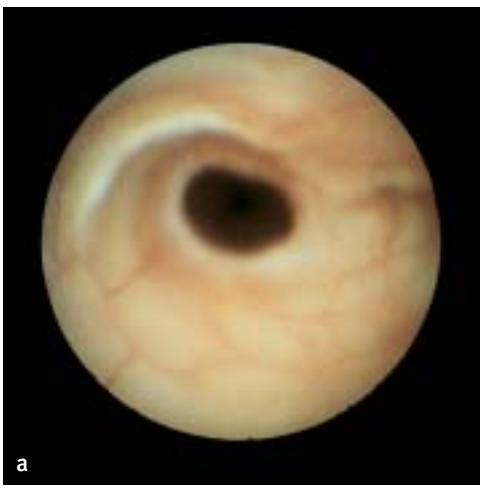


Figure 5.13: Granulation tissue and edema characterize the early bronchoscopic appearance of a tracheal stenosis in a cat.

A short stenosis in the cervical trachea should preferably be resected. The technique of resection and reanastomosis of the segments is as has been described for excision of a tracheal tumor (Chapter 5.6). Since the causative trauma occurred weeks earlier, the healing of the tracheal wound will be uncomplicated and drainage of the surrounding tissues is unnecessary. Resection of a tracheal segment equivalent to 20 to 25 % of the trachea in a puppy or 20 to 50 % in an adult dog is tolerated, but tension on the anastomosis can lead to dehiscence or stenosis.[40]

5.8 Airway management

Airway management is important in diseases of the larynx and the trachea, both to prevent obstruction and to bypass an existing obstruction. Safe anesthesia includes establishment of a patent airway. In the unconscious dog and cat the larynx is slightly open but there is no abduction of the vocal folds during inspiration. The tongue is flaccid and may obstruct the airway, and the pharyngeal muscles are also inactive. The conscious reaction to obstruction of the larynx and trachea—stretching the neck, opening



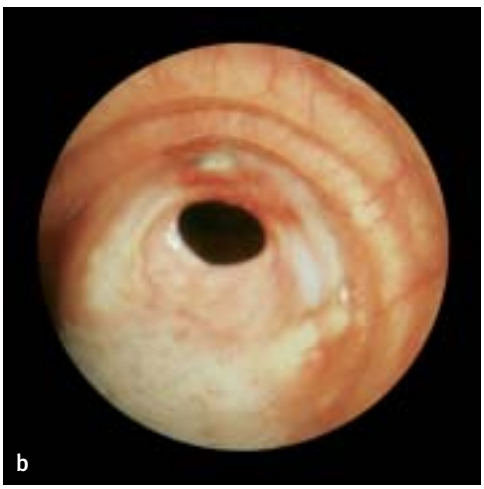


Figure 5.14 a, b: (a) A mature tracheal stenosis in a cat, characterized by a fibrotic rim. (b) A mature tracheal stenosis in a dog, with scar tissue (fibrosis) narrowing the lumen.

the laryngopharynx, protruding the tongue, and abducting the vocal folds—is eliminated. An endotracheal tube prevents obstruction of the airway when the animal is under anesthesia. When a life-threatening obstruction of the larynx or cranial part of the trachea is evident, immediate anesthesia followed by endotracheal intubation is essential. In addition to endotracheal intubation, other airway bypassing techniques—cricothyroidotomy, tracheostomy, permanent tracheostoma, and insertion of a T-tube—also have a place in the rescue management.

5.8.1 Endotracheal intubation

Endotracheal intubation is the most frequently used method of establishing a patent airway and is also used to prevent or to bypass an obstruction of the airway in dogs and cats. It is used during anesthesia both to prevent obstruction due to laxity of the tissues forming the entrance to the larynx and the laryngeal glottis, and to ensure adequate ventilation and oxygenation of the unconscious animal. The choice of endotracheal tube and the technique of intubation are dictated by the anatomy of the upper airway of the individual dog or cat. Murphy-type and Cole-type endotracheal tubes are commonly used in veterinary anesthesia.[24] The Murphy tube has an opening in the wall opposite the bevel at the tracheal end of the tube, to allow gas flow even if the beveled end is occluded. The tube is slightly curved and the angle of the bevel is 38°. The Murphy tube is cuffed and the narrow tube used to inflate and deflate the cuff is provided with a small balloon and a valve at the outer end. The Murphy tube is available in all sizes required for dogs, but the smallest size provided with a cuff is 3.0 mm i.d., and it will pass through the larynx of smaller adult cats but not that of kittens. Small Cole tubes may be used in kittens. Cole tubes are uncuffed and are both slightly curved and smaller in diameter near the distal (tracheal) end. The larger diameter of the laryngotracheal part creates a seal which prevents leakage of gas from and aspiration of material into the trachea.[24] The length of the endotracheal tube, measured along the side of the animal's neck, should reach from the incisor teeth to near the thoracic inlet. The appropriate diameter is selected by estimating the size of the laryngeal opening during the preliminary laryngoscopic examination.

Laryngoscopy. For laryngoscopy the animal is placed in a sphinx posture with its head supported by an assistant standing at its side. Its mouth is then opened and its neck extended by the assistant, using one hand to hold the upper jaw and the other to hold the lower jaw and flatten the tongue. The laryngoscope is introduced over the tongue for inspection of the mouth, oral pharynx, and ventral side of the epiglottis, before the epiglottis is depressed for inspection of the glottis. The size of the laryngeal opening is the first concern, for spontaneous breathing requires an adequate laryngeal opening and if the opening is obstructed, endotracheal intubation must be performed immediately. Artificial ventilation via the tube will then be needed, at least for a short while. If intubation is not required, the glottis is inspected, the movement of the glottis and the color of the laryngeal mucosa are evaluated, and the cartilages are inspected for deformities. The blade of the laryngoscope should not be passed through the glottis if laryngoscopy is only being performed to insure safe endotracheal intubation and there are no apparent abnormalities. The laryngeal mucosa is anesthetized locally with lidocaine spray and the endotracheal tube is inserted through the larynx into the trachea. In cats the laryngeal mucosa is prone to develop edema and intubation should be performed carefully. In cats the blade of the laryngoscope may also be placed under the epiglottis to avoid irritation of the laryngeal mucosa.

Laryngoscopy is the most important tool for diagnosis of laryngeal obstructive disease, since not only the size of the laryngeal opening, the glottis, and the surrounding structures can be examined, but also laryngeal function can be evaluated under the appropriate level of anesthesia. Emergency intubation under anesthesia is possible in almost all cases of laryngeal obstruction. The rationale for this is that if the animal was breathing through the upper airway before anesthesia was induced, there is a patent opening through the larynx. If clinical examination has suggested that the obstruction is severe, several endotracheal tubes of diameters smaller than usual for the size of the animal should be prepared.

Endotracheal intubation in brachycephalic dogs. Endotracheal intubation is especially complicated in brachycephalic dogs with a narrow pharynx, an overlong soft palate, a thick tongue base, and hypoplasia of the larynx. During the induction of anesthesia (hypnosis), there is often a moment when the dog is severely dyspneic but still too conscious to accept endotracheal intubation, which may cause the dog to struggle in fear of suffocation. The only solution is to wait a few seconds more for the onset of hypnosis, but then intubation should be accomplished immediately. Several lubricated small-diameter endotracheal tubes should be ready and in addition to the laryngoscope a long forceps should be at hand to lift the soft palate if necessary. Possible complications of the dyspnea and fearful struggling are a massive accumulation of foamy mucus in the pharynx, lung edema, or even death by suffocation. Lung edema is recognized by the appearance of pink foamy mucus in the larynx or the endotracheal tube. The steps to be taken depend on the results of monitoring of the ECG and pulmonary gas exchange. Administration of oxygen before the induction of anesthesia is a simple and effective measure of support in dogs which have been chronically struggling to breathe. This should be done by quietly bringing the open end of the oxygen hose near the animal's nose or open mouth without causing it any anxiety. After intubation oxygen is given without delay by use of a manual resuscitation bag. Then the anesthetist's routine procedures begin.

Complications in endotracheal intubation. Various complications can develop during endotracheal intubation. The tube can become obstructed by excessive mucus or vomitus. Only one lung may be ventilated as the result of inserting the tube into one main stem bronchus; this can occur especially when the endotracheal tube is inserted via a tracheostoma, as is routine during laryngeal surgery. Leakage of the cuff can occur and is the immediate concern of the anesthetist. Laryngeal nerve paralysis may result from pressure on the recurrent laryngeal nerve by inflation of the cuff inside the thyroid cartilage or the use of too large a tube in small dogs or cats with soft laryngeal cartilages.[7]

Prolonged endotracheal intubation. Laryngeal injury from prolonged endotracheal intubation was described in 14 dogs. The injury was primarily the result of pressure exerted by the wall of the tube. Ischemic injury, initiated when this pressure exceeds capillary perfusion pressure, is underway within the first few hours of intubation. Due to undefined host factors, the injury is variable in degree and not clearly related to the duration of intubation. Reduction of the pressure by dispersing it over a larger area of contact will prevent mucosal ischemia and its sequel of cartilage destruction, cricothyroid joint injury, and laryngeal stenosis.[49]

In general, the use of an endotracheal tube is a safe procedure, as long as the patient's ventilation is monitored routinely.

5.8.2 Cricothyroidotomy

This technique is used to obtain bronchial material in cases of bronchopulmonary disease with limited lung function when culture is necessary but the risk of anesthesia is high. It can be performed after disinfecting and anesthetizing the area over the cricothyroid junction. The same approach is sometimes used to obtain an emergency surgical airway. The technique involves the creation of an opening through the cricothyroid ligament and introduction of a small stenting tube.[44] The procedure cannot be carried out as rapidly in dogs and cats as in humans because hair must first be clipped away to find the site for the incision. To create an opening large enough for a small tube or a thick cannula, a skin incision should be made over the place were the cricoid ligament can be palpated. The opening into the airway can be used for emergency administration of oxygen, but it should be replaced by a tracheostomy if long-term bypassing of the upper airway is needed, for the opening is too small for spontaneous ventilation. It has the advantage of rapid closure after removal of the tube: applying pressure over the opening for 5 minutes usually prevents the occurrence of subcutaneous emphysema.

Complications in cricothyroidotomy. Possible complications include bleeding, tube displacement, and the development of subcutaneous emphysema, but these rarely occur when emergency cricothyroidotomy is soon followed by anesthesia and endotracheal intubation or tracheostomy.

5.8.3 Tracheostomy

Tracheostomy is a surgical intervention to provide a direct connection to the trachea from the outside. It is useful when the airway through the larynx, the mouth, or the nose is obstructed, i.e., when the airway obstruction is located cranial to the opening in the trachea. It is most often used as a temporary bypass during the time that it takes to remove the obstruction or for obstructive edema to disappear.

Indications for tracheostomy. Laryngeal surgery is one of the indications for tracheostomy. During and after laryngeal surgery, edema of the laryngeal mucosa may cause life-threatening dyspnea. A similar indication is severe trauma to the nose. Although obstruction of the nose obviously does not preclude breathing through the mouth, neither cats nor dogs appear to be able to do this continuously while at rest or sleeping. Hence they usually become hypoxic and hypercapnic during sleep if the nose is obstructed. Tracheostomy can prevent this distress and even death due to insufficient oxygenation of the heart and brain and respiratory acidosis.

Procedure. Tracheostomy is always preceded by intubation. Remember that in the dyspneic animal there is always an opening to the airway, however small and difficult to find, for without it the animal would not be alive. Introducing an endotracheal tube always requires anesthesia. In the dyspneic patient the brain may be more sensitive to anesthetic drugs and the safest approach is to use an intravenously administered anesthetic in low doses, while observing the effect during injection. Medetomidine and propofol is an appropriate combination. Once the endotracheal tube is in place and the patient's condition is stable, the patient is placed in dorsal recumbency and the ventral side of the neck is clipped and otherwise prepared for sterile surgery. Sterile tracheal cannulas of several sizes are made ready. The diameter of the tracheostomy should be just large enough to permit insertion of the cannula—as a rule a round hole with a diameter just spanning two tracheal rings. The cannula should hang loosely in the tracheal lumen, with its inner

opening pointing in the direction of the lungs. A small *transverse* skin incision is made over the cervical trachea and the subcutis is removed until the tracheal rings are visible. A pointed scalpel is used to make a round opening in the trachea exactly fitting the tracheal cannula. Before inserting the cannula, the previously placed endotracheal tube should be slowly withdrawn until it is no longer visible in the opening. The cannula is then inserted, sutured to the skin, and also anchored with a soft cord around the neck. In cats it is important to also tape the cannula in place, because cats often try to remove it, which dogs seldom do. The string and tape should be loose enough to avoid compressing the jugular vein.

Complications in tracheostomy. Although complications can develop, most can be prevented by careful forethought. A tracheal cannula introduces dry air and so mucus or pus being expelled via the trachea tends to congeal in the lumen of the cannula, obstructing it. The best way to handle this problem is to use cannulas consisting of an outer and an inner part. The inner cannula can be removed and cleaned without disturbing the fixation of the outer cannula. The inner cannula should be cleaned every 2 hours, 24 hours a day. Cannulas for cats have an even smaller lumen and may become obstructed by excessive mucus. If this occurs, it may be necessary to place the cat under day and night observation.

A tracheal cannula should be made of rigid material and not plastic that softens at body temperature. Those that soften often flatten in the tracheal wound so that the lumen becomes dangerously narrow. Cannulas for dogs should be extra long so that they do not slip out of the tracheal opening and become lodged in the subcutis, which is very dangerous and may easily escape the caretaker's attention.

Bronchopneumonia is another risk for an unprotected airway. The surroundings should be kept clean, the caretakers' hands should be washed and gloves worn to handle the inner cannula, and a broad-spectrum antibiotic should be administered for the duration of the use of the tracheal cannula.

The cannula can be removed safely when inspection under anesthesia reveals satisfactory resolution of the initial obstructive lesion. After the cannula is removed, the patient is kept under close observation for 20 minutes or longer. To avoid the development of subcutaneous emphysema, the wound in the trachea and skin is never sutured. Its spontaneous closure is effective in 20 minutes and complete in 3 days. The transverse incision of the skin over the trachea aids rapid skin healing. Healing may be complicated if the opening of the airway cranial to the stoma is not wide enough for normal respiration. Continuous forced inspiration and expiration can cause negative pressure in the trachea, drawing the soft tissue covering the tracheostoma into the lumen. In rare cases this has become permanently fixed in the tracheal lumen, causing obstruction. Since the primary cause is a too-narrow airway cranial to the site, treatment requires both removal of the obstructing tissue and enlargement of the cranial airway.

Tracheostomy is a wonderful tool in capable hands and every veterinarian should be able to perform it correctly. The care of the patient with a tracheal cannula should be planned, for the cleaning procedure is a burden. After the tracheostomy has been performed it may be worth considering referral of the patient to a specialized clinic for further care.

5.8.4 Permanent tracheostoma

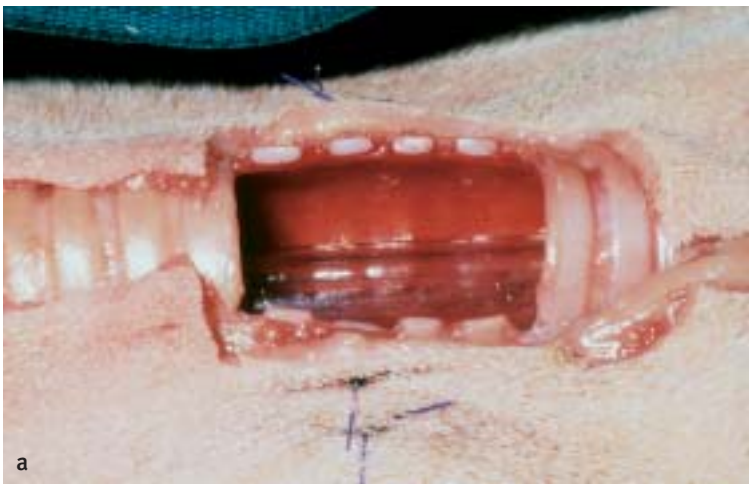
Indication for permanent tracheostoma. The surgical creation of a permanent tracheostoma is indicated in both dogs and cats which require maintenance of a tracheal opening for an indefinite period.[27] Indications include severe hypoplasia of the larynx, severe stenosis of the lar-

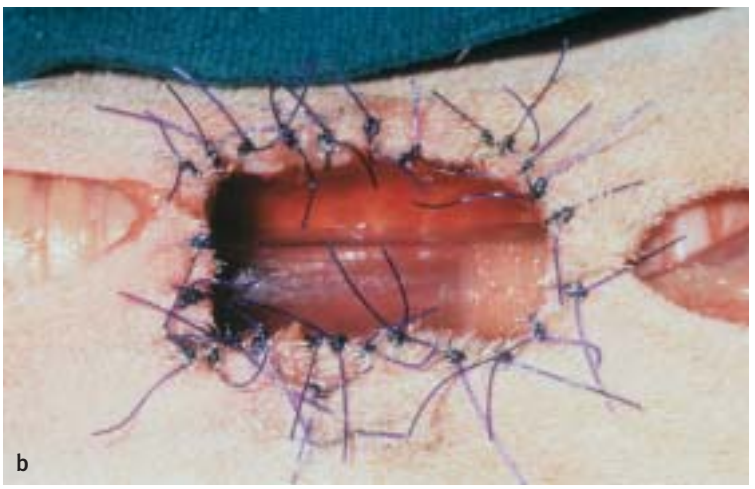
Figure 5.15 a–c: (a) The length of a permanent tracheostoma should be about 4 tracheal rings. (b) The edges of the rectangular gap in the skin are sutured to the corresponding edges of the tracheal mucosa with interrupted sutures of fine, polydioxanon suture material. (c) A permanent tracheostoma in a dog after removal of the sutures. The opening in the trachea was satisfactory.

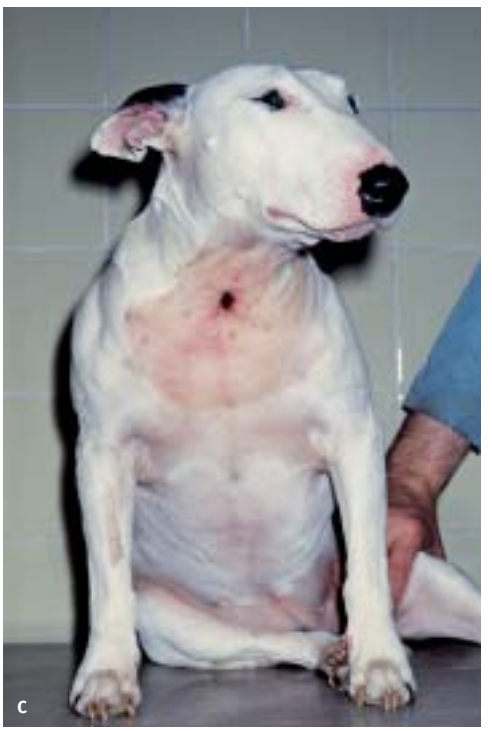
ynx after trauma, permanent nasal obstruction, and other acquired obstructions. The advantage of a permanent tracheostoma over a permanent tracheal cannula in that there is no complication of tube displacement—which is extremely dangerous if it results in obstruction of the tracheostoma by the tube—and no permanent irritation of the tracheal mucosa.

Procedure. The creation of a permanent tracheostoma begins with an endotracheal tube in place. The tube may have been introduced through the larynx or through an existing tracheostoma. In the latter case the surgeon must decide whether to make a second opening caudal to the existing tracheostoma or to use the existing tracheostoma and work around the endotracheal tube.

The animal is prepared for surgery and placed in dorsal recumbency. The neck is straightened so that the trachea is in the midline. The skin is incised on the midline 2 to 4 cm cranial and caudal to the site chosen for the tracheostoma. The paired sternohyoid muscles are separated to expose the trachea. Retracting each muscle laterally and a slightly dorsally allows the trachea to be lifted up to the level of the skin. The trachea is then fixed in that position by placing one or two horizontal mattress sutures of absorbable material through the sternohyoid muscles to appose them anatomically dorsal rather than ventral to the trachea. A rectangular segment of the ventral wall of the trachea is then removed with a pointed scalpel blade. The opening should be twice as long for a tracheostoma as for a trachea cannula, about 4 tracheal rings, but the same width, equivalent to about 2 tracheal rings. The skin is then







sutured directly to the external fascia of the trachea lateral to the opening, leaving enough free skin over the stoma to allow removal of a small rectangular flap on each side to closely match the rectangular outline of the stoma. The edges of this rectangular gap in the skin are sutured to the corresponding edges of the tracheal mucosa with interrupted sutures of fine, polydioxanon suture material (Figures 5.15 a–c). The midline incision of the subcutis and skin cranial and caudal to the tracheostoma is then closed routinely.[27, 29] Broad-spectrum antibiotics are prescribed for 14 days and the sutures around the stoma are covered with an antibiotic ophthalmic ointment for one week.

Caring for a tracheostoma involves removal of clinging mucus whenever it tends to obstruct the opening. The owner is also instructed to absolutely avoid allowing the dog near any water in which it might attempt to swim. Owners may find this difficult, but **swimming is fatal for a dog with a tracheostoma.**

5.8.5 Tracheal T-tube

As originally designed, the silicone tracheal T-tube is a flexible tracheal tube in the shape of a T, the top of which fits in the cervical trachea while the leg protrudes through the tracheostoma and opening in the skin. This outside end is closed with a silicone plug.[37] The silicone tracheal T-tube is designed to maintain an adequate tracheal airway as well as to provide support in the stenotic or reconstructed trachea. To insert the tube into the trachea through the stoma, the cranial—usually shorter—end and the leg of the tube are pressed together while the caudal end is directed caudally into the trachea and, with the aid of a hemostat, is pushed deeply enough to allow the cranial end to slip into the trachea in the cranial direction. The tube is brought into the correct position by pulling the external leg until it exits at an angle of 90° (Figures 5.16 a, b and Figure 5.17), and the external end is then firmly closed with the plug. The intraluminal part of the T-tube is in contact with the segment of the trachea requiring support or stenting. The T-tube is designed to remain in the trachea for weeks or months. The plug remains in place but can be removed when a direct opening to the trachea is needed. The tube is eventually removed by simply pulling on the external part and the tracheostomy wound then heals spontaneously.

Silicone T-tube. The silicone T-tube has several advantages: (a) its flexibility makes it both less irritating to the tracheal lining and easy to insert

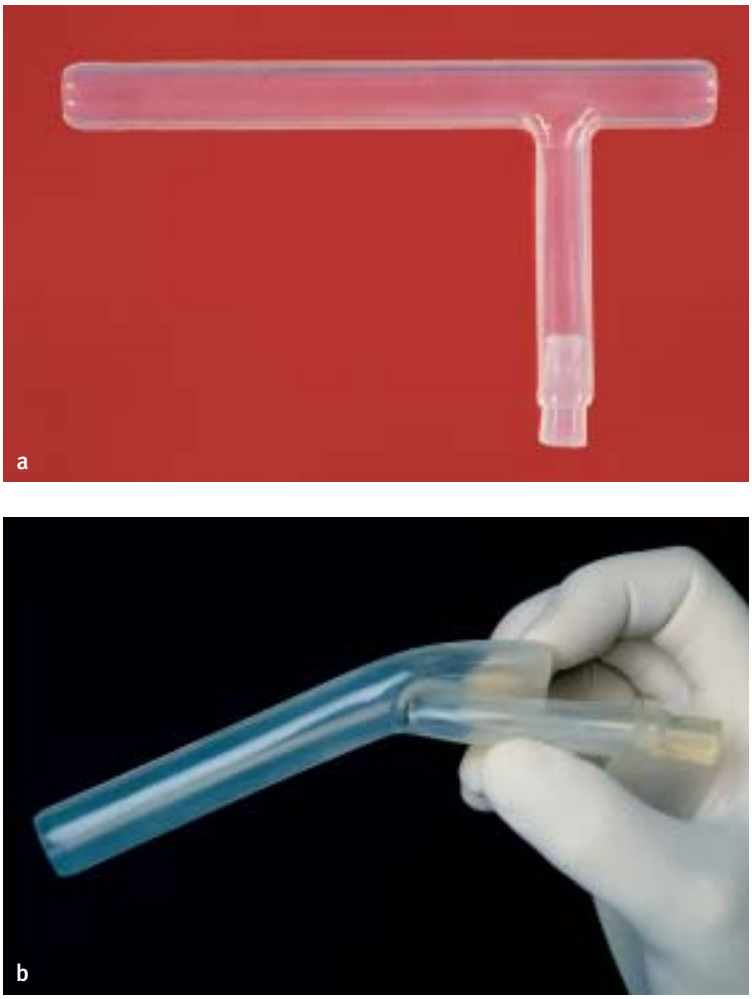


Figure 5.16 a, b: (a) Tracheal T-tube used in infected tracheal wounds to prevent stenosis. (b) To insert the T-tube into the trachea, the shorter (cranial) end and the leg of the tube are pressed together while the caudal end is inserted into the tracheal stoma, directed caudally, and pushed deep enough to let the cranial end slip into the trachea in the cranial direction.

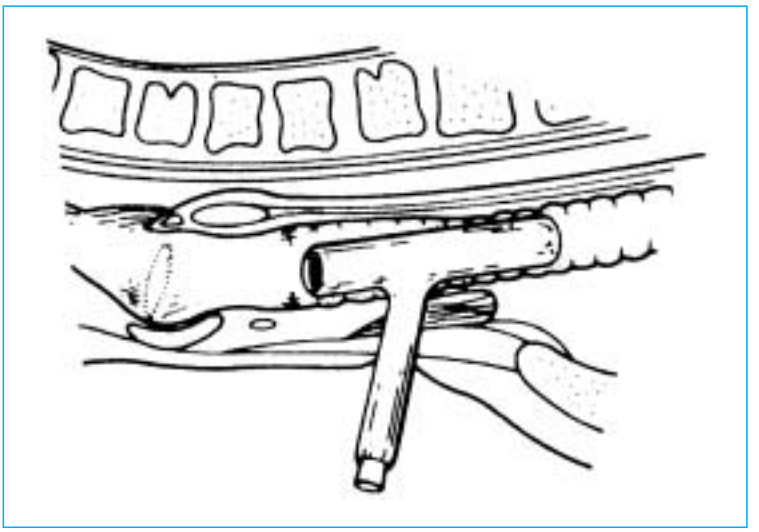


Figure 5.17: T-tube placed in the correct position: the short end is toward the larynx and the leg protrudes out through the stoma.

and remove; (b) there is little or no tissue reaction to silicone; (c) mucus does not readily adhere to the surface of the tube, so there is little formation of crusts while the tube remains plugged; (d) silicone is not hardened by prolonged contact with body temperature and secretions, as are most other plastics; (e) any or all three arms of the tube can be shortened with a knife or scissors if necessary; (f) the plugged tube allows normal coughing and barking and normal olfactory function; and (g) frequent changing is not necessary. The silicone tube is known to have remained in place for 15 months in humans.[38]

Indication for using a T-tube. The tracheal T-tube is used successfully in both humans and dogs. In dogs it can prevent stenosis which could otherwise be expected after severe trauma to the trachea, and it can be used as a stent in a trachea in which stenosis is already developing. It can also be used in an infected tracheal wound in which reconstruction has an adverse healing prognosis. The progress of the tracheal repair can be monitored by tracheoscopy (Figures 5.18 a, b).

The tracheal T-tube is available in many sizes and models[39] and can also be made to order to meet special requirements. An average model could be kept on hand for use until delivery of a specific size or model for a given patient. Coughing is a common complication, but mild sedation and resting of the dog will usually reduce it sufficiently when necessary. In most cases 6 to 8 weeks of stenting with a T-tube is sufficient to obtain the desired result in dogs.

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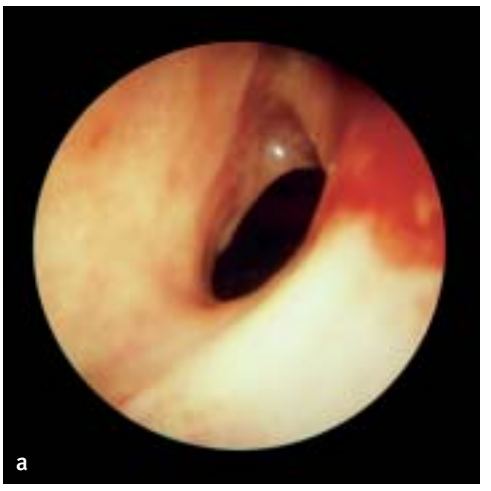

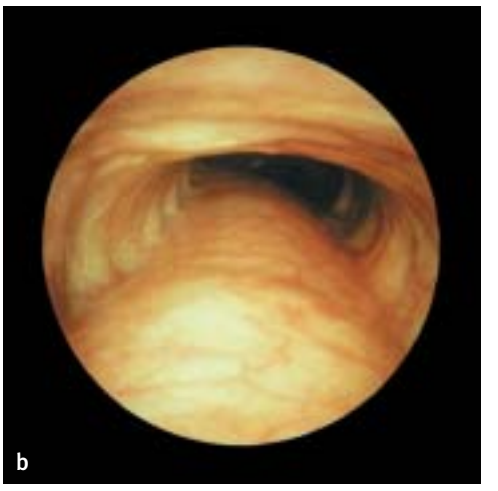

Figure 5.18 a, b: (a) Infected tracheal stenosis in a dog following a bite wound. A T-tube was inserted to stent the trachea for 6 weeks. (b) Three weeks after removal of the T-tube, the trachea was healed and the stoma was closed by scar tissue (top left).

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6 Cranial Neuralgias and Facial and Trigeminal Paralysis

6.1 Cranial neuralgias

Cranial neuralgias are disorders affecting cranial nerves that have sensory functions, causing paroxysmal or persistent pain that is severe. In humans the pain is described as »throbbing« and »stabbing« in the area of distribution of the cranial nerve involved.[3] The following descriptions are a guide to interpretation of the behavior of dogs and cats with these disorders.

6.1.1 Glossopharyngeal neuralgia

Clinical signs. This uncommon but dramatic phenomenon has been described in dogs.[5, 6] The history and clinical signs are characterized by attacks of severe cramping of the neck muscles, during which the head is drawn in toward the shoulders while the dog screams and salivates profusely and its behavior expresses agonizing pain. The attacks may only last for several seconds but can recur several times daily. In humans with this disorder the trigger point is described as being located in the tonsillar fossa and attacks are provoked by swallowing or yawning. In dogs the location and trigger mechanism are not so clear, but the clinical signs are similar to those of the severe pharyngeal pain of acute pharyngitis, but much more intense, at times overwhelming. An immediate cause is seldom apparent or mentioned in the history and the frequency of the attacks is described by the owner as progressing until they dominate all other functions and habits. During the attack-free intervals physical examination reveals no abnormalities other than wetness of the coat under the neck from the drooling of saliva.

Routine laboratory studies should include measurement of serum or plasma electrolytes, for plasma potassium is usually decreased as a result of the profuse loss of saliva. Further diagnostic studies, in search of a possible cause of pharyngeal pain, consist of pharyngeal inspection under anesthesia and then radiographic examination of the pharyngeal area, the hyoid bone, the cervical region, and the skull. CT is indicated to examine the brain for possible tumors, as these may cause glossopharyngeal neuralgia as part of the clinical signs.[5]

Therapy. Treatment with the analgesic carbamazepine can be tried but plasma potassium concentration must first be normalized, because the combination of hypokalemia and carbamazepine may cause heart block. The therapy should be continued for at least 2 weeks to evaluate its effectiveness, during which time the potassium plasma level must be kept under control. A combination of carbamazepine and phenytoin is also recommended. The prognosis in severe cases is guarded, for it is extremely difficult to suppress the attacks so effectively as to allow the dog to regain its normal life.

6.1.2 Trigeminal neuralgia

Clinical signs. Trigeminal neuralgia is well known in humans but is rarely encountered in dogs or cats. It is characterized by attacks of pain which cause moaning and self-mutilation brought about by scratching the face. In contrast to Aujeszky's disease, it is usually restricted to one side of the face. In the rare cases which we have diagnosed, the attacks were less frequent than those in glossopharyngeal neuralgia but were just as difficult to suppress with carbamazepine. Sometimes the signs are caused by neuritis of the sensory part of the trigeminal nerve and are accompanied by hyposensitivity of the affected side of the face.

In these cases there may be complete remission of the pain after several weeks.

6.2 Facial and trigeminal paralysis

6.2.1 Facial paralysis

Clinical signs. Facial paralysis is rather common in dogs and cats. It can be complete or partial and is usually unilateral. It is recognized by the dysfunction of the facial musculature which results in ipsilateral drooping of the lower eyelid with inability to blink, ipsilateral drooping of the upper and lower lips, and sagging of the ipsilateral auricle. The muscles of the upper eyelid are doubly innervated, from the facial nerve (m. palpebralis oculi) and the oculomotor nerve (m. levator palpebrae superioris), and it is because of the latter innervation that the upper eyelid does not droop. The third eyelid is passive and moves when the globe is retracted by the retractor bulbi muscle, which is innervated by the abducens nerve. The most debilitating consequence of facial nerve paralysis is the leaking of saliva and food from the drooping lateral commissure of the lips (Figure 6.1).

Figure 6.1: Left-sided facial paralysis in a boxer, causing drooping of the lower eyelid and the lips. Leakage of saliva and food from the lateral commissure of the paralyzed lips is the most debilitating consequence of facial paralysis.

Facial paralysis in dogs and cats is most often caused by injury to the nerve during surgery. Removal of the external ear canal in dogs and cats may cause injury to the facial nerve because it is closely joined to the cartilage of the medial part of the external ear canal. An increase in the circumference of the external ear canal caused by chronic proliferative otitis externa may increase the risk of injuring the nerve during the procedure to free it from the cartilage. In cats the nerve is thin and especially sensitive to pressure, which can cause facial nerve paralysis lasting 6 weeks to 3 months after surgery.

Prognosis. Especially in cases in which facial paralysis is due to known trauma to the nerve and thus spontaneous recovery of the motor innervation of the facial muscles cannot be expected, surgical correction is indicated if drooling becomes a serious problem. The cheiloplasty technique used for this consists of fixing the caudal part of the lower lip to the inside of the caudal part of the upper lip.[1, 4] A mucocutaneous flap is created from the lower lip, 2 to 3 cm rostral to the lateral commissure. Equidistant from the commissure the mucosa and submucosa of the upper lip are incised to form a defect equal in length to the mucocutaneous flap. The lower lip flap is then secured into the defect in the upper lip mucosa with a row of prepared simple interrupted sutures of monofilament absorbable material. The sutures are tightened to appose the two mucosal incisions. Before the surgery is completed, the animal's mouth should be opened and closed a few times to ensure that adequate motion is preserved.[1]

The facial nerve runs inside the temporal bone, near, but not in direct contact with, the

middle ear cavity. Tumors in the middle ear can cause facial paralysis, as can chronic destructive middle ear inflammation. CT is the special diagnostic technique of choice when destruction of the temporal bone is suspected.

When facial paralysis is partial and does not cause such prominent clinical signs, the diagnosis can be confirmed or disproved by electromyographic examination of the facial muscles. This can be done without anesthesia or sedation. The finding of denervation potentials—fibrillation potentials, positive waves, and complex repetitive discharges—is conclusive for facial paralysis. The facial muscles are adjacent to the facial skin and should be differentiated from the masticatory muscles, which are deeper. The facial muscles are normally active in the conscious animal.

6.2.2 Trigeminal paralysis

Clinical signs and therapy. The pathogenesis of trigeminal paralysis in dogs is not known, but the clinical signs can be recognized without difficulty. The dog is unable to close its mouth and its mandible hangs down several centimeters. The dog is thus unable to eat or drink without assistance. The condition has also been also called »canine dropped jaw syndrome«.[2] The clinical signs are acute and only the motor branches of the trigeminal nerve are affected. The disease is self-limiting and most affected dogs recover in 4 to 8 weeks. In the meantime, the dog must be assisted to eat and drink. A small band is placed around the muzzle to partially close the mouth, leaving an opening through which the tip of the finger just passes. Food should be pureed in a mixer and should be quite liquid during the initial period. The water pan should be cleaned regularly, for it will accumulate food that adheres to the dog's face, since the dog can only suck up the food rather than take bites. The band can be removed after the dog eats and drinks, although most dogs do not appear to find it uncomfortable. However, since it prevents the dog from opening its mouth wide enough for heavy breathing or panting, the band should be removed when the dog is very active or excited, and high ambient temperatures should be avoided. Glucocorticoids do not alter the course of the disease and should be avoided, also because they increase the dog's appetite and thirst, adding unnecessarily to the already complicated care required of the owner, as well as making the dog feel unnecessary restless under these restricted circumstances.

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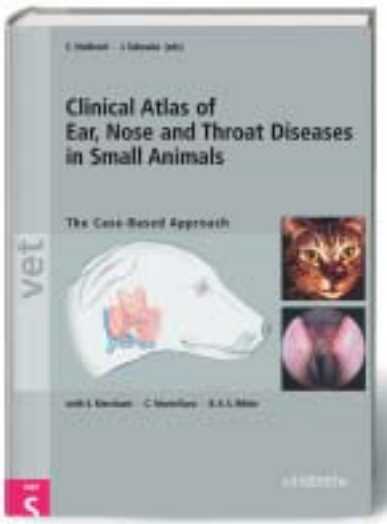

Cheryl Hedlund · Joe Taboada (eds.)

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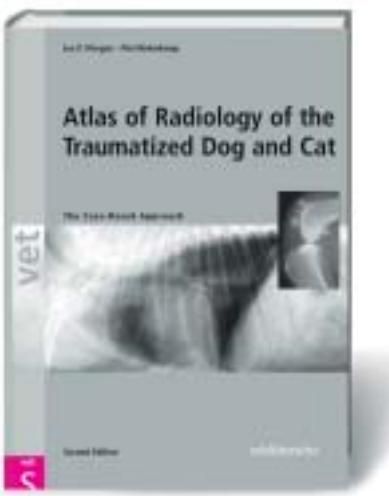

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