Atlas of
Ear Diseases of the Dog and Cat

Sue Paterson and Karen Tobias



WILEY-BLACKWELL

VetBooks.ir

ATLAS OF EAR DISEASES OF THE DOG AND CAT

VetBooks.ir

For Richard, Sam and Matt

VetBooks.ir

ATLAS OF EAR DISEASES OF THE DOG AND CAT

SUE PATERSON

&

KAREN TOBIAS

WILEY-BLACKWELL
A John Wiley & Sons, Ltd., Publication

VetBooks.ir

This edition first published 2013 © 2013 by Sue Paterson and Karen Tobias

Wiley-Blackwell is an imprint of John Wiley & Sons, formed by the merger of Wiley's global Scientific, Technical and Medical business with Blackwell Publishing.

Registered office: John Wiley & Sons, Ltd, The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK

Editorial offices: 9600 Garsington Road, Oxford, OX4 2DQ, UK
The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK
2121 State Avenue, Ames, Iowa 50014-8300, USA

For details of our global editorial offices, for customer services and for information about how to apply for permission to reuse the copyright material in this book please see our website at www.wiley.com/wiley-blackwell.

The right of the author to be identified as the author of this work has been asserted in accordance with the UK Copyright, Designs and Patents Act 1988.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by the UK Copyright, Designs and Patents Act 1988, without the prior permission of the publisher.

Designations used by companies to distinguish their products are often claimed as trademarks. All brand names and product names used in this book are trade names, service marks, trademarks or registered trademarks of their respective owners. The publisher is not associated with any product or vendor mentioned in this book. This publication is designed to provide accurate and authoritative information in regard to the subject matter covered. It is sold on the understanding that the publisher is not engaged in rendering professional services. If professional advice or other expert assistance is required, the services of a competent professional should be sought.

Library of Congress Cataloging-in-Publication Data

Paterson, Sue.
Atlas of ear diseases of the dog and cat / Sue Paterson & Karen Tobias.
p. cm.
Includes bibliographical references and index.
ISBN 978-1-4051-9326-9 (hardcover : alk. paper) 1. Dogs–Diseases–Atlases.
2. Cats–Diseases–Atlases. 3. Ear–Diseases–Atlases. I. Tobias, Karen M. II. Title.
SF991.P375 2013
636.089'78–dc23

2012010185

A catalogue record for this book is available from the British Library.

Wiley also publishes its books in a variety of electronic formats. Some content that appears in print may not be available in electronic books.

Figures 1.15, 1.16, 1.24, 1.37, 1.38, 1.39b, 1.41, 7.1 and 7.23 were illustrated by Samantha Elmhurst.
Cover design by hisandhersdesigns.

Set in 10/13 pt Minion by Toppan Best-set Premedia Limited

1 2013

VetBooks.ir

CONTENTS

Foreword vii
Preface ix
Acknowledgements xi

1 ANATOMY OF THE EAR 1
1.1 External Ear: Pinna and Ear Canal 1
1.2 Middle Ear of the Dog 5
1.3 Middle Ear of the Cat 11
1.4 Inner Ear 15

2 DIAGNOSTIC TECHNIQUES 23
2.1 Ear Pinna 23
2.2 External Ear Canal 32
2.3 Middle Ear 35

3 ADVANCED DIAGNOSTIC TECHNIQUES 37
3.1 Video Otoscopy 37
3.2 Radiography 38
3.3 CT 43
3.4 MRI 44
3.5 Brainstem Auditory Evoked Response 46
3.6 Auditory Tympanometry 49

4 DISEASES OF THE EAR PINNA 51
4.1 Erythematous Diseases 51
4.2 Diseases with Crust and Scale 55
4.3 Papular Diseases 63
4.4 Pustular Diseases 67
4.5 Nodular Diseases 70
4.6 Ulcerative Diseases 72
4.7 Diseases with Alopecia 81

5 CONDITIONS OF THE EAR CANAL 87
5.1 Allergy 87
5.2 Endocrine Disease 87
5.3 Ectoparasites 88
5.4 Keratinization Disorders 90
5.5 Autoimmune Disease 90
5.6 Idiopathic Disease 90
5.7 Foreign Bodies 91
5.8 Neoplastic and Hyperplastic Conditions of Ear Canal 93
5.9 Predisposing Factors in Otitis Externa 97
5.10 Perpetuating Factors 100

6 CONDITIONS OF THE MIDDLE EAR 107
6.1 Aetiology and Pathogenesis of Acute Otitis Media 107
6.2 Chronic Otitis Media 115
6.3 Investigation of Otitis Media 115

7 DISEASES OF THE INNER EAR 125
7.1 Vestibular Apparatus 125
7.2 Cochlea 136

8 SURGERY OF THE EAR 145
8.1 Pinna Lacerations 145
8.2 Pinnectomy 146
8.3 Auricular Haematoma Drainage 146
8.4 Lateral Wall Resection 148
8.5 Vertical Ear Canal Resection 151
8.6 Total Ear Canal Ablation with Lateral Bulla Osteotomy 154
8.7 Subtotal Ear Canal Ablation with Lateral Bulla Osteotomy 157
8.8 End-To-End Anastomosis 158
8.9 Ventral Bulla Osteotomy 160
8.10 Laser Ear Surgery 162

Index 167

VetBooks.ir

FOREWORD

My interest in ear diseases began during my first job as a new veterinary graduate in Florida, in 1978. It seemed as if almost every other dog that I saw had ear disease. In those days, otic medications were limited and ear problems rarely came under control easily.

Over the last 34 years, my interest in trying to understand ear disease has led me to write 2 ear disease books, lecture at over 350 veterinary conferences worldwide, help develop diagnostic and therapeutic tools, and teach veterinarians to use video otoscopy in their practices. I have had the honor of lecturing with Sue Paterson and in my opinion, she has an amazing way of captivating an audience with such an obscure topic as ear diseases.

The evolution of improved otic diagnostics allows us to actually see and work in the ear canal much easier than in the past. We are now able to identify tumors and middle ear disease, which are quite prevalent in both dogs and cats. New treatments of ear diseases in the dog and cat based on scientific evidence help our patients recover faster. Our pet owning clients are grateful to us for the comfort of their pets.

Through a collaborative effort, many veterinarians both in academia and in private practice have chosen to pursue their interest in veterinary otology bringing new ideas, exciting research, new drugs, and new surgical techniques, such as laser surgery, to the practice of otology. In their important contribution to the veterinary literature, Sue Paterson and Karen Tobias share with us a detailed atlas based upon their clinical experiences to help us understand the anatomy, physiology, and pathogenesis of ear disease. Sue Paterson also shares with us her unique knowledge as well as her experience in the area of hearing and audiology in the dog.

As our profession changes, we should strive to improve our diagnostic skills to survive in the competitive environment that exists presently. Learning about ear disease, acquiring the knowledge and the skill necessary to do good ear work serves to increase the value of you personally as an animal healer. Proper ear disease treatment will solidify your clients to your veterinary practice. The value of otology cannot be overemphasized in today's small animal practice.

Louis N. Gotthelf, DVM
Montgomery, Alabama USA

VetBooks.ir

PREFACE

It has been suggested that ear cases may make up more than 10% of the typical companion animal veterinarian's case load, so the ability to manage ear disease well is essential for the primary care veterinarian. Otology is one of the most rapidly expanding branches of small animal medicine and surgery and although there are many excellent textbooks already available on this subject, this is the first – and I hope one of the most complete – illustrated atlases of ear disease. I am grateful that Wiley-Blackwell has indulged my fascination for ear disease by allowing me to compile it. Ear disease for me is a passion: not only is it the challenge to diagnose and treat the dogs and cats that present to me every day in the clinic but also the challenge to pass on knowledge to fellow professionals, so that the care of ear disease in the species we deal with can be improved. The pain that otitis can cause is in my opinion often underestimated. Frequently it is only after medical therapy has resolved disease, or radical surgery has removed an ear canal, that owners can recognise how much discomfort their pet has been in, as they perceive dramatic changes in their dog's or cat's behaviour. Ear disease should never go untreated; even with the financial constraints placed on us by many of our clients we have a duty to make animals with otitis comfortable.

Working with Karen Tobias, a true goddess of surgery, has been a joy and an honour and to be able to include her enormous wealth of surgical experience in this book has enhanced it hugely. I recognize that she has spent hours preparing pictures and text that will hopefully help guide inexperienced clinicians through basic techniques and provide detailed explanation for the more experienced surgeons of the more complex procedures. I know she has enjoyed preparing her sections of the book as much as I have mine. Thanks, Karen.

Sue Paterson

VetBooks.ir

ACKNOWLEDGEMENTS

There are numerous people who have helped and guided me throughout my dermatological career; however, the person who must take the most responsibility for kindling my interest in ear disease is Craig Griffin, a true master of his art and an inspirational teacher. Thanks must go to my two business partners, Ian and Duncan who as surgeons have indulged my dermatological madness for years. I hope all the dermatology nurses at work, strictly alphabetically Bernie, Charlotte, Emma and Lydia, know how much I appreciate them; without their hard work my life would be impossible. And of course Janie my practice manager and friend, she knows I can't manage without her. Finally a huge thanks to all the loyal referring veterinary surgeons who provide me with one of the most rewarding day jobs you could ask for and of course all the material for the book.

Sue Paterson

Doing a total ear canal ablation and bulla osteotomy in a greasy Cocker Spaniel is a lot like cleaning the bathroom – the procedure is not always pleasurable, but the product is much more tolerable. Writing can be like that as well – the impetus to start the process is not always present, and sometimes the distractions prove stronger than the will to persist with the task at hand. In the end, though, the promise of a useful, attractive and educational text provides the drive needed to get the job done. I am grateful to Sue Paterson for providing that promise – who else but a dermatologist would be so excited about greasy Cocker Spaniels? I would also like to acknowledge my administrators and co-workers at University of Tennessee College of Veterinary Medicine for supporting me in this endeavour. Special thanks to photographers Phil Snow and Greg Hirshoren for their contributions and Deb Haines for her contract work and finalization of the images. And, as always, a big thanks to my two talented and attractive offspring, Jacob and Jessica, for their love and encouragement. Ears to you!

Karen Tobias

VetBooks.ir

CHAPTER 1
ANATOMY OF THE EAR

Karen Tobias

1.1	External Ear: Pinna and Ear Canal	1	1.3	Middle Ear of the Cat	11
1.2	Middle Ear of the Dog	5	1.4	Inner Ear	15

1.1 EXTERNAL EAR: PINNA AND EAR CANAL

The *pinna* is the most prominent portion of the external ear (Fig. 1.1). It has an inner, concave surface and an outer, convex surface. In the standing ear, the concave surface forms a conchal cavity that is directed rostrally or laterally, while the convex surface faces medially or caudally. The distal tip of the pinna is called the *apex*, and the lateral and medial free margins of the pinna are called the *helix* (Fig. 1.2). The rostrolateral boundary of the distal portion of the ear canal is called the *tragus*. A notch caudal to the tragus, the *intertragic incisure*, separates it from the *antitragus*, which is a thin elongated piece of cartilage that extends up to the lateral margin of the helix at the *cutaneous marginal pouch*.

The margins of the pinna are divided into medial, or rostral, and lateral, or caudal (see Fig. 1.1). These variations in directional description can make the anatomy very confusing.

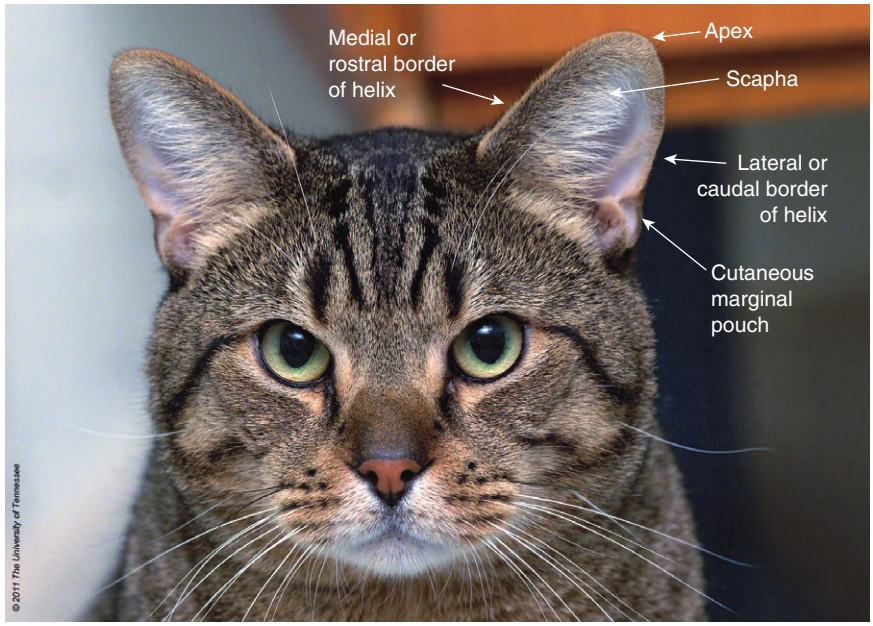


Figure 1.1 General anatomy of the pinna. The conchal cavity of the concave surface of the ear can be directed rostrally or laterally. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

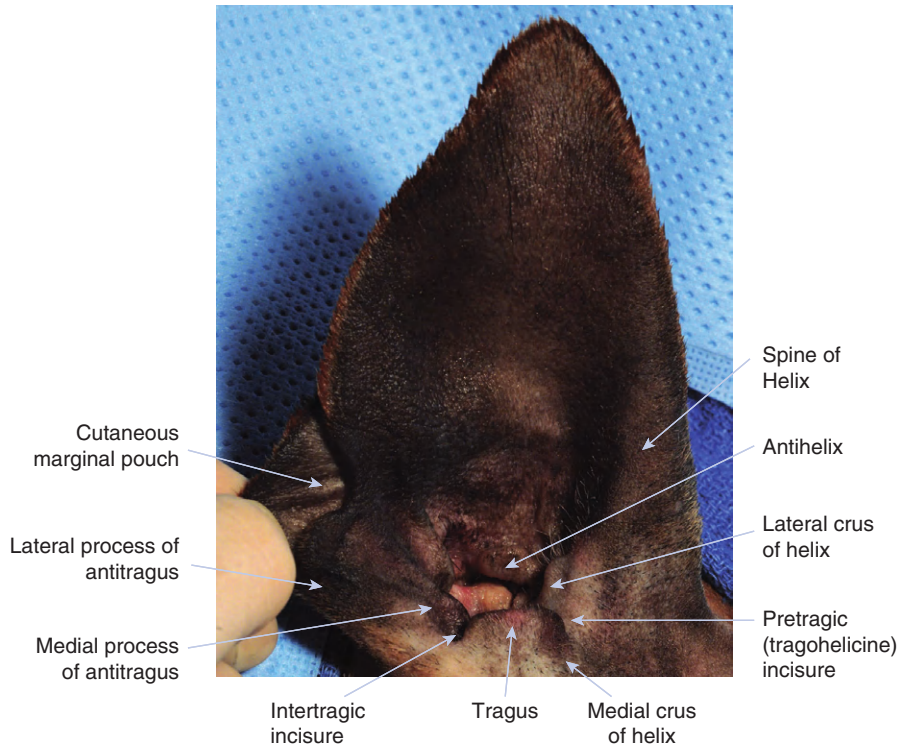


Figure 1.2 Concave surface of the right dog pinna. The antihelix and tragus form the boundaries of the ear canal opening. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

The external ear is composed of three cartilages: *annular*, *auricular*, and *scutiform*. The ear canal is formed proximally (near the skull) by the annular cartilage and distally (away from the skull) by the auricular cartilage, which fans out to form the pinna (Fig. 1.3).

The auricular cartilage is divided into three sections: the *scapha*, the *concha*, and the *tubus auris*, or conchal tube (Fig. 1.4). Whereas the scapha is distally located and flattened, the concha is rolled into a trumpet shape to form the conchal cavity (Fig. 1.5). The scapha and concha are divided on the concave surface by the *antihelix*, a transverse cartilaginous fold.

The concha forms a funnel shape that thickens proximally as it becomes the *conchal tube*. The conchal tube forms the *vertical ear canal*. This canal is up to an inch (2.5 cm) deep and, as it progresses proximally towards the head, is directed ventrally, medially, and slightly rostral, spiralling inwards. It is partially surrounded along its proximal lateral border by the parotid salivary gland.

The annular cartilage is a separate, rolled, cartilaginous band that fits inside of the base of the conchal tube. It forms the *horizontal ear canal*, which runs medially toward the skull. In turn, the annular cartilage overlaps the osseous external acoustic meatus. Junctions of the auricular and annular cartilages and the annular cartilage and skull are connected by a fibrous tissue sheath. Because of these moveable joints, the auditory canal can be straightened during otoscopic examination. Epithelium lining the auricular and annular cartilage contains sebaceous and ceruminous glands and hair follicles (Fig. 1.6).

Terminology for the ear canal varies within and amongst texts. Some authors consider the osseous extension of the skull that encompasses the tympanic membrane to be the *external acoustic meatus* or *osseous external acoustic meatus*, while others consider the external acoustic meatus to be the opening of the conchal tube at the level of the tragus and antihelix. The cartilaginous tube that extends from the meatus to the concha, which is a combination of conchal tube (auricular) and annular cartilage, is sometimes called the *auditory canal*.

A variety of muscles attach the ear rostrally, ventrally, or caudally to the head (Fig. 1.7); these muscles are innervated by the facial nerve. Some of these muscles are continuous with the cervical portion of the platysma. The plate-like, L-shaped scutiform cartilage, which is medial to the auricular cartilage, lies within the muscles that attach the auricular cartilage to the head (Fig. 1.8). By acting as a fulcrum, the scutiform cartilage improves mobility of the auricular cartilage.

The major portion of the blood supply to the external ear comes from the caudal auricular artery, which arises

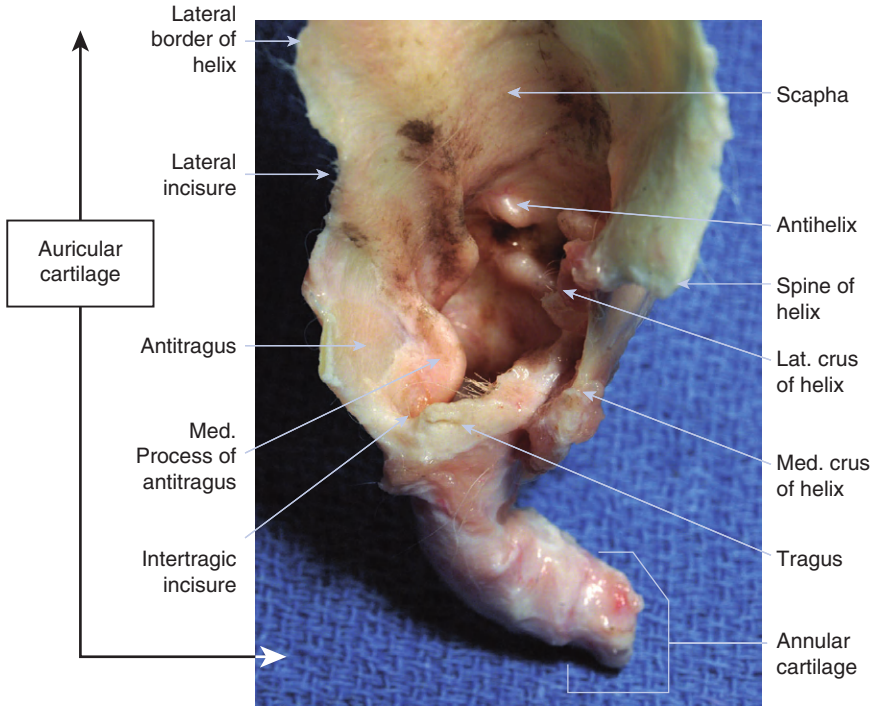


Figure 1.3 Auricular and annular cartilage of the right ear of a dog, lateral view. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

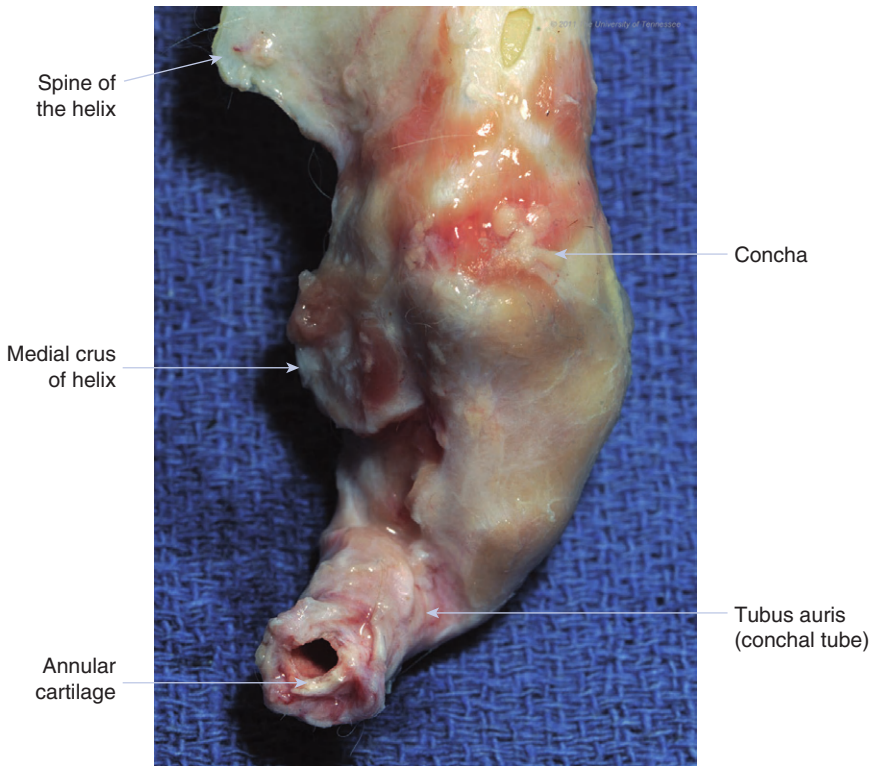


Figure 1.4 Auricular and annular cartilage of the right ear, caudal view. The annular cartilage is nestled within the auricular cartilage, which forms the pinna and vertical ear canal. Note that the proximal portion of the auricular cartilage spirals inward as it bends. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

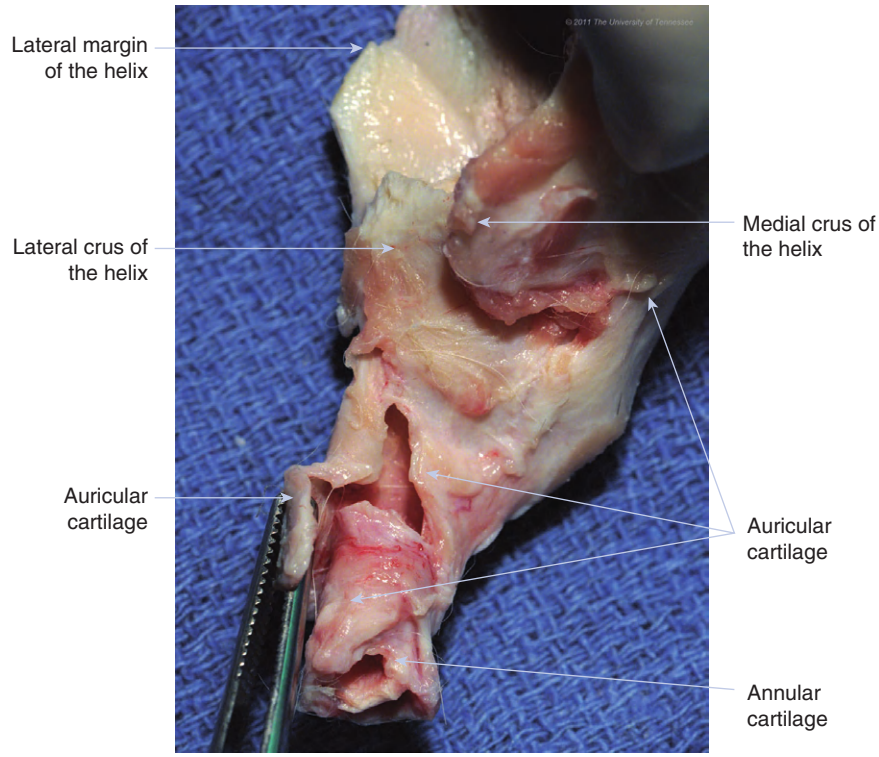


Figure 1.5 Medial view of cartilage of the right ear. A portion of the auricular cartilage that forms the conchal tube has been elevated; underneath is another extension of auricular cartilage that wraps around the annular cartilage. Note that the ear canal is not a solid funnel: the auricular and annular tubes are each formed by overlapping flaps of cartilage that allow flexibility. Animals with severe otitis externa or conchal obstruction may develop periauricular abscesses from disruption of the fibrous connective tissue sheath surrounding either the tube flaps or the auricular-annular or annular-osseous junction. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

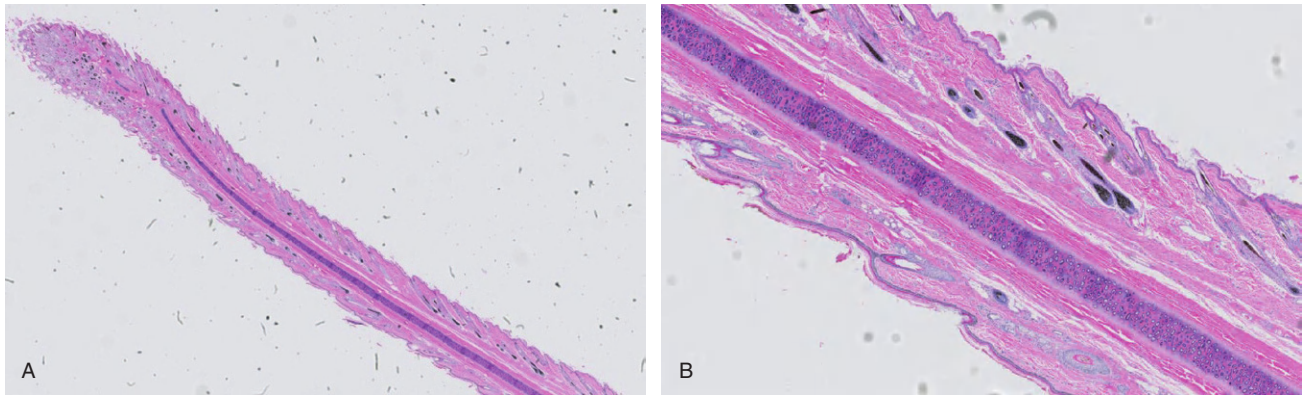


Figure 1.6 **(A)**, Cross-section of the pinna of a dog. **(B)**, Components including hyaline cartilage, muscle, and hair follicles are easily visible. (Courtesy, UTCVM Virtual Microscope) © 2012 The University of Tennessee.

from the external carotid artery at the base of the annular cartilage and medial to the parotid salivary gland (Fig. 1.9). The caudal auricular and superficial temporal veins, which terminate at the maxillary vein, provide drainage of the external ear (Fig. 1.10). Perforations in the auricular cartilage permit passage of blood vessels and nerves from the convex to the concave surface.

Sensory innervation to the concave surface of the pinna is provided primarily by branches of the facial nerve (Fig. 1.11) and, at the rostral extent of the pinna, by branches of

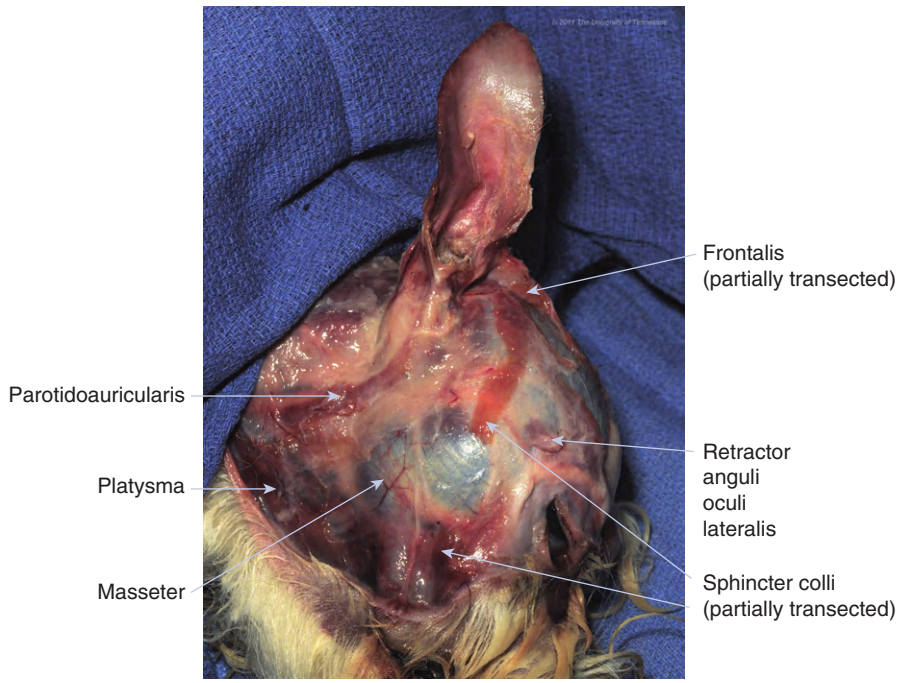


Figure 1.7 Muscles of the canine ear and face: right lateral view. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

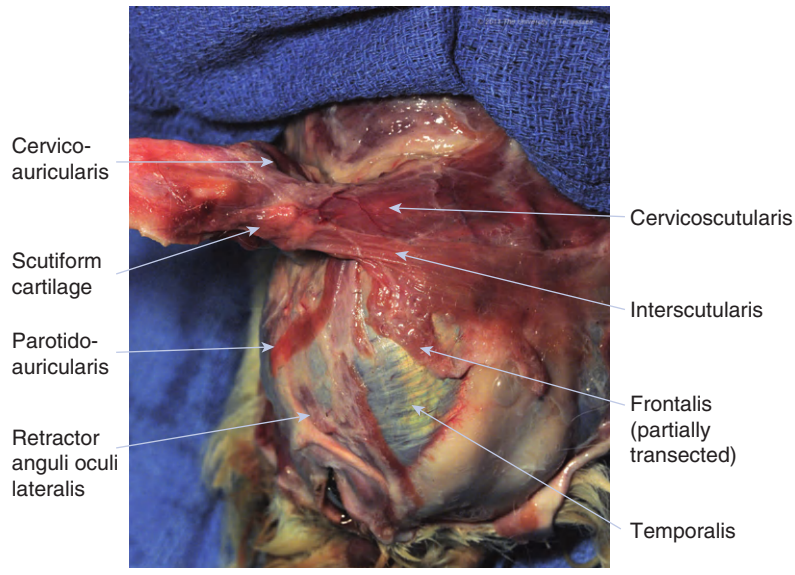


Figure 1.8 Muscles of the canine ear and head: dorsal view. The scutiform cartilage is enveloped within the dorsal group of muscles. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

the trigeminal nerve. The lateral auricular branch of the facial nerve provides sensation to the majority of the vertical canal, along with a portion of the horizontal canal, while the auriculotemporal branch of the trigeminal nerve provides sensory innervation to the horizontal canal and tympanic membrane. The convex surface of the pinna receives sensory innervation via the second cervical nerve. Communications between vagal and facial nerve branches may also be present.

1.2 MIDDLE EAR OF THE DOG

The canine middle ear (Figs 1.12 and 1.13) consists primarily of an air-filled tympanic cavity that is separated from

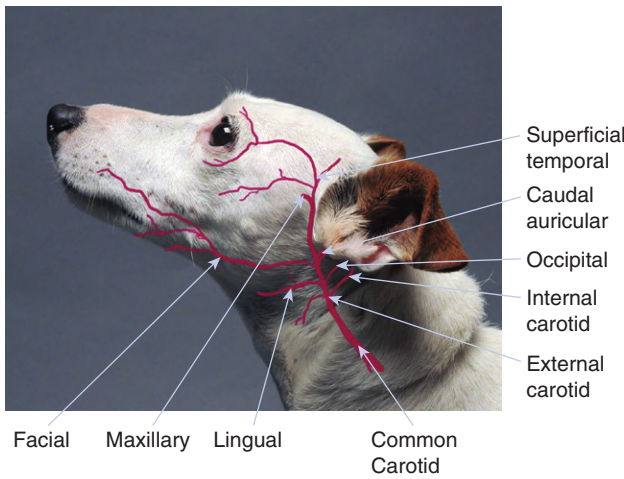


Figure 1.9 Selected branches of the common carotid artery. The external carotid artery gives off the caudal auricular artery and then travels around the ventral and rostral aspects of the horizontal canal before terminating in the maxillary and superficial temporal arterial branches. (Background photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

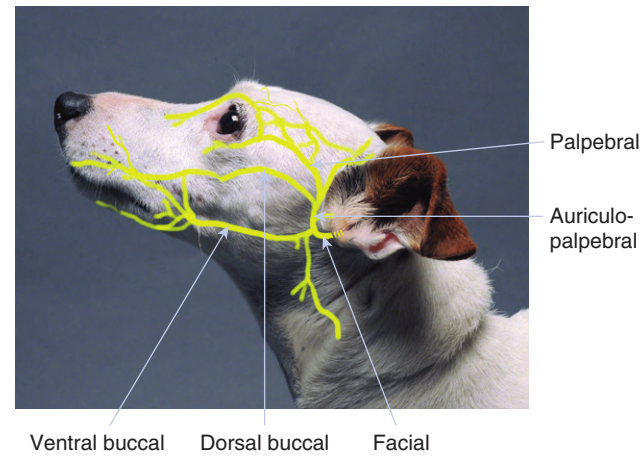


Figure 1.11 Selected branches of the facial nerve. After exiting the stylomastoid foramen, the facial nerve travels near the caudal, ventral, and rostral aspects of the horizontal ear canal. (Background photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

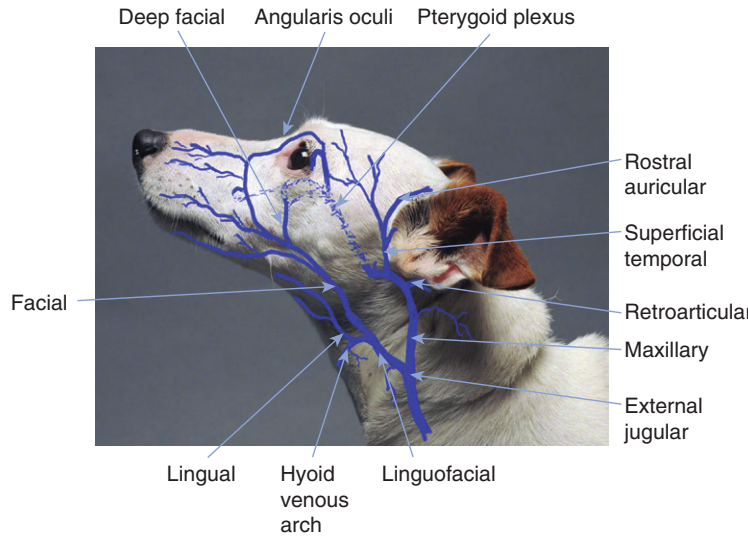


Figure 1.10 Selected tributaries of the external jugular vein. The superficial temporal vein travels ventrally around the rostral aspect of the horizontal canal and then joins the maxillary vein, which lies ventral to the canal. (Background photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

the external ear by the tympanic membrane and from the inner ear by the vestibular and cochlear windows. The middle ear is divided into three parts: (1) a large, ventral *tympanic bulla* within the temporal bone; (2) a small, dorsal *epitympanic recess*, which sits above the level of the tympanic membrane; and (3) the *tympanic cavity proper*, which connects the two and is bounded on its lateral surface by the tympanic membrane (Fig. 1.14).The tympanic cavity proper is partially separated from the ventral tympanic bulla by an incomplete septum. The tympanic cavity proper contains the cochlear (round) window along its caudal aspect. The *ossicles* of the ear – the *stapes*, *incus*,



Figure 1.12 Left lateral view of the canine skull. In this image, the mandible has been removed and the skull has been rotated slightly. Note how the bulla is less prominent than the retroarticular and jugular processes. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

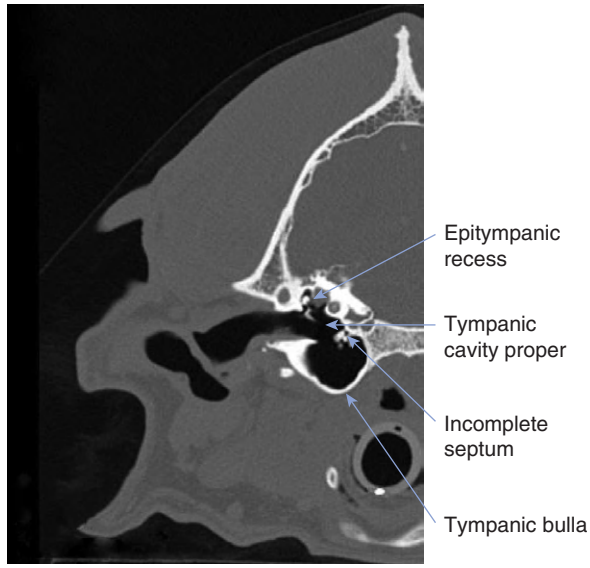


Figure 1.14 Parts of the canine middle ear. In this dog, the manubrium of the malleus is visible as an L-shaped structure, and a portion of the septum can be seen along the medial wall of the bulla. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

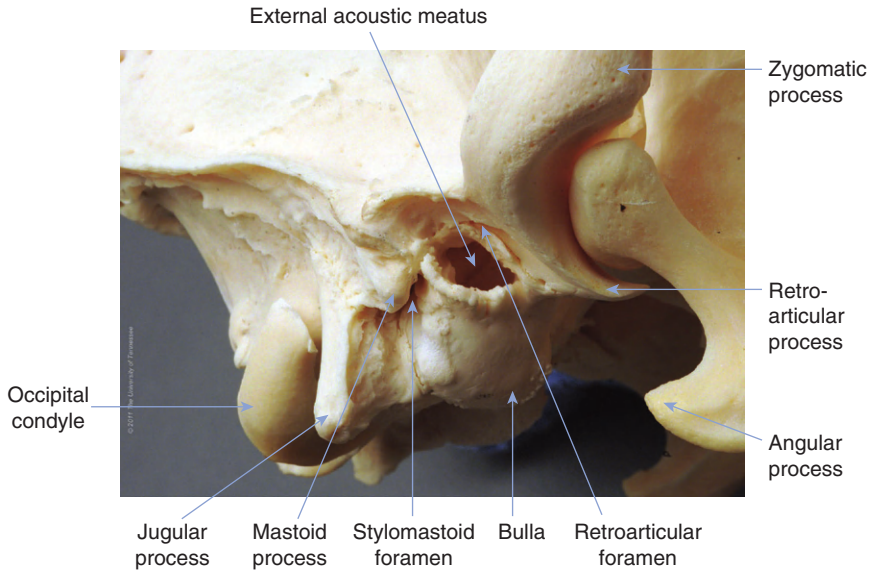


Figure 1.13 Right lateral view of the canine bulla with the mandible in place. During ventral bulla osteotomy, the position of the canine bulla is estimated by palpating the jugular and angular processes. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

and portions of the *malleus* – reside within the epitympanic recess and span the distance from the inner ear to the tympanic membrane (Fig. 1.15). The tympanic cavity is lined by simple squamous or cuboidal epithelium, except at the orifice of the auditory tube.

The *tympanic membrane* is oval in shape and concave from an external viewpoint because of medial traction by the attached malleus (Fig. 1.16). In the dog it lies at a 45° angle to the long axis of the horizontal canal, with its ventral aspect farther from view than the dorsal portion.

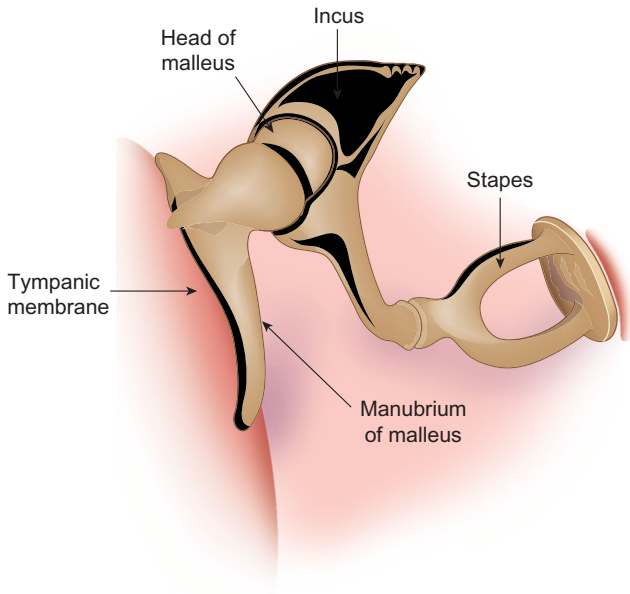


Figure 1.15 The auditory ossicles – the malleus, incus, and stapes – span the distance from the tympanic membrane to the oval window membrane.

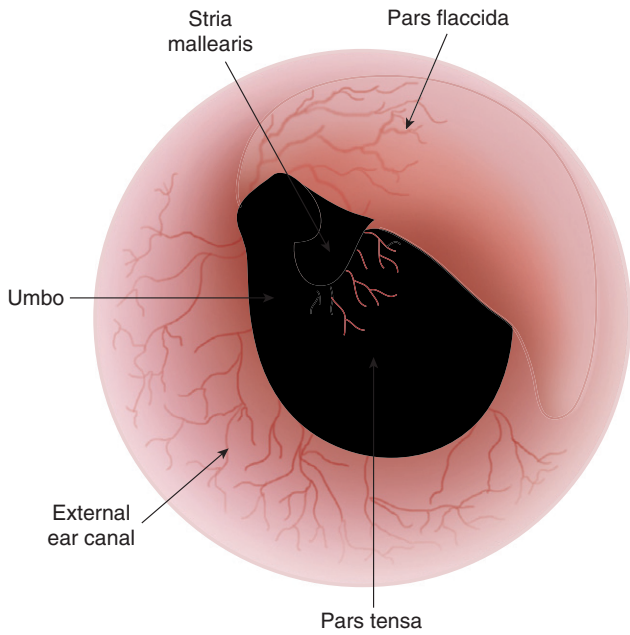


Figure 1.16 Diagram of the tympanic membrane. The tympanic membrane curves away from the external canal because of inward tension of the malleus, which attaches to it. Epithelium regenerates from the umbo outwards; this area should be avoided during myringotomy.

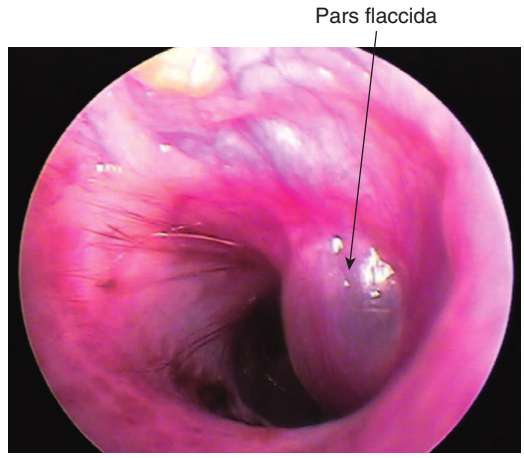


Figure 1.17 In normal dogs, the pars flaccida may be very prominent and therefore easily confused with a mass. (Courtesy, UTCVM Dermatology Service) © 2012 The University of Tennessee.

The largest portion of the tympanic membrane is called the *pars tensa*, a taut, semi-transparent, fibrous membrane. The pars tensa is firmly attached to the surrounding osseous external acoustic meatus by the *annulus fibrocartilaginous*, a fibrocartilage ring. The much smaller, dorsal portion of the tympanic membrane, known as the *pars flaccida*, is loose, opaque, and richly vascularized (Fig. 1.17).

The tympanic membrane is formed by a layer of fibrous tissue covered on its external surface with stratified squamous epithelium and on its inner surface with simple squamous or cuboidal epithelium. The *manubrium* of the malleus is embedded in the fibrous layer of the membrane (see Fig. 1.16), resulting in an inward depression called the *umbo*. The tympanic membrane regenerates radially from the umbo pars tensa and becomes thicker toward its periphery. Visibility of the malleus through the pars tensa results in a white streak known as the *stria mallearis*.

The incus and the head of the malleus almost entirely fill the small epitympanic recess (Fig. 1.18). The malleus has three attachments: the tympanic membrane, petrous temporal bone, and incus. The incus is suspended between the stapes and malleus, and the footplate of the stapes is attached to the membrane over the oval window. The malleus is controlled by the tensor tympani muscle, which originates in the tympanic bulla and is innervated by the tensor tympani nerve, a branch of the trigeminal nerve. Contraction of the tensor tympani muscle makes the tympanic membrane more rigid. The stapedius muscle also originates in the tympanic bulla. It inserts on the stapes

and is innervated by the stapedial branch of the facial nerve. The stapedius muscle contracts reflexively with loud noise, decreasing movement of the stapes to protect the ear from damage.

The *promontory* is a bony eminence on the dorsomedial wall of the tympanic cavity that houses the cochlea (Fig. 1.19). The promontory lies opposite of the tympanic membrane and medial to the epitympanic recess. The *cochlear (round) window* is found in the caudolateral portion of the promontory and opens to the perilymph in the scala tympani of the cochlea. The cochlear window is covered with a thin, secondary tympanic membrane that oscillates to dampen vibrations within the cochlear perilymph. The *vestibular (oval) window* lies on the dorsolateral surface of the promontory and is covered by a thin membrane to which the foot of the stapes is attached. Facing the vestibular window is a slit-like opening into the facial canal, through which the facial nerve travels.

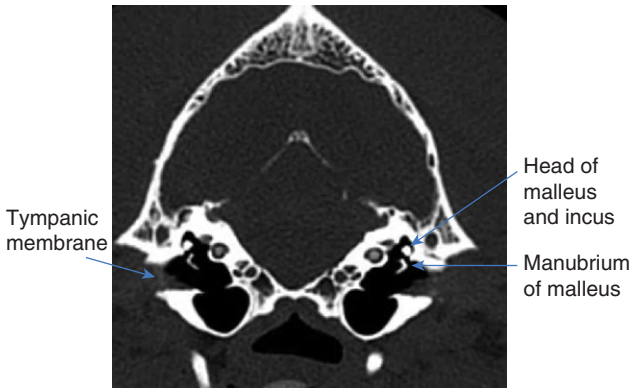


Figure 1.18 Transverse CT image of a dog skull showing the position of the malleus and tympanic membrane. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

The *auditory* or *eustachian tube* connects the tympanic cavity to the nasopharynx (Fig. 1.20). It is oval in shape, 5–15 mm long, and 1–3 mm in diameter. The auditory tube begins as a short osseous tube – a canal through the temporal bone – that exits the skull rostromedial to the bulla as the *musculotubal canal* (see Fig. 1.19). Within the tympanic cavity, its proximal ostium can be seen on the rostral surface of the tympanic cavity proper. The distal end of the tube is supported by a narrow cartilaginous trough and opens on the lateral wall of the nasopharynx, dorsolateral to the soft palate at its midpoint.

The auditory tube functions to equalize pressure across the tympanic membrane. Its distal end can be actively

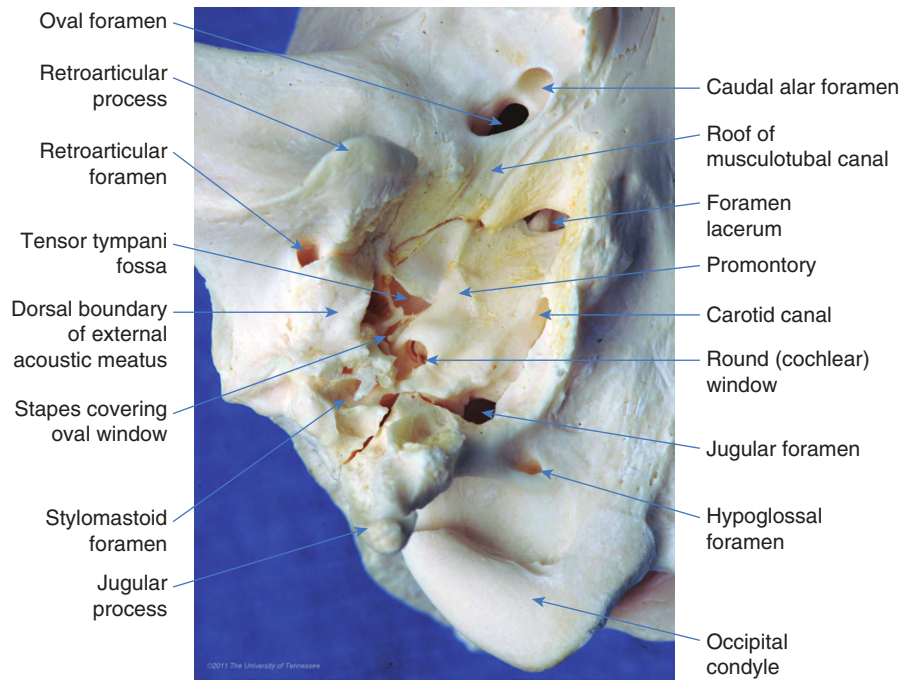


Figure 1.19 Ventral view of the right caudal canine skull with the tympanic bulla removed. The vestibular, or oval, window is on the dorsolateral surface of the promontory and just rostral to the cochlear (round) window. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

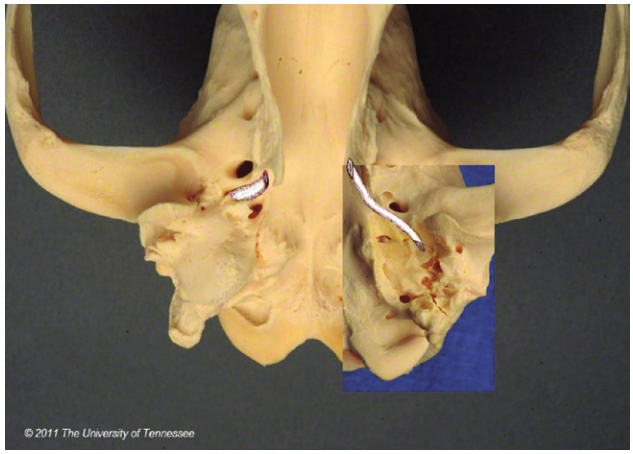


Figure 1.20 Ventral view of the caudal half of the canine skull (mandible removed). The auditory tube originates from the dorsolateral wall of the bulla (inset), just rostral to the tensor tympani muscle attachment, as the musculotubal canal. The distal end opens rostrally on the dorsolateral wall of the nasopharynx, just medial to the ipsilateral pterygoid process, or hamulus. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

opened through tension by the tensor veli palatini muscle but otherwise remains closed because of surface tension caused by contact between air and mucus. Like the respiratory tract, the auditory tube is lined by ciliated pseudostratisfied columnar epithelium containing goblet cells.

The tympanic cavity is closely associated with several nerves and vessels that can become damaged with middle ear disease or surgical trauma (Fig. 1.21). The sympathetic postganglionic nerves to the eye and orbit are collectively called the *internal carotid nerves*. In the dog they travel along with the internal carotid artery through the carotid canal, which is separated from the tympanic cavity by a thin bony plate.

The *facial nerve* travels through the sigmoid-shaped *facial canal* of the petrous temporal bone. The facial canal is an incomplete tunnel that opens into the tympanic cavity lateral to the vestibular window. The facial nerve leaves the petrous part of the temporal bone through the *stylomastoid foramen*.

The *chorda tympani*, a branch of the facial nerve, travels medial to the base of the malleus in the epitympanic recess. It is sometimes called the *tympanic nerve*. The chorda

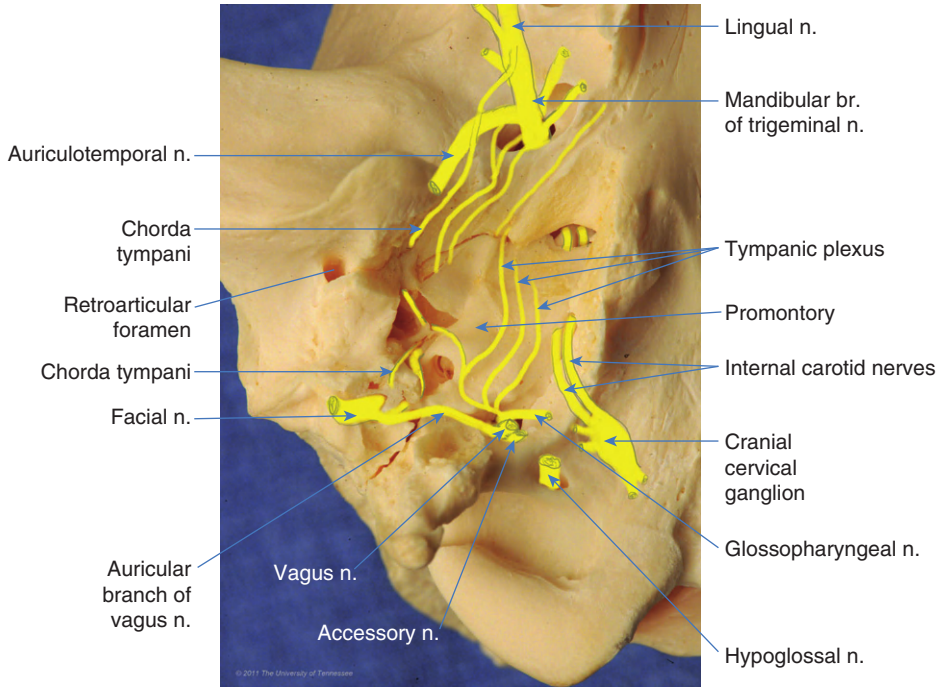


Figure 1.21 Ventral view of the canine skull (right bulla removed) with diagram of regional nerves. The facial nerve travels through the facial canal, an S-shaped trough in the epitympanic recess, and exits at the stylomastoicd foramen just caudal to the external acoustic meatus. The retroarticular vein, which is the termination of the temporal sinus, exits from the retroarticular foramen, while the internal carotid artery travels under a thin shelf of bone with the internal carotid nerves. The jugular process serves as the caudal attachment site of the digastricus muscle. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

tympani provides innervation to the mandibular and sublingual salivary glands and fungiform papillae on the rostral two thirds of the tongue. Ipsilateral papilla may atrophy if the nerve on the affected side is damaged from otitis media.

The *tympanic plexus* lies on the promontory and is formed primarily by fibres from the tympanic branch of the glossopharyngeal nerve (cranial nerve IX). The tympanic branch of the glossopharyngeal nerve supplies innervation to the lining of the tympanic bulla, providing pressure and pain sensation, and to the parotid and zygomatic salivary glands.

The *auriculotemporal nerve*, a branch of the mandibular nerve, passes medial and caudal to the retroarticular process of the temporal bone and emerges between the base of the auricular cartilage caudally and masseter muscle cranial. One of its branches – the *external acoustic meatus nerve* – is sensory to the external acoustic meatus near the tympanic membrane. Another branch – the *rostral auricular nerve* – supplies the skin over the lateral aspect of the tragus and a rostroventral portion of the pinna's concave surface.

The *internal carotid artery* (Fig. 1.22) enters the *jugular foramen* and travels with the sympathetic fibres of the internal carotid nerve via the *tympano-occipital* or *petro-occipital fissure* into the middle ear, where it travels through the *carotid canal* (Fig. 1.23). It exits the canal at the rostromedial edge of the tympanic bulla at the *foramen lacerum*. The nearby petro-occipital canal transmits the ventral petrosal venous sinus. Axons of the glossopharyngeal (IX), vagus (X), and accessory (XI) nerves also pass through the jugular foramen and travel in the tympano-occipital fissure.

Beyond its bifurcation with the internal carotid artery, the *external carotid artery* travels rostrally, forming an S shape (see Fig. 1.22) that traverses from medial to lateral over the base of the bulla and then rostrally under the external acoustic meatus. It gives off several branches, including the caudal auricular and superficial temporal arteries. The caudal auricular artery circles the caudal half of the ear at the base of the annular cartilage. The superficial temporal artery is found at the rostral extent of the base of the auricular cartilage.

1.3 MIDDLE EAR OF THE CAT

As in the dog, the ossicles in the cat rest high up within the epitympanic recess (Fig. 1.24). Cats have a much less

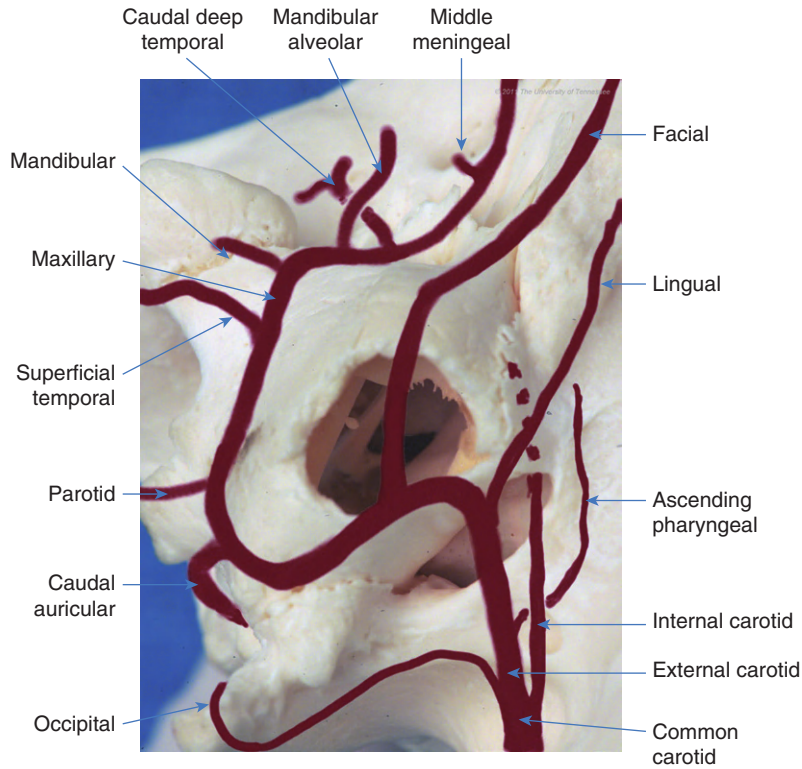


Figure 1.22 Relationship of selected arteries with the right bulla of a dog. In this view, a ventral bulla osteotomy has been performed and the manubrium is visible. The internal carotid artery passes through the tympano- (or petro-) occipital fissure, travels within the carotid canal, and passes out and then back into the foramen lacerum before travelling on to the brain. (Background photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

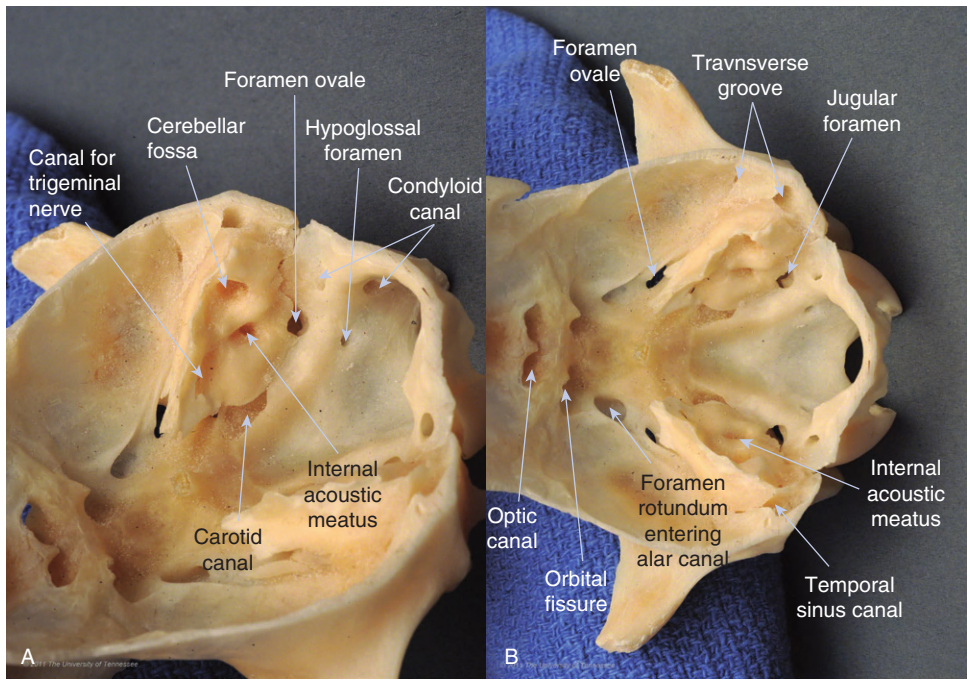


Figure 1.23 Dorsolateral (**A**) and dorsal (**B**) views of the open canine skull. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

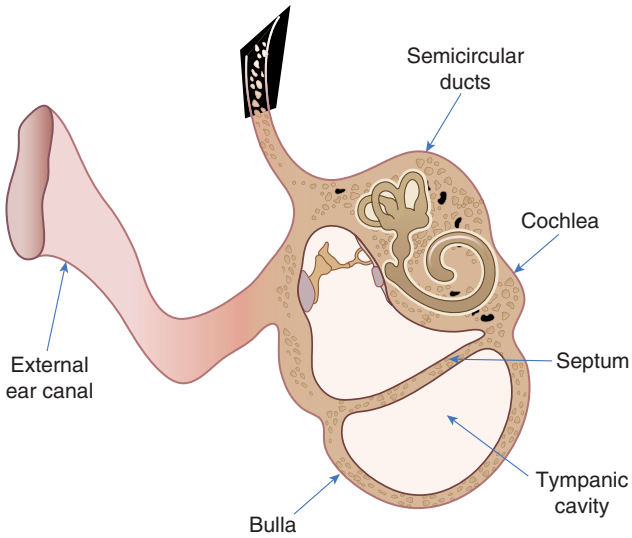


Figure 1.24 As in the dog, the ossicles of the cat lie within the epitympanic recess and extend from the tympanum to the oval window. A bony septum separates the bulla into two cavities.

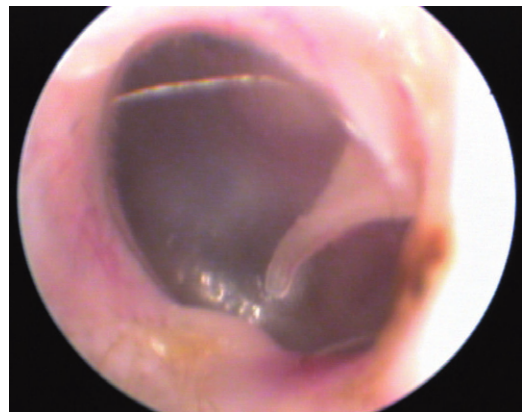


Figure 1.25 The manubrium in this cat is easily visible through the tympanic membrane. (Courtesy, UTCVM Dermatology) © 2012 The University of Tennessee.

prominent pars flaccida, so the manubrium is more easily visible through the tympanic membrane (Fig. 1.25). The bulla is more prominent in cats that in dogs, making it easier to locate by palpation during ventral bulla osteotomy (Fig. 1.26). In cats, the tympanic cavity is divided into two compartments by a thin bony septum (Fig. 1.27) that runs from a midrostral to a midlateral position (Fig. 1.28). The dorsolateral compartment is the smaller of the two (Fig. 1.29). Its lateral wall is comprised primarily by the tympanic membrane, which is oriented perpendicular to the long axis of the horizontal canal. Much of the dorsolateral compartment is occupied by the auditory ossicles immediately medial to the tympanum. The opening of the auditory tube is within the rostromedial aspect of the dorsolateral

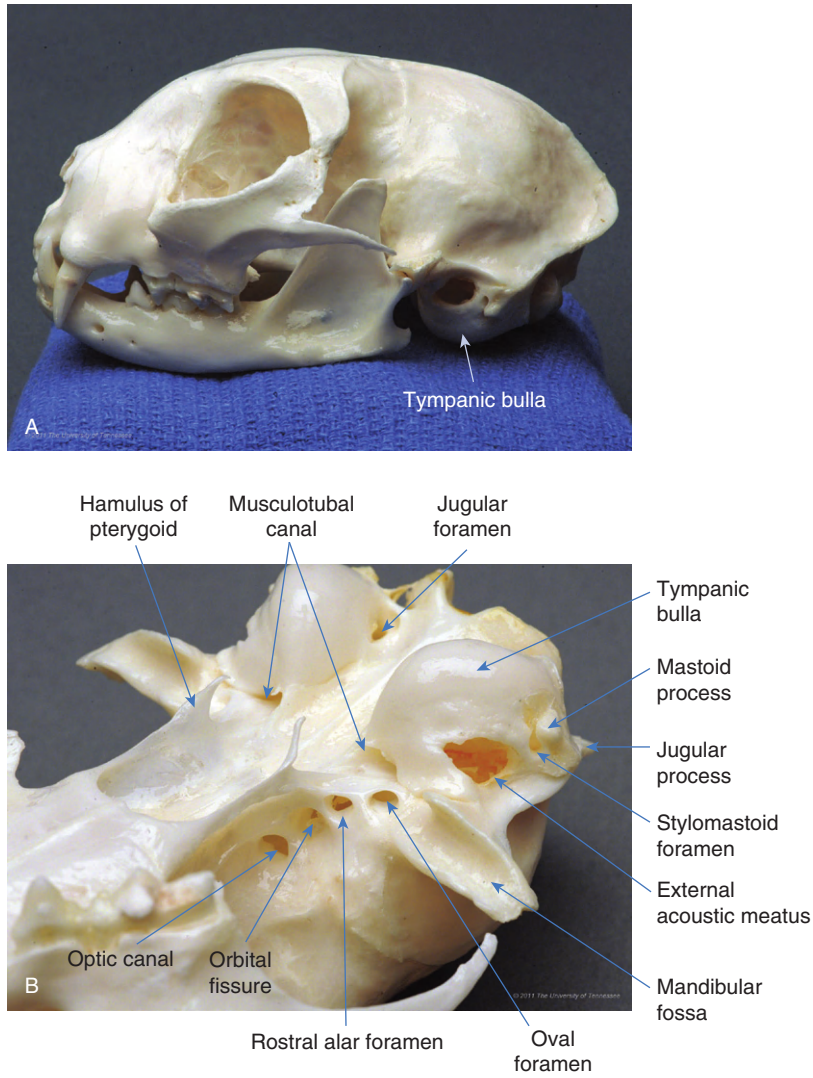


Figure 1.26 Anatomy of the cat skull. (**A**) Left lateral view. (**B**) Right ventrolateral view. The bulla extends ventral to the retroarticular and jugular processes, making it easily palpable. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

compartment. The ventromedial compartment, which also extends caudal to the dorsolateral compartment, is primarily an air-filled bulla.

The septum dividing the bulla is incomplete dorsally: a narrow fissure on the caudomedial aspect of the dorsolateral compartment permits communication with the ventromedial compartment (Fig. 1.30). The caudal end of the fissure enlarges into a triangular foramen, which is occupied along its medial wall by the cochlear (round) window, which faces laterally. The promontory on the dorsal wall of the bulla is medial to the cochlear window and extends to both sides of the septum.

The chorda tympani, which originates from the facial nerve caudal to the tympanic membrane, travels rostrally, medial to the auditory ossicles (Fig. 1.31). The origin of the auditory tube is found on the dorsomedial portion of the rostral wall of this compartment.

The postganglionic sympathetic nerves enter the tympano-occipital fissure caudal to the bulla, travelling adjacent to the internal carotid artery. The tympanic branch of the glossopharyngeal nerve joins them within the tympano-occipital fissure so that collectively they form the caroticotympanic nerves. The nerves enter the ventromedial compartment of the tympanic cavity at the caudal end of the promontory, then branch and fan out to form the tympanic plexus. The plexus lies on the exposed surface of the promontory and is thus vulnerable to trauma. Damage to these fibres results in Horner's syndrome, with signs of

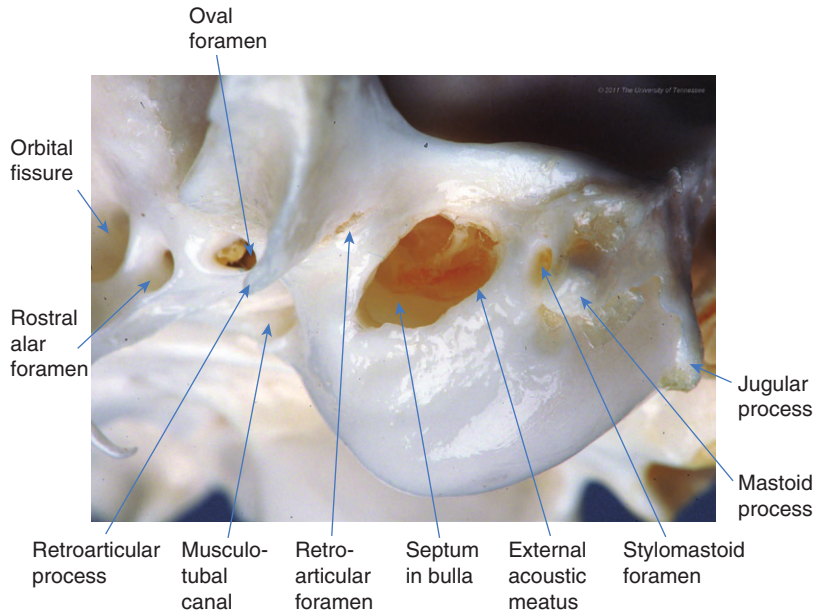


Figure 1.27 On the close-up view of the left feline bulla, the septum is visible through the osseous external acoustic meatus. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

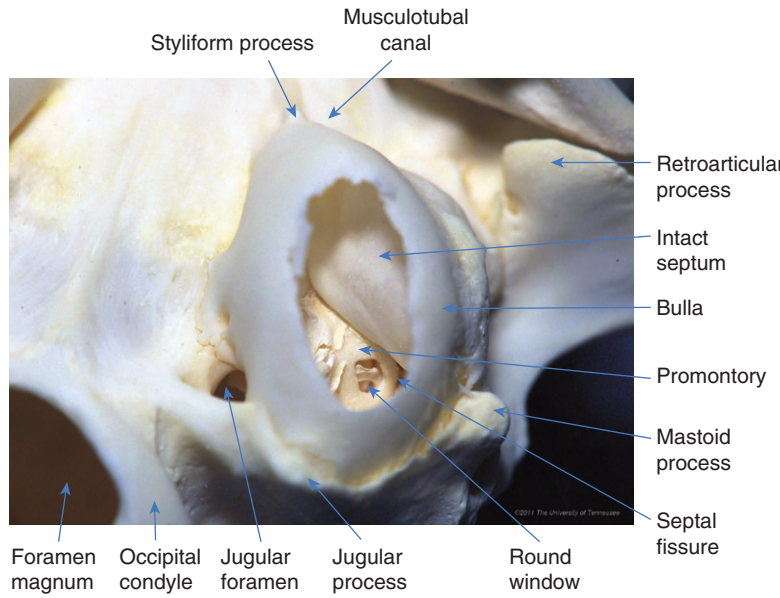


Figure 1.28 Ventral view of a cat skull: the floor of the left bulla has been removed (similar to ventral bulla osteotomy) to show the intact septum. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

meiosis, enophthalmos, and third eyelid prolapse. Along the rostral edge of the promontory the plexus fibres enter through the communicating fissure into the dorsomedial compartment, where they are somewhat protected by a ridge of bone ventral to the promontory. The sympathetic fibres of the tympanic plexus exit the middle ear through the petrous temporal bone medial to the os of the auditory (eustachian) tube (Fig. 1.32) and join the ophthalmic branch of the trigeminal nerve (V), which carries them to the eye. The auditory (eustachian) tube terminates in the dorsolateral nasopharynx just medial to the hamulus of the pterygoid bone (Fig. 1.33).

1.4 INNER EAR

The inner ear contains the sensory organs for hearing and balance (Figs 1.34 and 1.35). It resides within the petrous part of the temporal bone (Fig. 1.36) and consists of a membranous labyrinth enclosed within a bony labyrinth.

The *bony labyrinth* includes the *semicircular canals*, *cochlea*, and a central chamber known as the *vestibule* that connects the two (Fig. 1.37). The bony vestibule is divided into the *utricle* and *saccule*. The *membranous labyrinth* resides within the bony labyrinth, and its components have names similar to those of their surrounding osseous structures. They include the *semicircular ducts*, *cochlear duct*, *utriculus*, and *sacculus*. The membranous utriculus and sacculus give rise to the semicircular ducts and cochlear duct, respectively.

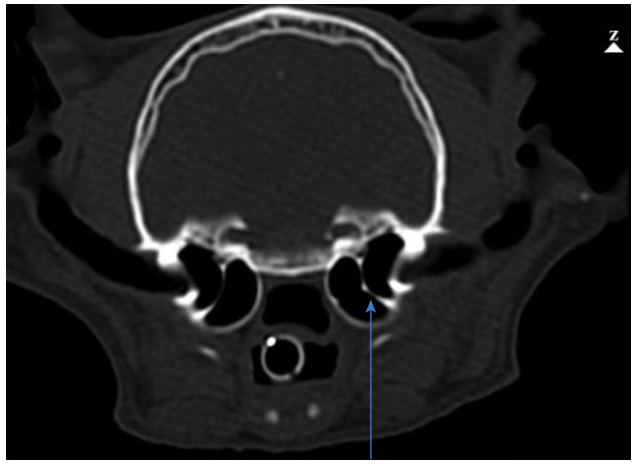


Figure 1.29 Normal cat CT scan. Note the septum (arrow) that divides the dorsolateral and ventromedial chambers. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

Fluid within the membranous labyrinth is called *endolymph*, and fluid that surrounds it is called *perilymph*. The system can be likened to an egg, in that the membranous labyrinth – the egg yolk – is suspended in a layer of perilymph within the bony labyrinth – the eggshell. The utriculus and sacculus are connected via the intervening *utriculosaccular duct*, which permits endolymph flow between the cochlear and semicircular ducts and therefore between the organs for hearing and balance.

The *cochlear aqueduct* connects the perilymphatic space of the scala tympani with the subarachnoid space (Fig. 1.38). Via the *perilymphatic duct* within the cochlear aqueduct, perilymph is continuous with cerebrospinal fluid, allowing middle ear infections to spread to the meninges. The endolymphatic duct travels through the *vestibular aqueduct*. Although the endolymphatic duct is not directly connected to the subarachnoid space, it ends in an *endolymphatic sac* that lies along the meninges in the epidural space.

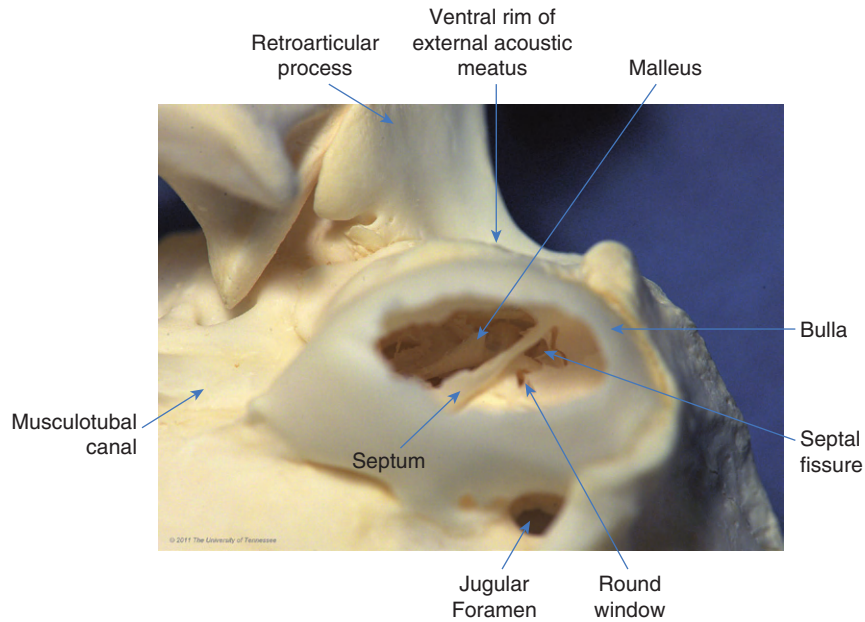


Figure 1.30 Ventral view of a cat skull showing interior of the left bulla after partial removal of the septum. A fissure in the caudodorsal aspect of the septum allows connection of the two compartments. The round window is located along the dorsal aspect of the fissure. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

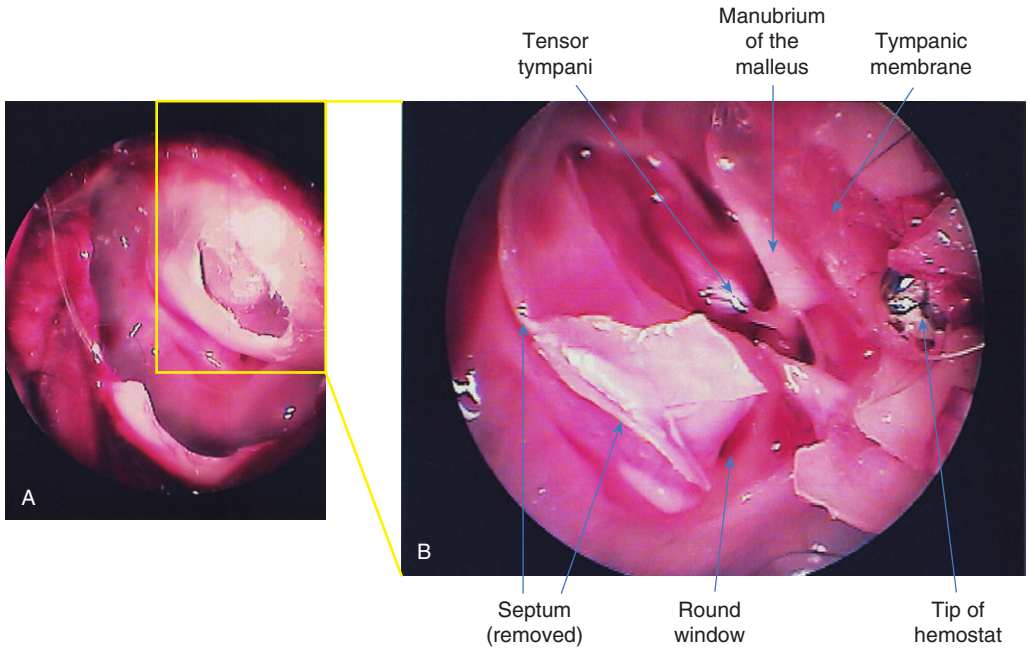


Figure 1.31 Ventral view of the left feline bulla (cadaver specimen) through a video otoscope. (**A**) The septum has been partially removed, but the inner lining is still intact. (**B**) Most of the septum and lining has been removed and the video otoscope advanced into the dorsolateral compartment. For orientation, a haemostat was inserted down the external ear canal and through the ventral portion of the tympanic membrane. The manubrium of the malleus is embedded within the dorsal half of the tympanic membrane.

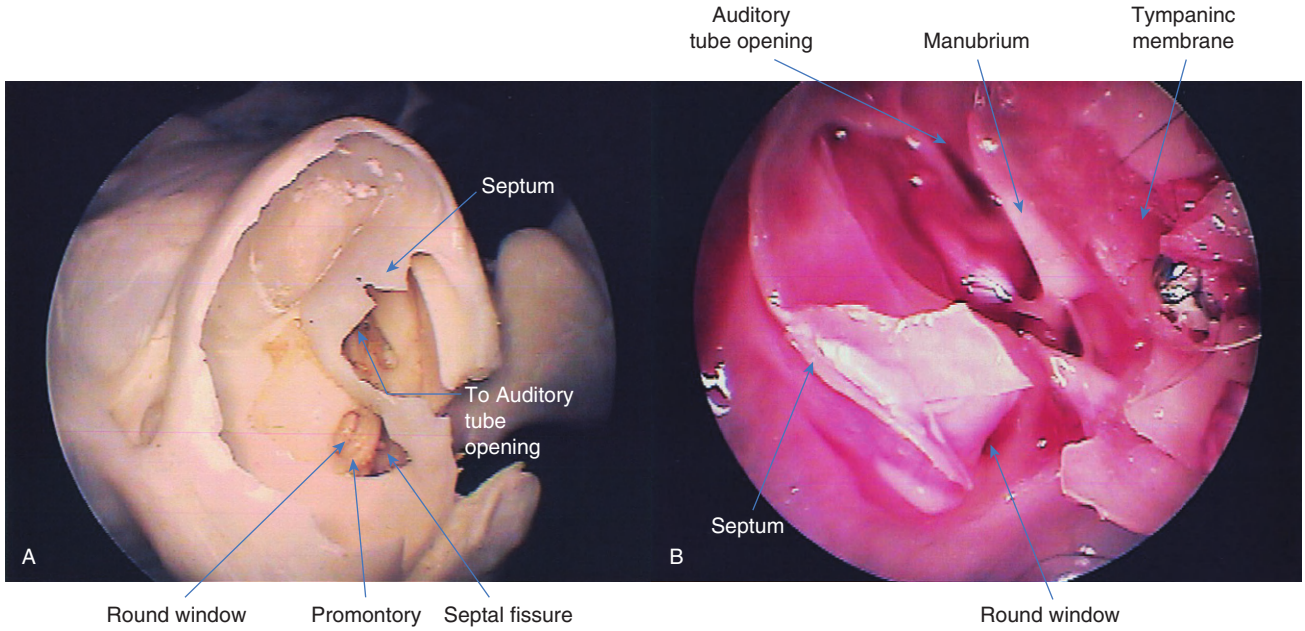


Figure 1.32 Opening of the auditory tube within the left feline bulla. The tympanic bulla has been removed and septum has been opened. The opening of the musculotubal canal is visible in the dorsolateral compartment of the cat skull (**A**) and appears as a slit-like opening in this otoscopic view (**B**) of a ventral bulla osteotomy in a cat cadaver.

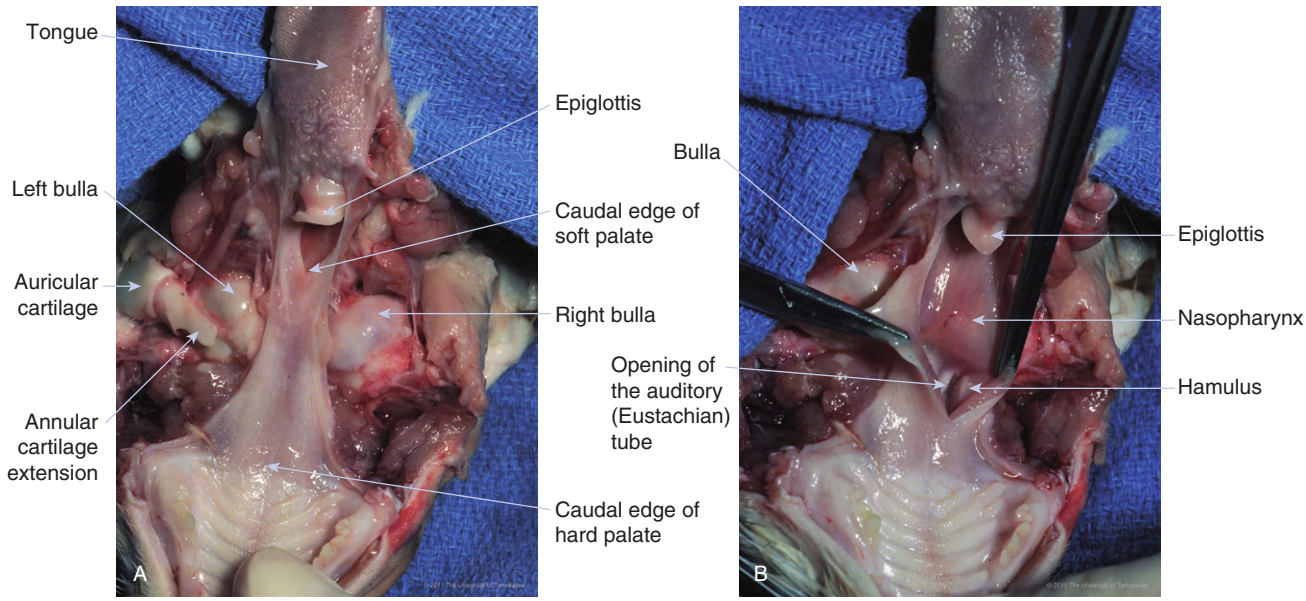


Figure 1.33 Ventral view of the cat head after removal of the mandible. (**A**) The bullae are closely associated with the nasopharynx. In the cat, the annular cartilage has a tongue-like extension that normally attaches to the ventral surface of the bulla. In this cadaver, the extension has been removed from the right side and shifted cranially on the left. (**B**) The opening of the right eustachian tube is visible once the soft palate is incised and retracted. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

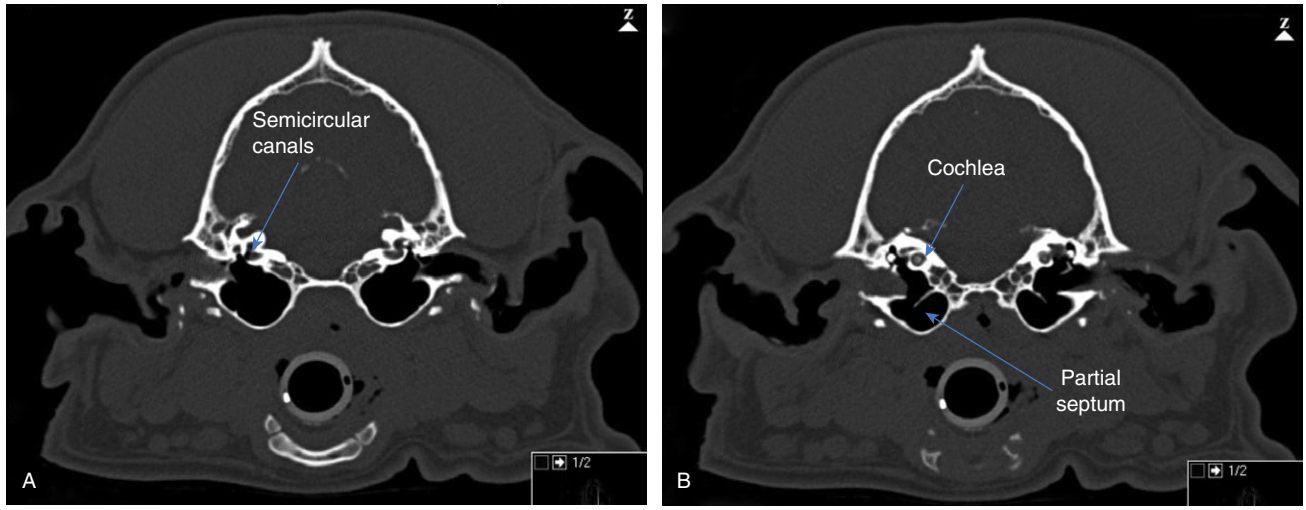


Figure 1.34 CT scan of the middle and inner ear of a dog. (**A**) The semicircular canals are visible above the right bulla, while the vestibule and caudal edge of the cochlea are visible above the left bulla. (**B**) In a more rostral image, the cochlea can be seen above the tympanic cavity proper of the right bulla, with the foot of the stapes covering the oval window. The round window is visible entering the cochlea of the left bulla. The malleus and septa are evident in both bulla. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

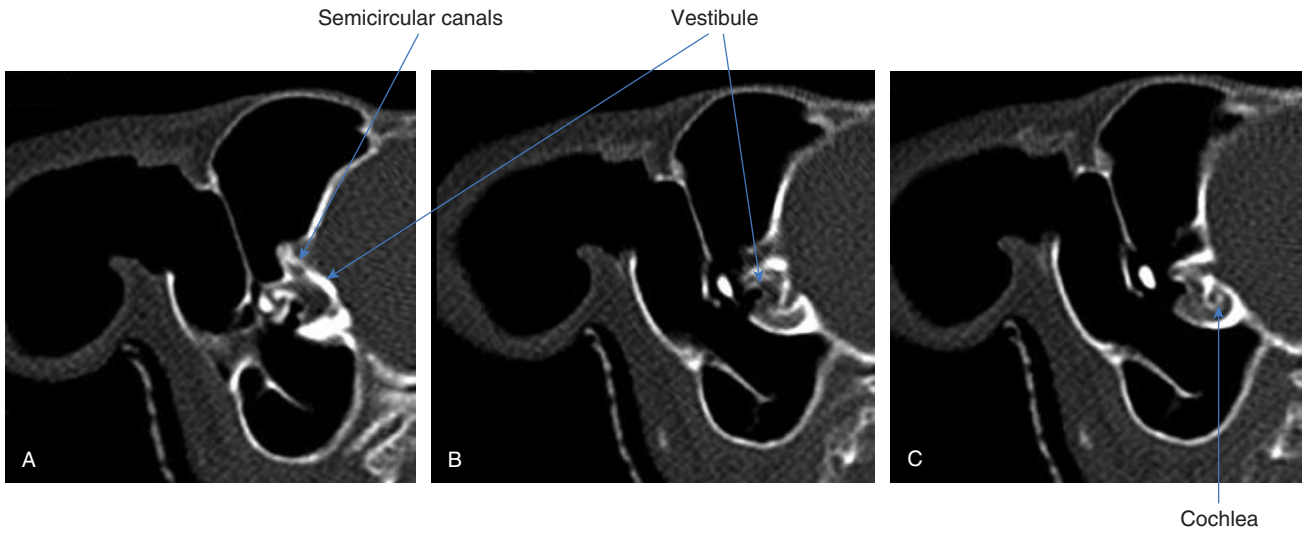


Figure 1.35 CT series of the right inner ear of a chinchilla, moving from caudal to rostral. The semicircular canals (**A**), vestibule (**B**), and cochlea (**C**) are easily detectable on CT in this species. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

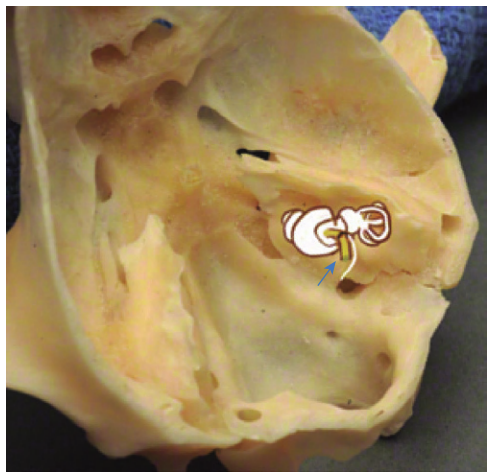


Figure 1.36 The inner ear (diagram) resides in the petrous part of the temporal bone, which is visible from the inside of the skull. The vestibulocochlear nerve (blue arrow) exits the petrous temporal bone via the internal acoustic meatus. (Background photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

1.4.1 Balance

Dogs and cats have three semicircular ducts that are oriented at right angles to one another. Each semicircular duct terminates in an *ampulla* that contains a ridge or crest called the *crista ampullaris* (Fig. 1.39). This crest is oriented transversely to the course of endolymph flow within the associated semicircular duct. Sensory cells on the crista ampullaris are covered by *cilia*, or hair cells, that are embedded in a gelatinous matrix, or *cupula ampullaris*. This matrix reacts to rotational acceleration as endolymph flows in or out of the associated duct. The resulting movements of the cilia stimulate action potentials that are carried by the vestibular nerve to the brain. Within the utriculus and sacculus are sensory fields called *macula* that are oriented in horizontal and vertical planes, respectively. Macular sensory cells are also covered with cilia that react to linear acceleration and deceleration in different directions and are responsible for the sensation of static head position.

1.4.2 Hearing

The cochlea is the primary organ involved in translation of sound waves to nerve impulses (Fig. 1.40). The cochlea spirals from dorsal to ventral around a hollow core – the *modiolus* – that contains the cochlear nerve. A shelf (*spiral lamina*) projecting from the modiolus serves as the inner attachment site to the cochlear duct, which extends all the way to the bony outer wall. The spiral lamina and cochlear duct divide the cochlea into two perilymph-filled chambers: the *scala vestibuli* and the *scala tympani* (Fig. 1.41). The foramen connecting the scala tympani and scala vestibuli at the apex or *cupulla cochleae* of the spiral is called the *helicotrema*. The cochlear (round) window is found at the end of the scala tympani, while the vestibular (oval) window is found at the vestibule near the beginning of the scala vestibuli.

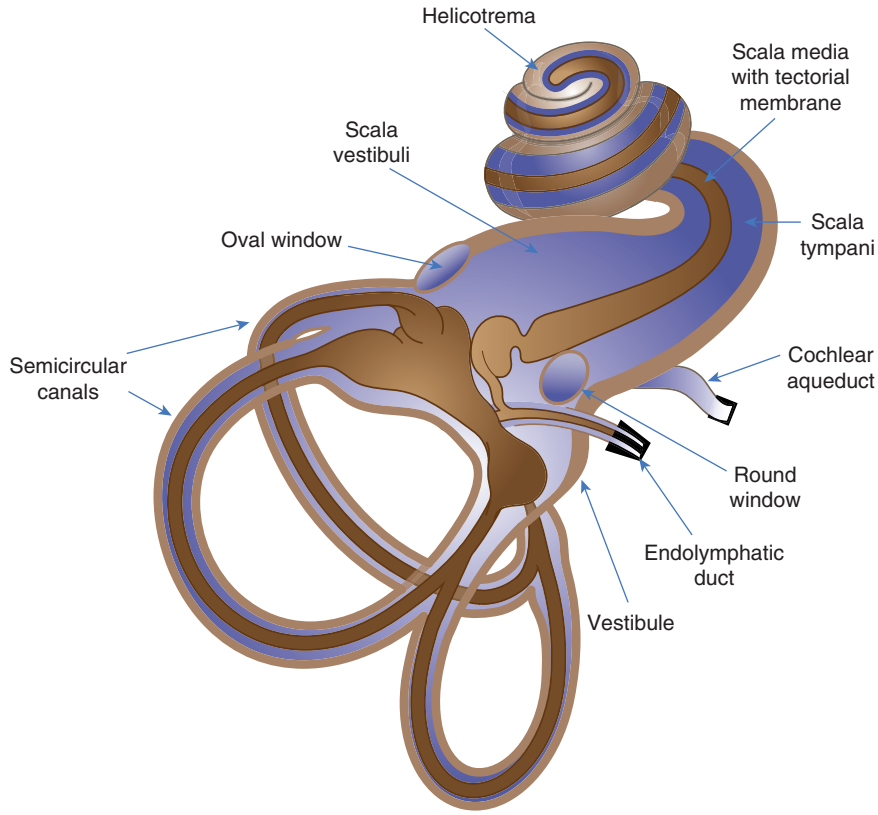


Figure 1.37 The membranous labyrinth, which contains the endolymph (brown), is surrounded by perilymph (blue), which is contained within the bony labyrinth and is continuous with cerebral spinal fluid via the cochlear aqueduct. Vibrations in perilymph propagate up the scala vestibuli to the helicotrema and back down the scala tympani. A, ampulla; S, sacculus; U, utriculus.

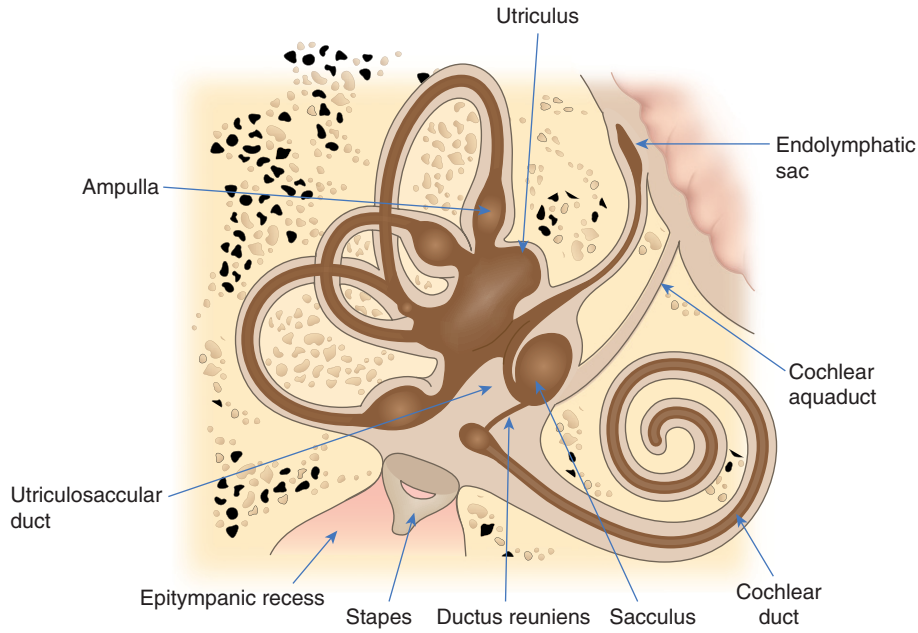
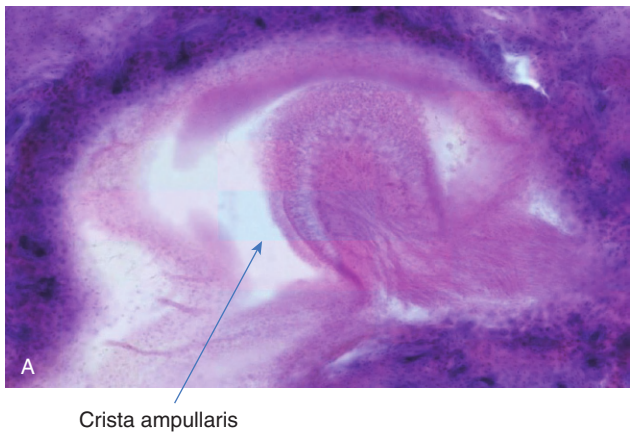


Figure 1.38 Endolymph (brown) flows into the endolymphatic sac, which lies along the meninges.



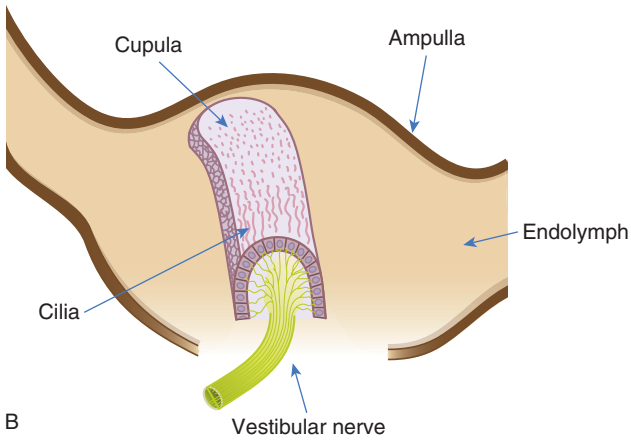


Figure 1.39 The crista ampullaris (**A**) consists of sensory cells covered by cilia that are embedded in a cupula of loose gelatinous tissue (**B**). Bending of the cupula ampullaris secondary to endolymph flow stimulates formation of action potentials that are carried by the vestibular nerve to the brain. (Specimen photo courtesy, UTCVM Virtual Microscope) © 2012 The University of Tennessee.

The endolymph-filled chamber within the cochlear duct is called the *scala media* (Fig. 1.42). The floor of the scala media is formed by the *basilar membrane*, and the ceiling is formed by *Reissner's membrane*. The wall along the outside curve of the cochlear duct is formed by the *spiral ligament*, a thickened segment of periosteum. A portion of the spiral ligament, the *stria vascularis*, contains numerous capillary loops and produces endolymph for the scala media.

The *organ of Corti* (Fig. 1.43) is a layer of specialized, thickened epithelium that spirals along the basilar membrane of the scala media. Sensory hair cells (*stereocilia*) of

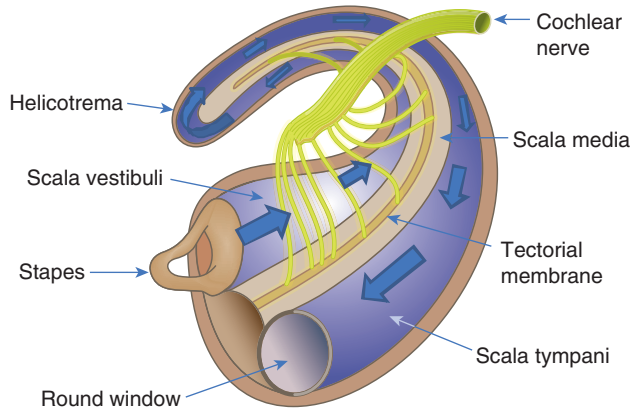


Figure 1.41 Schematic of cochlea, unrolled. Fluid waves travel up the scala vestibuli and down the scala tympani, reaching the round window, which provides a dampening effect. The waves vibrate the endolymph in the scala media. In turn, the tectorial membrane vibrates along hair cells, stimulating nerve impulses that are transmitted by the cochlear nerve to the brain.

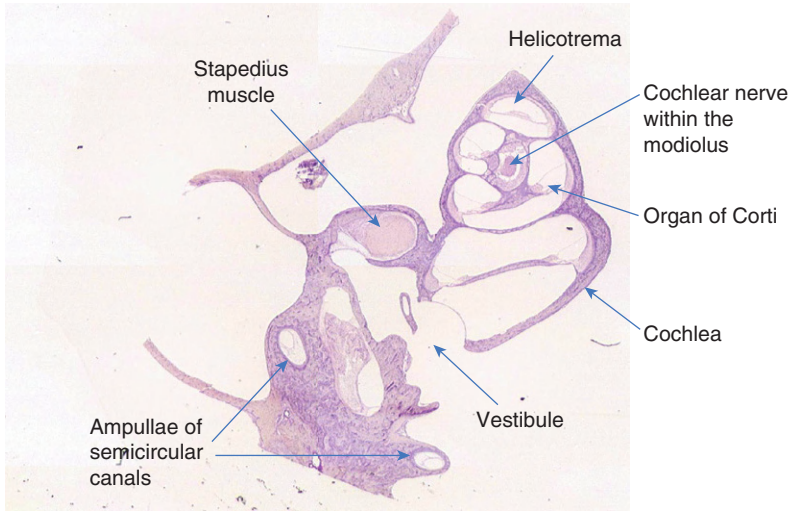


Figure 1.40 Cross-section of the inner ear of a rat showing the relationship of the cochlea, vestibule, and semicircular canals. (Courtesy, UTCVM Virtual Microscope) © 2012 The University of Tennessee.

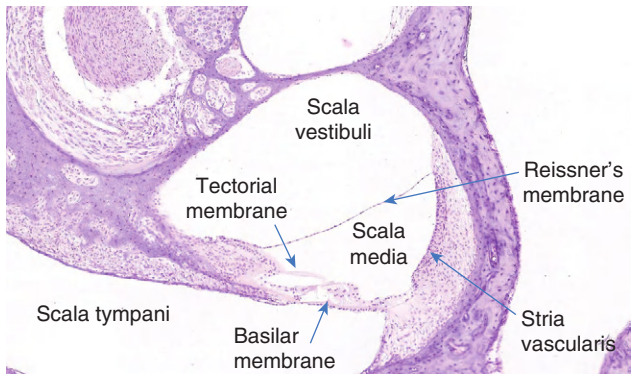


Figure 1.42 The organ of Corti, which is the sensory organ for hearing, lies along the basilar membrane. (Courtesy, UTCVM Virtual Microscope) © 2012 The University of Tennessee.

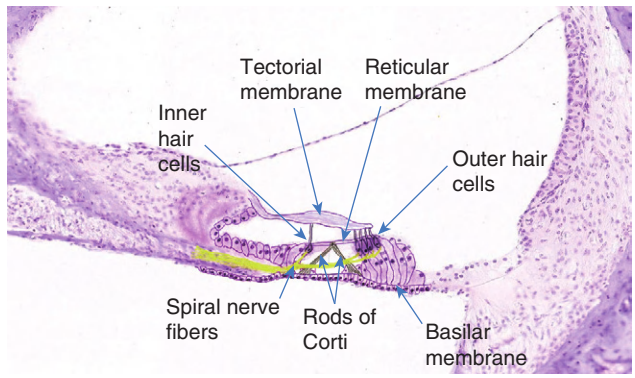


Figure 1.43 The organ of Corti consists of the tectorial membrane, hair cells, and support cells. Oscillations of the basilar membrane result in shearing forces between the stereocilia and tectorial membrane. Subsequent depolarization of the hair cells sends nerve impulses via cochlear nerve fibres to the brain.

the organ of Corti are innervated by cochlear nerve endings. The *tectorial membrane*, a leaf-like structure, lies over the stereocilia. The reticular membrane along the apex of the stereocilia is supported by the *rods of Corti*.

Sound waves collected by the pinna and external ear cause vibration of the tympanic membrane. These vibrations are transmitted via the ossicles through the vestibular (oval) window and into the scala vestibuli. Sound waves propagating through the scala vestibuli are transmitted by the intervening Reissner's membrane to the endolymph of the scala media. In turn, the tectorial membrane vibrates, touching the stereocilia. As the stereocilia bend they depolarize, initiating nerve impulses that are carried by the cochlear nerve to the brain.

VetBooks.ir

VetBooks.ir

CHAPTER 2
DIAGNOSTIC TECHNIQUES

Sue Paterson

2.1	Ear Pinna	23	2.3	Middle Ear	35
2.2	External Ear Canal	32			

A range of diagnostic techniques can be employed to investigate ear disease in the dog and cat. Many quick basic tests can be undertaken by the primary care veterinary surgeon within the confines of a normal practice laboratory. Other more advanced techniques such as radiography, CT, and MRI may also be useful in some circumstances, but the latter two are generally confined to referral institutions.

Both the ear pinna and the ear canal can yield diagnostic information and samples should be taken from both sites where possible. On the ear pinna the type of test that is suitable will depend on the clinical presentation, particularly the type of primary lesion that is present. Where samples are taken from the ear canal and middle ear a more formulaic approach is possible.

2.1 EAR PINNA

2.1.1 Basic Diagnostic Tests

For a quick summary see Table 2.1.

Diascopy

This is a useful and simple technique to assess the difference between vasodilatation and haemorrhage. A glass slide is placed over an erythematous lesion and gentle pressure is applied.

- Erythematous lesions blanch when pressure is applied, as they are caused by dilated blood vessels.
- Haemorrhagic lesions do not blanche when pressure is applied as they are caused by red blood cell leakage out of vessels (Fig. 2.1).

Table 2.1 Lesions on the ear pinna and most suitable diagnostic tests

Presenting sign	Most suitable tests	Major differential diagnoses
Erythema	Tape strips, diascopy	Allergy, malassezia, haemorrhage
Crust and scale	Superficial skin scrapes, tape strips, dermatophyte culture	Sarcoptes, dermatophytosis, endocrine disease, immune-mediated disease
Pustules	Pustule cytology, culture, excisional biopsy	Pyoderma, sterile pustular diseases (esp. pemphigus foliaceus)
Papules	Papule cytology, impression smear	Ectoparasites, pyoderma
Nodules	Fine needle aspirates, impression smear, excisional biopsy	Neoplasia, hyperplasia, infectious pyogranulomatous disease
Ulcers	Impression smears, biopsy of periphery of ulcer	Vasculitis, immune-mediated disease
Alopecia	Skin scrape, hair pluck, dermatophyte culture, biopsy	Demodex, dermatophytosis, endocrine disease

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.



Figure 2.1 Diascopy – haemorrhagic lesions do not blanche.

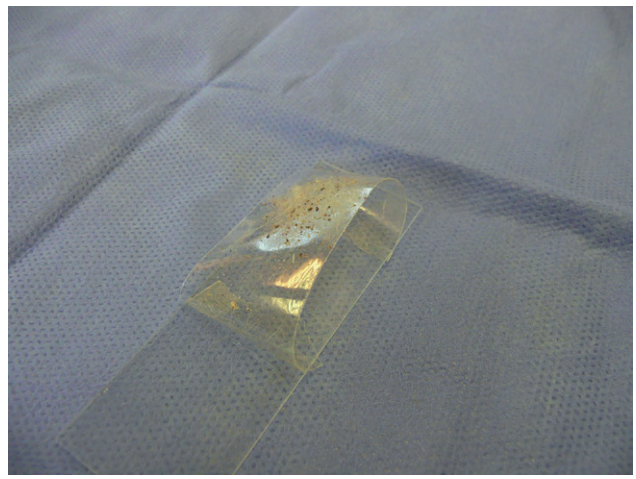


Figure 2.3 Tape is everted and stuck on to a microscope slide.



Figure 2.2 Tape is pressed firmly on to an alopecic area.

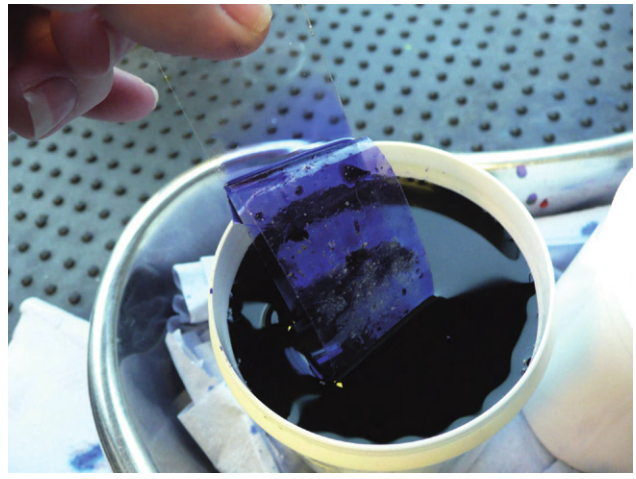


Figure 2.4 Tape is stained with a modified Wright's stain (Diff-Quik).

Acetate Tape Impression Smears
This technique can be performed on the skin or hair.

Hair
The tape is pressed repeatedly on to the coat to pick up such things as louse eggs from the hairs as well as parasites on the surface of the skin, e.g. lice, trombicula. This is a useful technique to trap some of the fast-moving parasites which can often be seen with the naked eye.

Skin
The tape is pressed firmly on to an alopecic area of skin on the ear pinna (Fig. 2.2). The author usually rubs the tape gently with a thumb nail to ensure good contact of the tape with the skin. The tape is then everted sticky side up on a

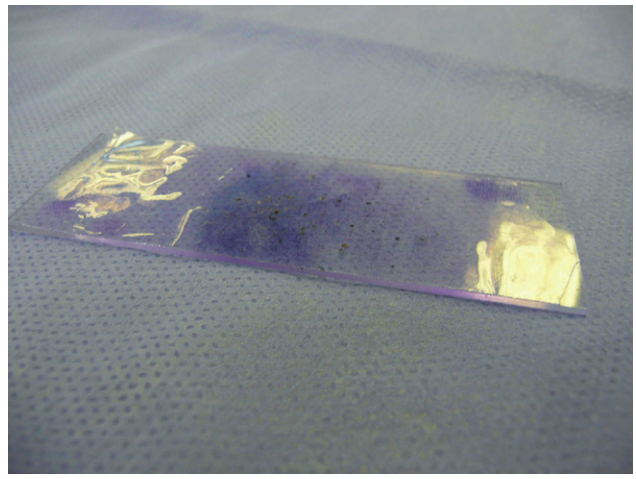


Figure 2.5 Tape is fixed to microscope slide, sticky side down, for examination.

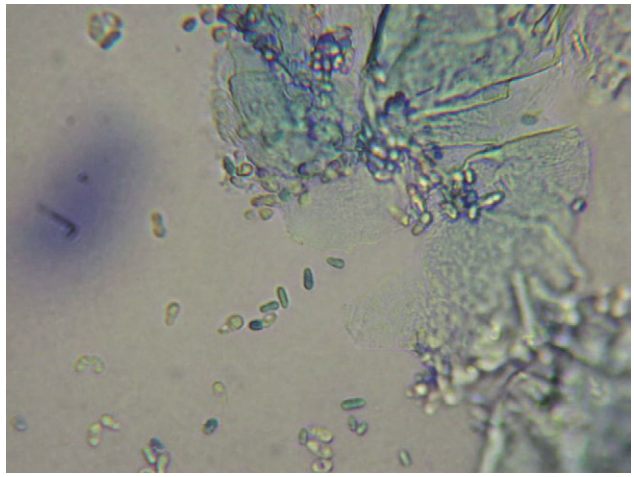


Figure 2.6 *Malassezia pachydermatis* on an acetate tape strip.

Table 2.2 Mounting material for skin scrapes – advantages and disadvantages

Liquid paraffin	Potassium hydroxide
Not irritant to skin	Potential skin irritant
Will not clear the sample	Will clear the sample by digesting keratin
Does not kill the mites which can thus be identified by movement across the slide	Mites killed
Mites will curl up if sample not examined soon after taking; identification may be difficult	Mites well preserved and observation of body parts easier

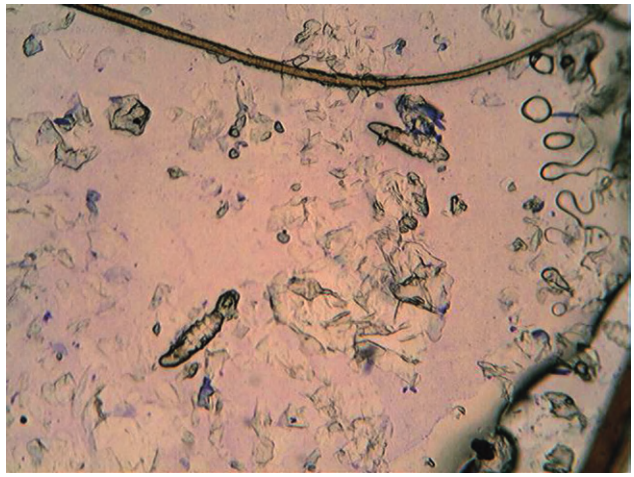


Figure 2.7 *Demodex corniei* from a tape stripping from an ear pinna.

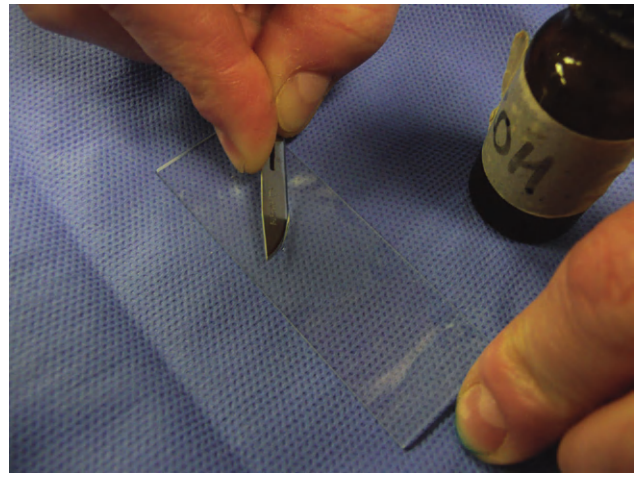


Figure 2.8 Scalpel blade is blunted before use.

microscope slide (Fig. 2.3). A modified Wright's stain such as Diff-Quik can then be applied to the tape (Fig. 2.4) which is then carefully inverted with the adhesive slide downwards on to a microscope slide for examination (Fig. 2.5). As the tape acts as a cover clip. the sample can be examined under high power (100× oil immersion) if necessary. This method can be used to identify surface bacteria; yeast, especially malassezia (Fig. 2.6); and parasites, especially surface-living demodex mites – *Demodex corniei* in dogs (Fig. 2.7) and *Demodex gatoi* in cats.

Skin Scrapings

Scrapes may be taken into either liquid paraffin or 10% potassium hydroxide. When liquid paraffin is used a few drops can be gently added to the sample before a cover slip is added. When potassium hydroxide is used the sample is best left to stand for 10–15 minutes and may be gently warmed before examining as this allow the potassium hydroxide to digest keratin to clear the field. There are advantages and disadvantages of both mounting materials (see Table 2.2).

Superficial Skin Scraping

A scalpel blade should be blunted by gently rubbing it on the microscope slide before use (Fig. 2.8). It is moistened with either 10% potassium hydroxide or liquid paraffin to provide better collection of material and scraped through the coat and superficial layers of the skin. Hair may be gently clipped away if the coat is very thick. The scalpel

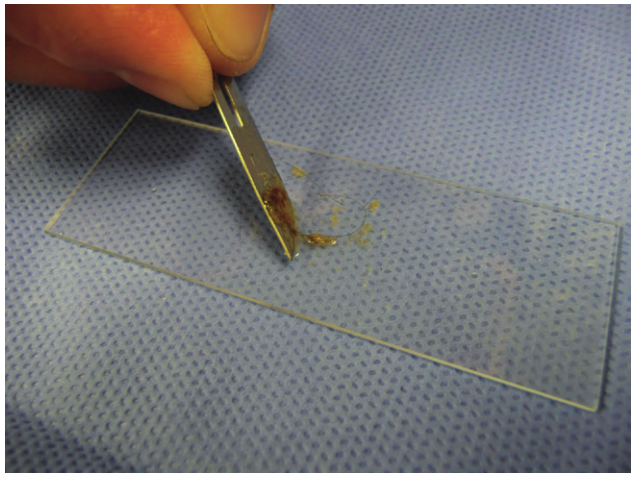


Figure 2.9 Material from the scraping is spread thinly on the slide.



Figure 2.11 *Neotrombicula autumnalis* from ear of a dog.



Figure 2.10 *Felicola subrostratus* from ear of a cat.

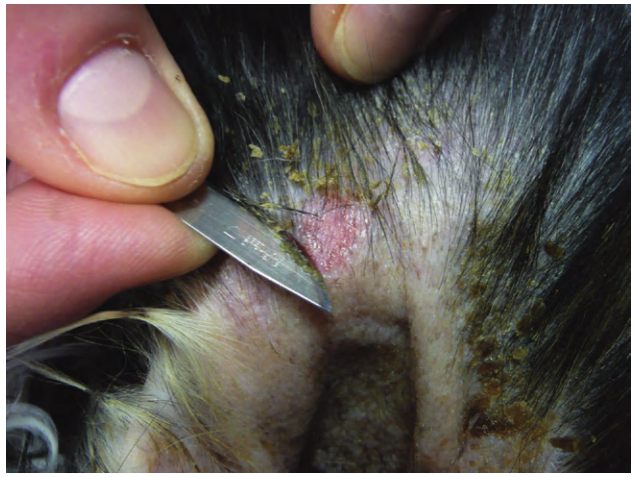


Figure 2.12 Deep skin scrapings should reveal capillary ooze.

blade is normally held perpendicular to the skin and the scraping should be in the direction of the hair coat. Material should be spread thinly on to the slides (Fig. 2.9). Multiple samples should be taken from non-excoriated areas. When potassium hydroxide is used then the slides may be gently warmed to break up keratin and/or left to stand for 10–15 minutes before examination. This technique is best employed for superficial parasites such as lice (Fig. 2.10) and trombicula (Fig. 2.11).

Deep Skin Scraping

The technique is identical to that for superficial scrapings except that scrapings should be to a taken to a depth where the skin is erythematous and mild capillary ooze is seen (Fig. 2.12). Where scrapings are taken to investigate sarcoptes they should be taken from the periphery of the ear pinna from areas of crusting (Fig. 2.13). Typical round-bodied mites, eggs (Fig. 2.14), and faeces can be found. Where follicular demodex (*Demodex canis or Demox cati*) is suspected then scrapes should be taken from the glabrous areas of the pinna, if possible from areas of comedone formation. The skin may be pinched to express the mites from the follicles.

Hair Plucking/Trichography

Hair plucks are a useful way to assess a variety of different factors including the presence of fungal infection, shaft defects, and hair growth phase. To take a sample, hair should be grasped firmly between the finger and thumb

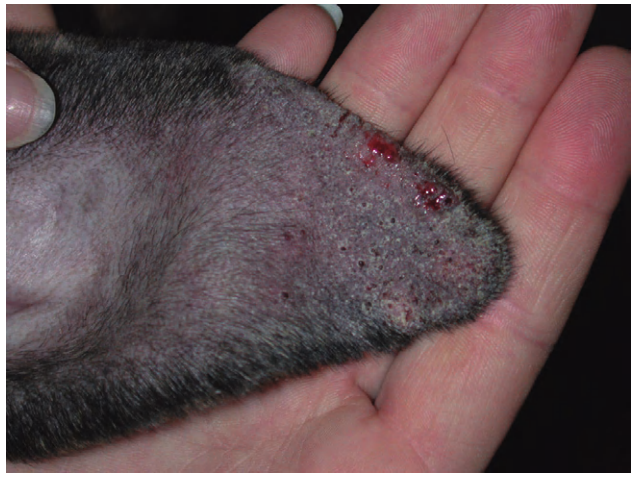


Figure 2.13 Position of deep skin scraping from the periphery of ear pinnae for sarcoptes.



Figure 2.15 Hair plucked from hair pinnae may be mounted in 10% potassium hydroxide for examination.

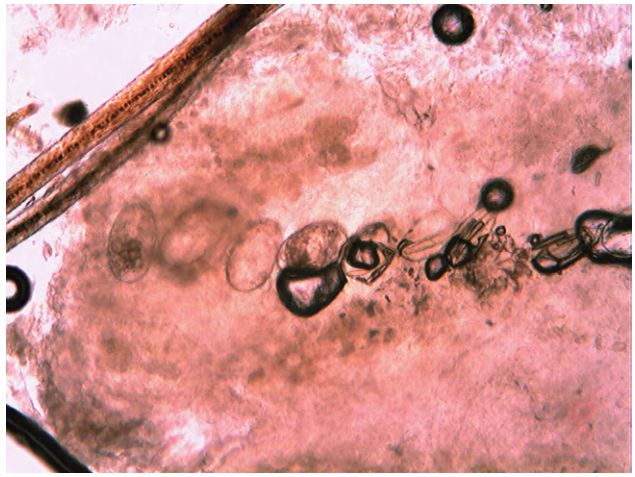


Figure 2.14 Sarcoptes eggs from skin scraping.

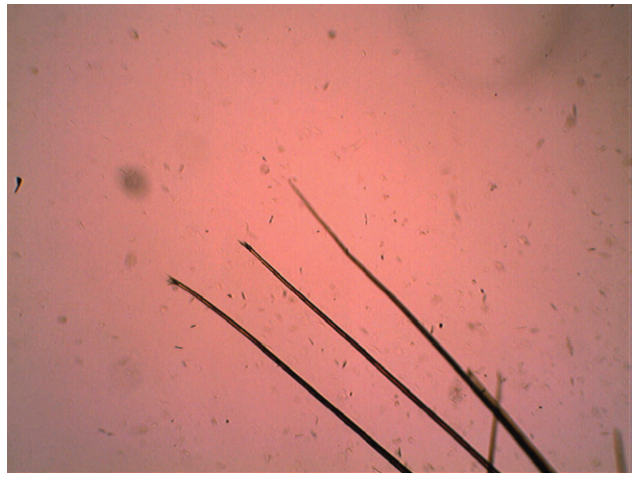


Figure 2.16 Damaged hair tips suggestive of self-inflicted trauma.

and sharply pulled out (Fig. 2.15). A small pair of artery forceps may be used to grasp the hairs but can cause crush artefact on the shaft of the hair. Hair can then be mounted in liquid paraffin or 10% potassium hydroxide.

Hair Tip

Examination of the tip is most useful where traumatic hair loss is suspected. Tip damage suggests self-inflicted trauma (Fig. 2.16). In hair loss through endocrine disease the tip remains finely tapered (Fig. 2.17).

Hair Shaft

Dermatophyte ectothrix spores can be identified on the shafts of the hairs. Spores appear as small spherical structures in rows along the shaft. Pigment changes such as



Figure 2.17 Normal tapered hair tips.

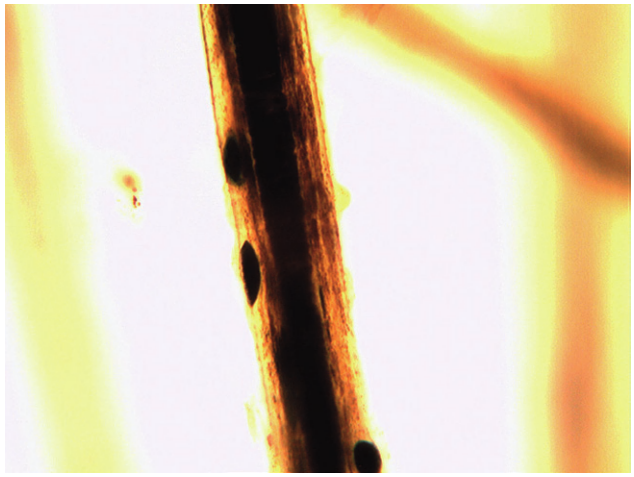


Figure 2.18 Pigment clumping on hair shaft in colour mutant alopecia.

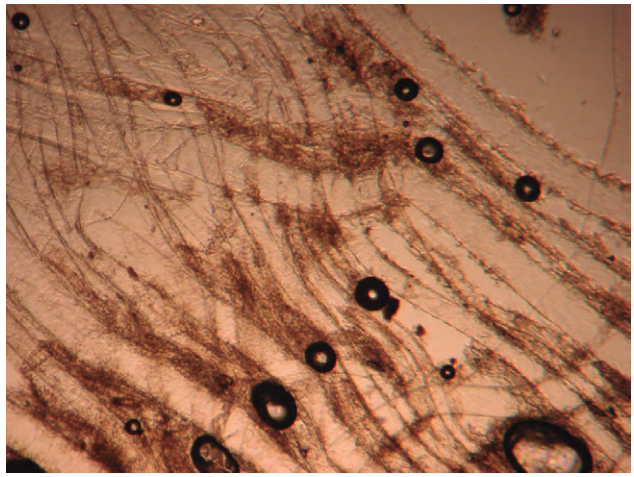


Figure 2.20 *Demodex canis* mites lined up along the hair shafts.

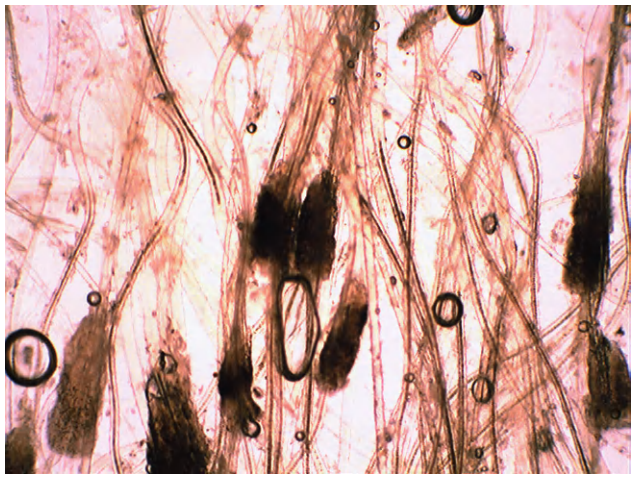


Figure 2.19 Follicular casts on hairs in demodicosis.

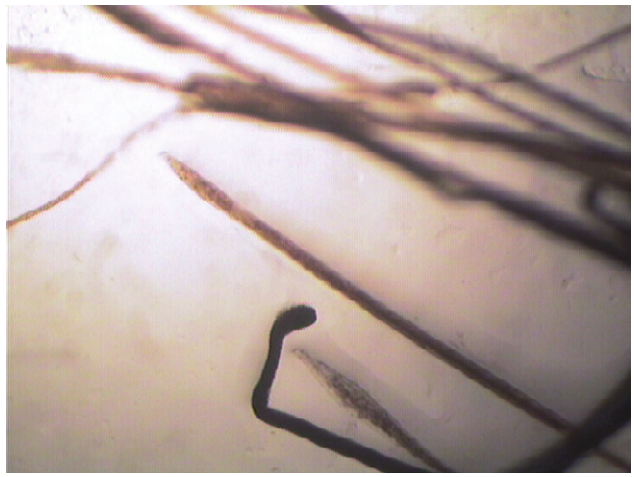


Figure 2.21 Bulbs of hairs showing telogen and anagen hairs.

clumping of pigment along the shaft are typical of colour mutant alopecia follicular dysplasia (Fig. 2.18). Follicular casts can be seen in sebaceous adenitis, follicular dystrophy, demodicosis (Fig. 2.19), and hyperadrenocorticism. Louse eggs can be seen attached to hair shafts, and follicular demodex can be identified parallel to deep hair shaft (Fig. 2.20).

Hair Bulbs

Examination of the bulbs allows assessment of the stage of the growth cycle of each hair (Fig. 2.21).

- *Telogen hairs* have elongated unpigmented spear-shaped roots. These are hairs in the resting phase and are most commonly seen in endocrine disease. Most hairs in normal dogs will be in this stage.
- *Anagen hairs* have bent, club-shaped roots that are heavily pigmented. These are growing hairs and should be identified in samples from normal dogs. In dogs with prolonged growth phase, e.g. poodles, most hairs will be in this stage.

Cutaneous Cytology

Cytological examination is a useful technique that allows the clinician to look for the presence of infection (bacteria, yeast, fungi) and also assess infiltrating cell types (neoplastic, inflammatory cells, acanthocytes).

Three techniques may be employed: direct impression smears, examination of pustule contents, or fine needle aspirates.

Direct Impression Smears

This techniques is suitable for any exudative lesion, e.g. erosions, ulcers, papules, and furuncules, or from the undersides of crusts.

- Non-exudative lesions can be picked with a sterile 25 gauge needle to express fluid (Fig. 2.22).
- The microscope slide is gently pressed on to the lesion to collect exudates (Fig. 2.23).
- The sample may be air-dried or carefully warmed to dry (the author uses a hand dryer).



Figure 2.22 Non-exudative lesions may be picked to express fluid.

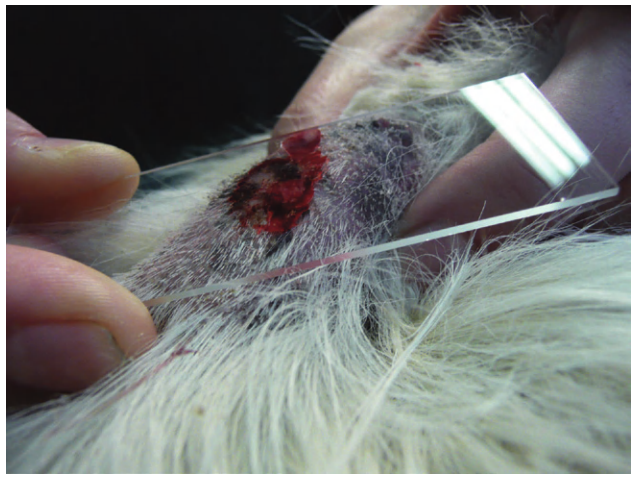


Figure 2.23 Slide is placed on to lesion to collect exudate.

- The slide is stained using a modified Wright's stain (Diff-Quik) and gently rinsed across the reverse of the slide and dried again.
- The sample should always be examined on low power (10×objective) initially to select areas for close inspection subsequently on high power (40×) or under oil immersion (100×).

Examination of Pustule Contents

This method is useful for pustular lesions to identify if they have an infectious or sterile aetiology. The pustule should be picked with a sterile 25 gauge needle and the contents gently pressed on to the slide. It should be stained as above. Findings include:

- bacteria plus degenerate neutrophils, identified in bacterial infection (Fig. 2.24)
- acanthocytes (rounded nucleated keratinocytes) and non-degenerate white blood cells (usually neutrophils and eosinophils), seen in immunological diseases such as pemphigus (Fig. 2.25)
- demodex mites, bacteria, and degenerate neutrophils – can be seen in cases of follicular demodicosis (Fig. 2.26).

Fine Needle Aspirates

Aspirates are most suitable for nodular lesions, especially neoplastic and pyogranulomatous disease.

- A 21–23 gauge needle attached to a 2 mL or 5 mL syringe should be used.
- The lesion should be sterilised with spirit and immobilized between finger and thumb.
- The needle is inserted into the lesion while pressure is applied to the plunger to create negative pressure. The

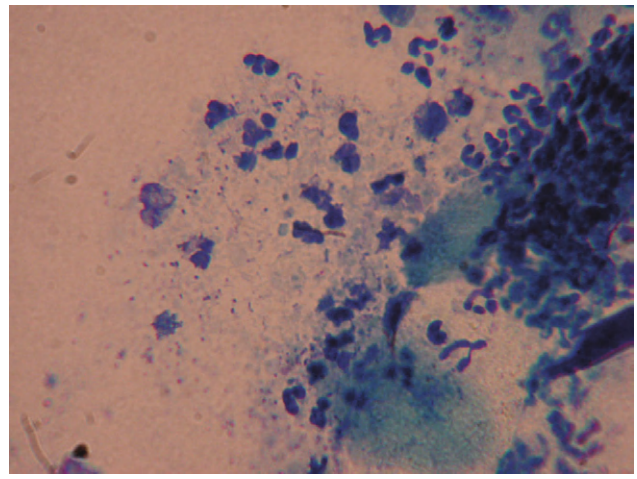


Figure 2.24 Bacteria and neutrophils from an area of infection.

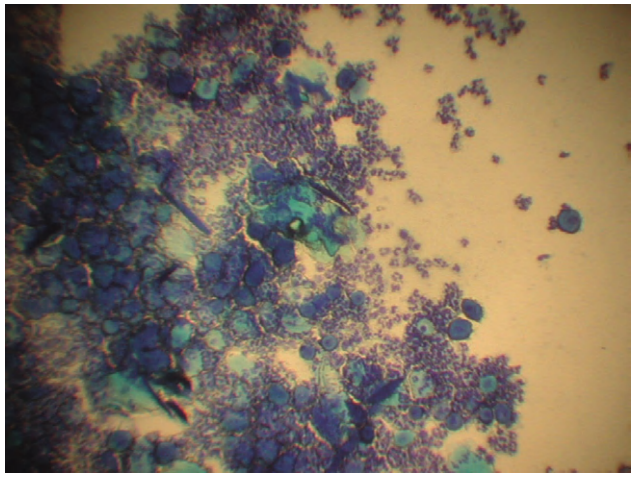


Figure 2.25 Non-degenerative neutrophils and acanthocytes from a case of pemphigus foliaceus.



Figure 2.26 Neutrophils and demodex from a pustule.



Figure 2.27 A 23 gauge needle is inserted into the lesion.

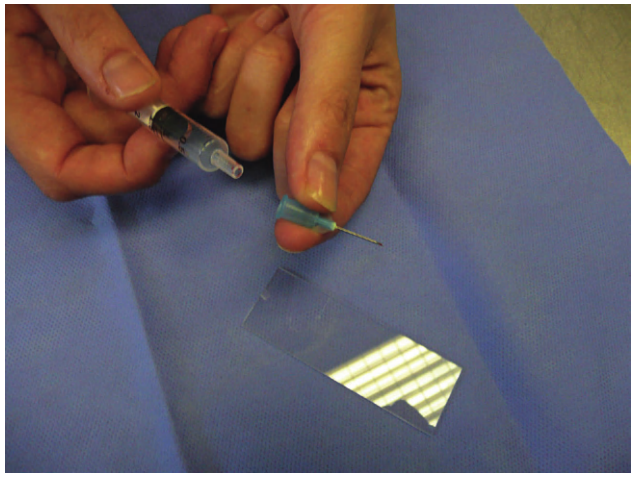


Figure 2.28 Needle is removed and plunger withdrawn.

needle can be constantly repositioned within the lesion without removing it, to draw up as much material as possible (Fig. 2.27).

- The pressure on the needle should be released before the needle is withdrawn. The sampling should be stopped if there is evidence of blood in the hub of the needle.
- The needle is then removed and the plunger withdrawn to fill the syringe with air (Fig. 2.28).
- The needle is then replaced and the contents of the needle expressed on to a clean microscope slide (Fig. 2.29).
- The material may be gently smeared if necessary and stained as above. The slide should be scanned initially on a low power (4× or 10×) to identify areas of interest before using high power (40×) or oil immersion (100×).

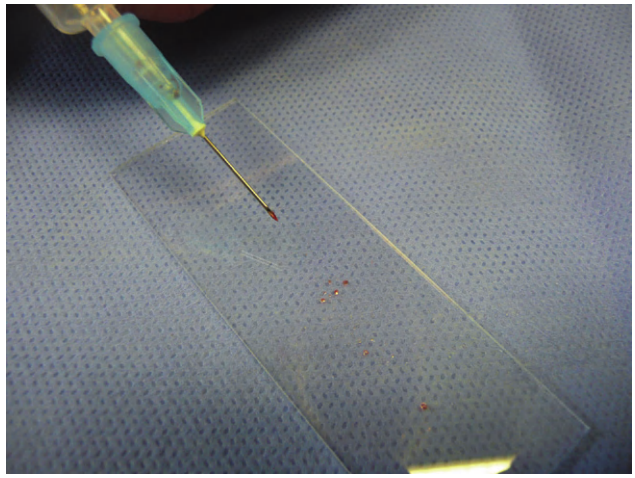


Figure 2.29 Contents of syringe are expelled on to a slide.

2.1.2 Biopsy

Biopsy of the ear pinna is rarely indicated. Where possible biopsy samples should be taken from early primary lesions. Punch biopsies are easy to take from the ear flap, but difficult to suture without causing deformity of the ear pinna so are probably best avoided. Elliptical biopsies are more successfully sutured and where papules or nodules are to be sampled excisional biopsies are best. The major indications for biopsy include

- suspected neoplastic lesions
- ulcerative or vesicular lesions
- skin disease unresponsive to rational therapy
- to make a diagnosis where expensive or potentially dangerous drugs are to be used.

Technique for Excisional Biopsy From the Ear Pinna

1. Select an appropriate area to biopsy. Where possible choose a primary lesion (e.g. pustule, papule, nodule) and remove the whole lesion. Where ulcers or erosions are present an ellipse should be taken that extends from the lesion across its edge on to normal skin. Biopsy of secondary lesions (e.g. lichenification, hyperpigmentation) is far less useful. Unlike biopsies from other sites it is difficult to take multiple samples.
2. Clip the area gently but do not prep the skin as this can distort findings due to removal of superficial pathology.
3. Inject a small bleb of local anaesthetic (e.g. lignocaine [lidocaine], novocaine, articaine) into the subcutaneous skin.
4. Make an elliptical excision around the lesion with a scalpel blade (Fig. 2.30). Fine forceps should be used to grab the very end of the sample and fine scissors may be used to dissect the sample away from the underlying tissue (Fig. 2.31). The sample should be full skin thickness but should not include the cartilage (Fig. 2.32).
5. Mount the sample on a piece of card and place in 10% formalin solution.
6. Where it is impossible to suture the biopsy it can be lasered to control haemorrhage (the laser should not where possible be used to take the biopsy) and left to heal by secondary intension (Figs 2.33, 2.34).

2.1.3 Fungal Culture

Dermatophyte culture should be considered in any feline pinnal skin disease. A sterile toothbrush, hairbrush, or carpet square coat brushing can be used.

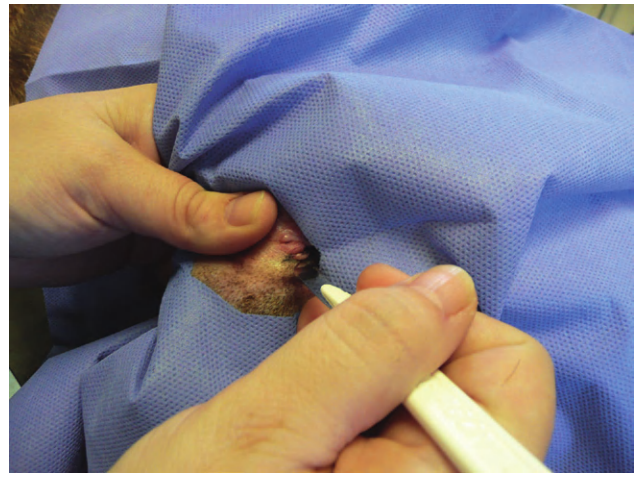


Figure 2.30 An elliptical incision is made around the lesion.



Figure 2.31 The sample is dissected away from underlying tissue.



Figure 2.32 The biopsy should be full thickness of the skin but should not normally include cartilage.



Figure 2.33 Lesions may be lasered if suturing is not possible.



Figure 2.35 Dermatophyte culture using a sterile toothbrush.



Figure 2.34 Lasered lesion after biopsy collection.



Figure 2.36 A guarded sample may be taken from the horizontal ear canal.

Sampling Lesions

Samples of hair and scale can be collected by brushing the lesion and an area of at least 3 cm around its periphery (Fig. 2.35). Alternatively, sterile forceps can be used to pick off scale, crust, and hair.

Wood's lamp examination of hairs may help select suitable hairs for sampling.

2.2 EXTERNAL EAR CANAL

2.2.1 Examination of Ear Wax/Discharge From External Ear Canal

Both plain unstained samples and stained samples from the external ear canal should be examined. This procedure can be used to make initial decisions for treatment possibly pending cultures and can also be used to monitor the progress of the disease.

Samples should where possible be taken from the horizontal canal. Often this is difficult to achieve without collecting commensals from the vertical ear canal. A clean otoscope cone can be employed to collect a guarded sample (Fig. 2.36).

Initial Sampling

Plain sample is prepared by rolling wax collected on a cotton wool swab from the external ear canal on to a microscope slide (Fig. 2.37). The sample should then be examined unstained. Initially this should be done on low power

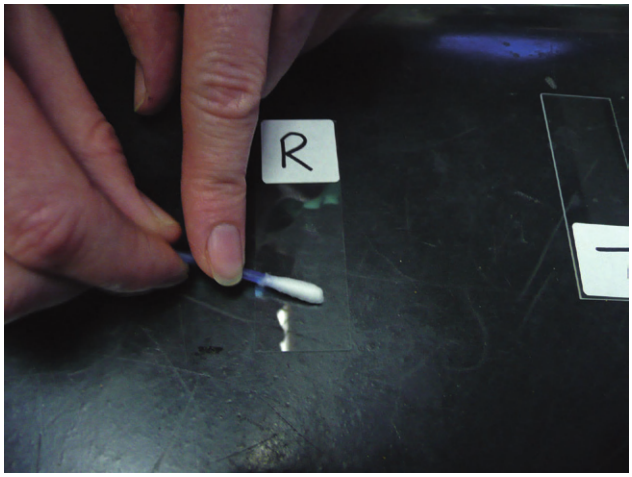


Figure 2.37 Swab is rolled on to slide.

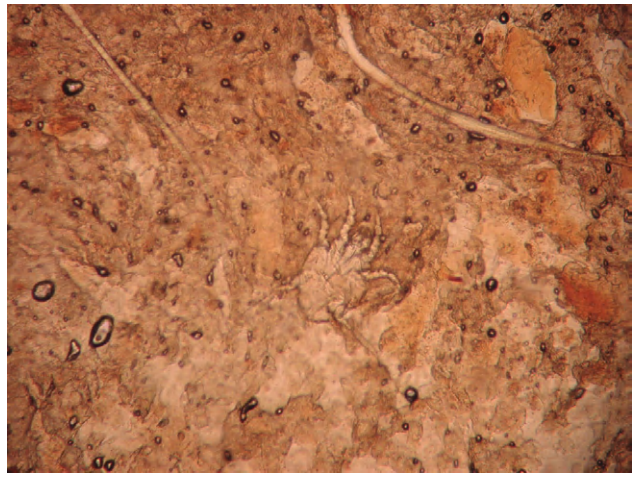


Figure 2.39 *Otodectes cynotis* in ear wax.

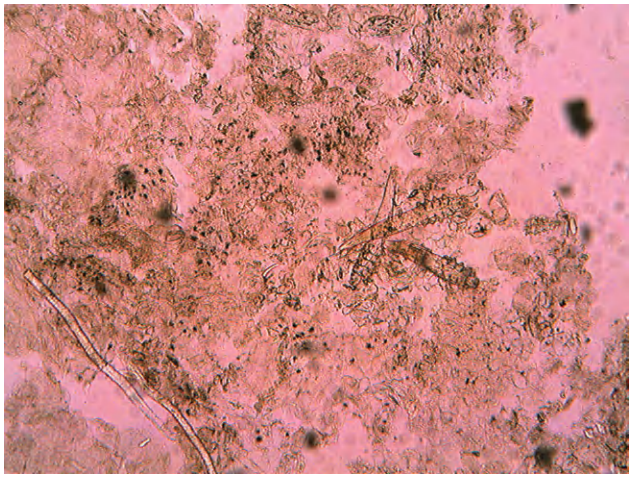


Figure 2.38 *Demodex canis* in ear wax.

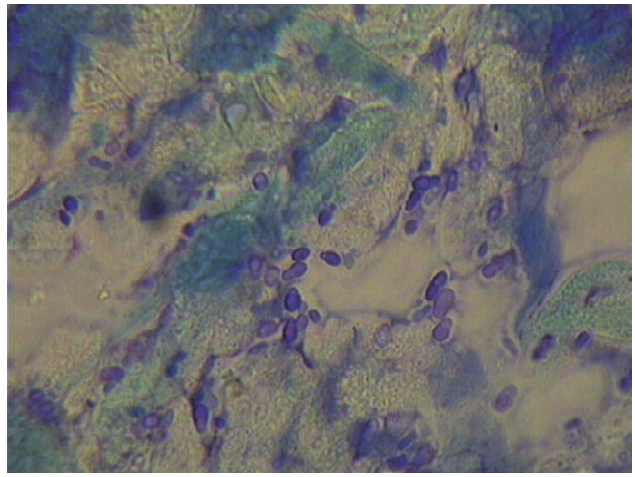


Figure 2.40 *Malassezia pachydermatis* in stained ear wax.

(4×, 10×); rarely is a higher power needed. Demodex (Fig. 2.38) and *Otodectes cynotis* (Fig. 2.39) can be identified by this technique.

Wax may also be collected and prepared in the same way but air-dried or gently warmed and then stained with Diff-Quik. Examination should be performed with both low- and high-power objectives. This technique will identify different cell types and microorganisms such as bacteria (rods and cocci) or yeast. Some bacteria such as pseudomonas produce slime which is also visible on samples. Where there is inflammation within the middle ear samples may also contain mucus.

Cytological Findings

Microorganisms stained with Diff-Quik stain will all appear blue in colour.

Yeast

- *Candida* appears as a bipolar 'cottage loaf'-shaped structure with a narrow waist
- *Malassezia* appears as a bipolar 'peanut'-shaped organism with a wide waist (Fig. 2.40).

Gram-Positive Bacteria

- *Staphylococcus* is a Gram-positive coccus. Cellular division usually takes place at right angles so that the bacteria appear grouped in 'grape like' clusters (Fig. 2.41).
- *Streptococcus* is a Gram-positive coccus. Cellular division takes place along a single axis so the bacteria appear to be grouped in pairs or chains (Fig. 2.42).
- *Enterococcus faecalis* (previously classified as a group D streptococcus) is a Gram-positive coccobacillus. It is

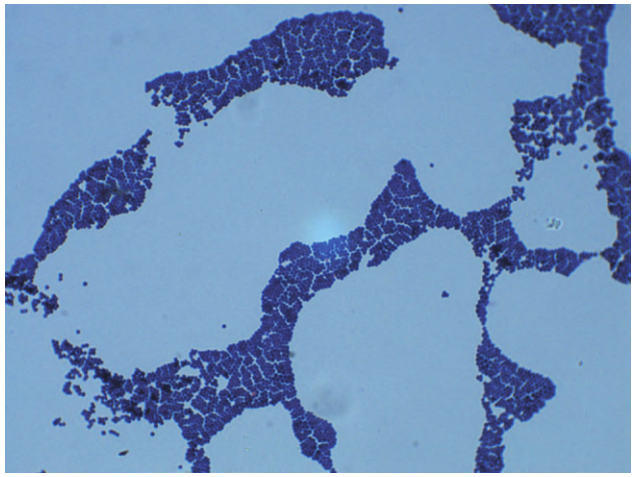


Figure 2.41 Staphylococcus (Gram stain). Courtesy of Stephen Steen.

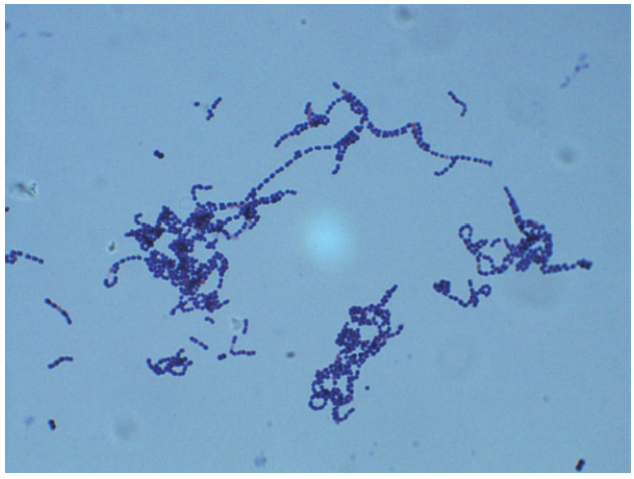


Figure 2.42 Streptococcus (Gram stain). Courtesy of Stephen Steen.

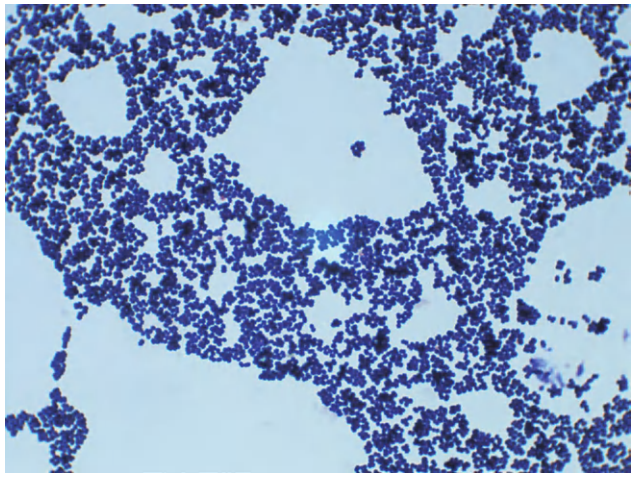


Figure 2.43 Enterococcus (Gram stain). Courtesy of Stephen Steen.

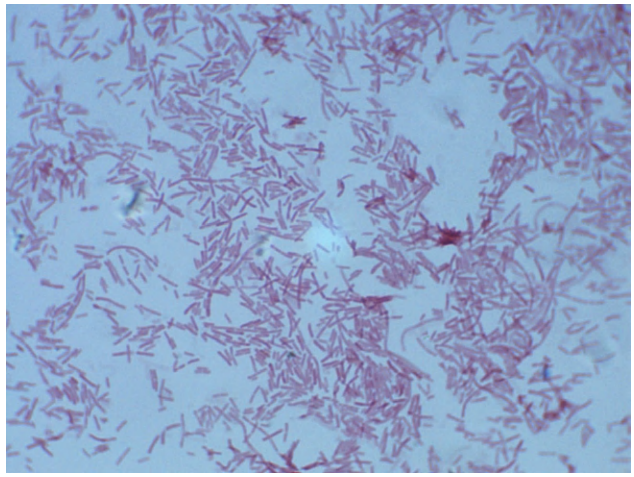


Figure 2.44 Pseudomonas (Gram stain). Courtesy of Stephen Steen.

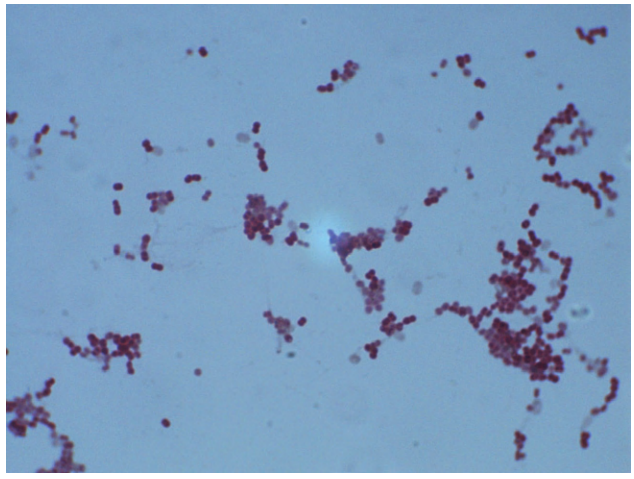


Figure 2.45 Pasteurella (Gram stain). Courtesy of Stephen Steen.

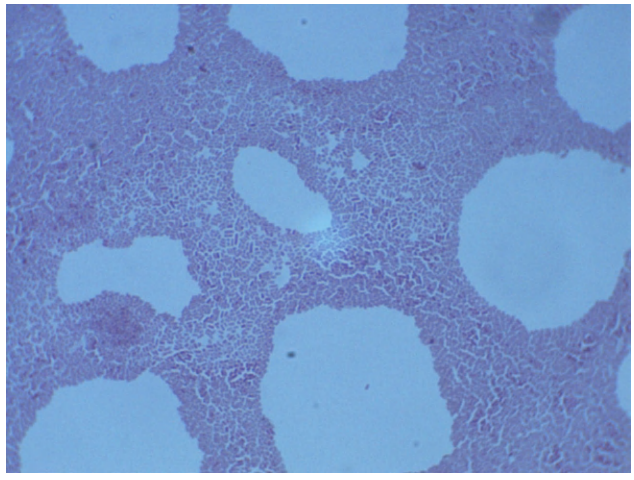


Figure 2.46 Proteus (Gram stain). Courtesy of Stephen Steen.

similar in arrangement to streptococcus, in short chains (Fig. 2.43).

Gram-Negative Bacteria

- *Pseudomonas* is a very long Gram-negative rod compared to other aural pathogens such as pasteurella and proteus (Fig. 2.44).
- *Pasteurella* is a Gram-negative coccobacillus with a clear bipolar staining (Fig. 2.45).
- *Proteus* is a relatively short Gram-negative rod (Fig. 2.46).

2.2.2 Culture

Culture is not indicated in all cases of otitis. Where malassezia is identified on cytology then treatment can be prescribed empirically. Where cocci are identified, treatment can usually be selected to use drugs with a recognized sensitivity to staphylococcus and streptococcus (the exceptions are listed in Table 2.3). Where rods are identified on cytology then culture should be routinely undertaken to include both aerobic and anaerobic culture. Where possible a culture should be obtained from deep within the vertical canal/horizontal canal to try to avoid isolation of any commensals. This is best achieved by using a guarded swab technique. A fine bacteriology swab is inserted down a sterile otoscope cone or down the working channel of a video otoscope to take a sample from the end of the device. This allows the swab tip to be protected from the discharge within the higher parts of the canal.

Table 2.3 Indications for culture from the external ear canal

Indications	Comments
Where cocci are present on cytology and have not responded to appropriate topical therapy	Infection may be due to methicillin-resistant bacteria which have an inherent resistance to aminoglycosides, fluoroquinolones, fusidic acid
Presence of unusual Gram-positive cocci	Infection may be due to *Enterococcus faecalis* which is a multiply resistant organism and can be associated with long-term antibiotic usage
Where mixed infection is present with both rods and cocci	Culture can help select the most suitable topical product especially where an ear drop contains two different antibiotics
Where rods are present on cytology	Sensitivity patterns are less predictable with Gram-negative organisms
Where there is a history of chronic otitis externa and multiple topical therapies	Chronic antibiotic therapy can predispose to anaerobic infection especially where aminoglycosides and fluoroquinolones have been used topically

2.2.3 Fine Needle Aspirates

The indications for fine needle aspirate are the same as those for lesions of the ear pinna. Needles inserted down the working channel of an otoscope are usually of not a wide enough bore to take an adequate sample. Better samples can be achieved by use of a spinal needle which can be inserted along the side of the otoscope cone and into the mass to be sampled (Fig. 2.47). The technique is the same as that outlined above for sampling the ear pinna.

2.2.4 Biopsy

Biopsy of the ear canal is most useful for masses within the wall of the canal. These can be taken as pinch biopsies by use of grabbing forceps (Fig. 2.48) or by using a sharp curette. Haemorrhage is not usually a major problem if the samples are taken by tearing the skin. Material should be submitted for analysis in 10% formalin.

2.3 MIDDLE EAR

2.3.1 Examination of Discharge From the Middle Ear

When the eardrum is ruptured a sample can be collected from the middle ear using a guarded swab technique as

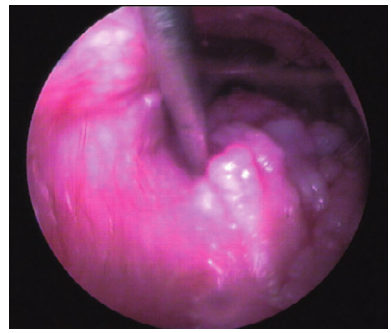


Figure 2.47 Fine needle aspirate of mass in ear canal.

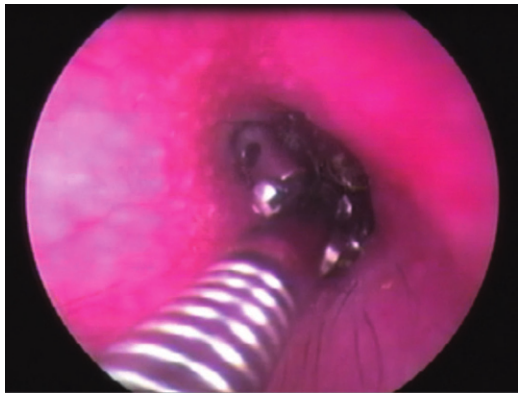


Figure 2.48 Pinch biopsy of mass in dogs ear.

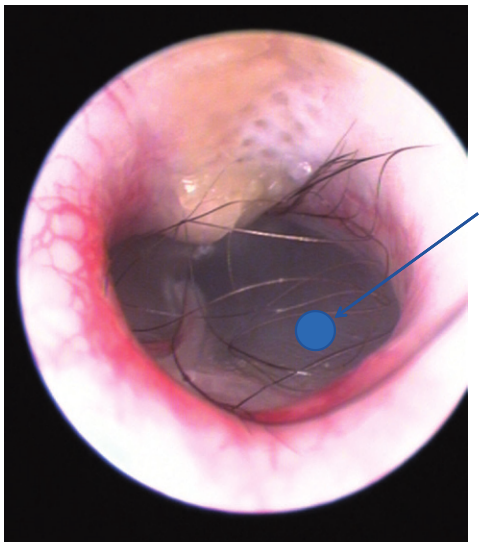


Figure 2.49 Normal tympanic membrane showing position of myringotomy incision.

described under the culture technique for the external ear canal. This can be examined cytologically and also submitted for culture.

When the eardrum is intact but abnormal and/or otitis media is suspected a sample may be collected from the middle ear by myringotomy. This is best achieved using a video otoscope.

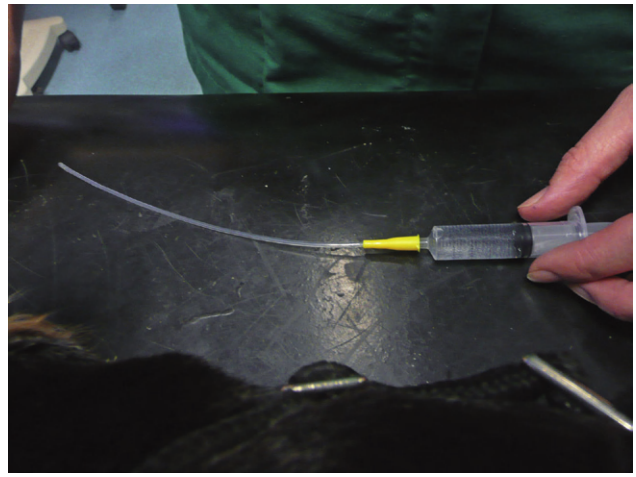


Figure 2.50 Urinary catheter with end cut off which can be used for myringotomy.

A hole can be made in the pars tensa (Fig. 2.49) to access the tympanic bulla using a sharpened 5–6 French gauge urinary catheter (this can be created by cutting the catheter off above the level of the terminal hole at an angle of about 45°; see Fig. 2.50) or CO_2 laser inserted down the working channel of the video otoscope. Care should be taken to avoid the vascular pars flaccida and the malleus. Once the catheter has been inserted a sterile 2–5 mL syringe can be attached. The plunger can then be gently withdrawn. If fluid is obtained by suction it can be submitted for cytology as well as culture and sensitivity. If no fluid is obtained 1.0 mL of sterile water can be instilled into the bulla through the catheter and then collected. Samples can then be examined by cytology and submitted for culture. Where otitis media is present an inflammatory exudate is present on stained cytological samples, often with the presence of microorganisms. However, culture is generally considered a more sensitive way to identify infection than cytology alone.

VetBooks.ir

CHAPTER 3
ADVANCED DIAGNOSTIC TECHNIQUES

Sue Paterson

3.1	Video Otoscopy	37	3.4	MRI	44
3.2	Radiography	38	3.5	Brainstem Auditory Evoked Response	46
3.3	CT	43	3.6	Auditory Tympanometry	49

Advanced diagnostic techniques are not indicated in all cases of otitis externa. Where otitis media is suspected then further tests become more important. Video otoscopy, which has become widely available to primary care veterinary surgeons as well as within specialist institutions, is useful to visualize the canal and also the tympanic membrane. Other diagnostic modalities that may be used include radiography, CT and MRI. Brainstem auditory evoked responses (BAER) are useful to assess an animal's hearing; auditory tympanometry can help assess the tympanic membrane and the middle ear.

3.1 VIDEO OTOSCOPY

Video otoscopy is now widely available in primary care veterinary practice. The video otoscope has several advantages over the hand-held otoscope. A conventional hand-held otoscope produces a bright light that is directed down a conical tube; light from the highlighted object is passed back to the user and viewed through a magnifying lens at the eyepiece at the end of the otoscope cone. The two major disadvantages of this type of system are that (1) much of the light is absorbed by the otoscope cone, meaning illumination of the object is often poor, and (2) insertion of instruments down the cone in the form of forceps or flushing tubes obscures the view for the observer.

Video otoscopes (Fig. 3.1) overcome the shortcomings of the hand-held instruments by providing excellent illumination and high levels of magnification that facilitate examination, cleaning and drying of the ear as well as performing minor surgical procedures (Fig. 3.2). All modern video otoscopes have working channels which accept a range of different instruments including flushing catheters, grabbing forceps, fine-bore needles and laser tips (Fig. 3.3). Insertion of a feeding tube or urinary catheter down the working channel allows efficient irrigation and suction of the canal and the middle ear. Similarly, such tubes can be used to undertake myringotomy as part of the investigation of otitis media. Grabbing forceps can be used to remove foreign material including ceruminoliths and take pinch biopsies of lesions in the canal. They can also be used to grasp and remove nasopharyngeal polyps in cats. Fine-bore needles can be used to inject pharmaceuticals such as



Figure 3.1 Modern video otoscope.

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.



Figure 3.2 Video otoscopy provides a high-quality, high-magnification images.

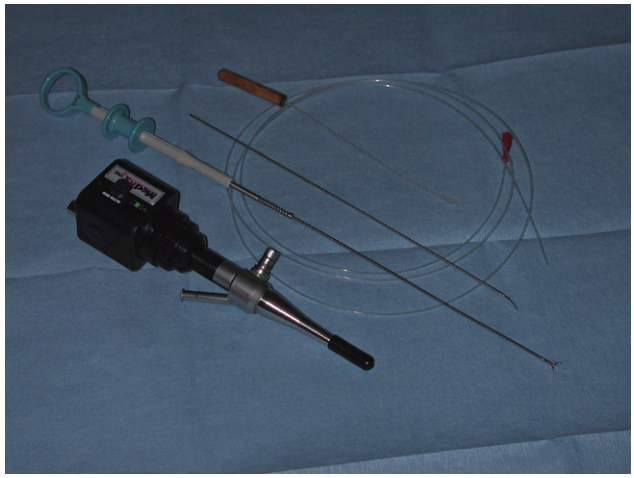


Figure 3.3 Flushing catheter, grasping forceps, and fine-bore needles for use down the working channels of a video otoscope.

glucocorticoids into discrete lesions and the wall of the canal to treat hyperplastic change. Laser tips inserted down the canal can be used to remove lesions from the canal or destroy overactive glandular tissue and also to perform myringotomy. Video otoscopes also have the ability to store information. Images from the ear canal can be captured, stored and then printed out (Fig. 3.4). This has the advantage that owners and referring veterinarians can be shown the condition of the ear canal before therapy and the changes in the ear as therapy progresses. Most instruments

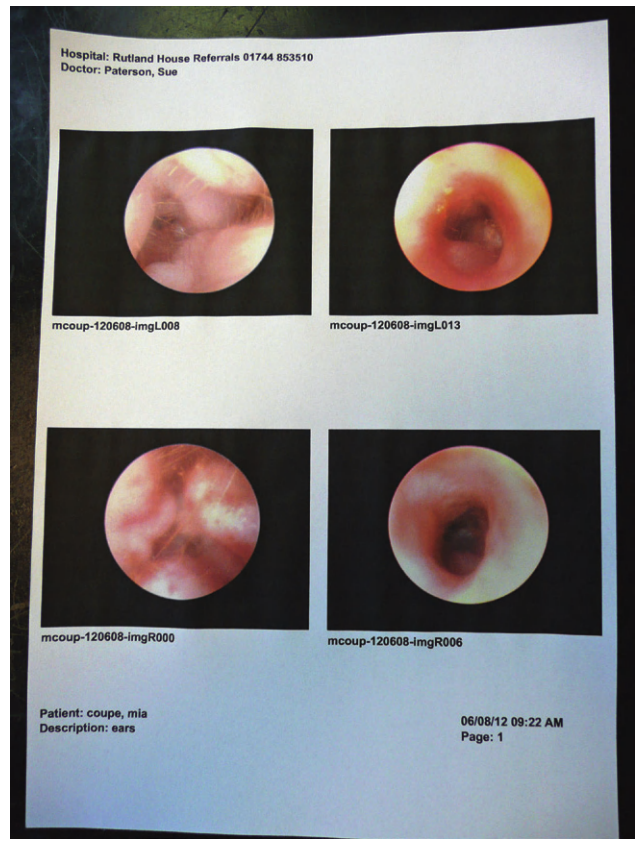


Figure 3.4 Typical printout from a video otoscope.

also have the capability to record video footage which again can be useful to show to clients and also may be used for instruction as a teaching aid.

3.2 RADIOGRAPHY

Radiography is a useful tool in the investigation of ear disease in the dog and cat, but it is not as sensitive a diagnostic tool as CT and MRI. To use it to its full potential accurate positioning of the patient is essential and a comparison of the two sides helps in assessing for abnormalities (Table 3.1). Radiography can be used to assess both the ear canal and the tympanic bulla. The most useful views are the dorsoventral, rostrocaudal (open mouth) and lateral oblique views. Subtle changes in soft tissue density in the middle ear or in the canals can be identified by comparing left and right sides. Bony change in the wall of the bulla such as sclerosis and thickening or lysis, or calcification of the canal, can be identified often without the need for comparison. Although the dorsoventral view can be taken in a sedated animal all of the views are better if the animal is anaesthetized.

The dorsoventral (DV) view is taken with the animal in ventral recumbency (Figs 3.5–3.6). The animal should lie

VetBooks.ir

Table 3.1 Advantages and disadvantages of each radiographic position

Position	Advantage	Disadvantage	Comments
DV	Positioning of animal easier than others as mandibles provide stability against rotation. Bullae are close to the plate	Some distortion can occur due superimposition of PT bones	Soft tissue change within the bullae can be identified by comparing the two sides. Air-filled ear canals should be visible at right angles to the sagittal plane
VD	The bulla septum of the cat can often be visualized in this view compared to the DV	Positioning is difficult. Unsuitable for brachiocephalic breeds. Bulla walls can appear artificially thickened due to superimposition of PT bones	Similar to DV
LO	Good visualization of tympanic bulla and PT bones	Only one bulla can be visualized at a time, so comparative studies difficult	Good detail of bulla which appear as smooth, thin-walled structures. Air shadow of ear canal should be visible
RC	Good visualization of tympanic bulla and PT bones. Both sides can be assessed at the same time	Endotracheal tube must be removed after anaesthesia to get good pictures. Positioning to get good radiographs can be difficult	Bullae appear as smooth, thin-walled structures. Soft tissue overlying them may make interpretation of changes within the bullae difficult

DV, dorsoventral; LO, lateral oblique; PT, petrous temporal; RC, rostrocaudal; VD, ventrodorsal.

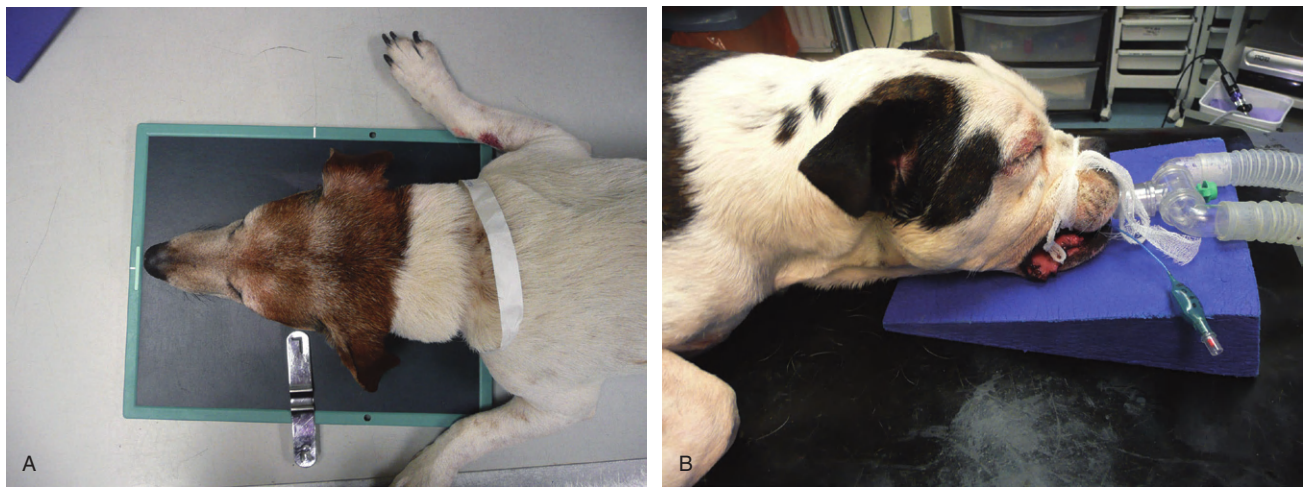


Figure 3.5 Dorsoventral (DV) view is taken with the animal in ventral recumbency. (**A**) View from above; (**B**) View from side.

symmetrically with the interpupillary line parallel to the film. The hard palate should be parallel to the table and the base of the skull should be as close to the plate as possible. The beam should be centred at the intersection of two imaginary lines: the first a sagittal line and the second, which should be at right angles to it, a lateral line drawn through the estimated position of the two tympanic membranes.

The ventrodorsal (VD) view is taken with the animal in dorsal recumbency (Figs 3.7–3.8). As in the previous view the animal should be positioned symmetrically with the hard palate parallel to the plate. Animals in this position are generally more unstable than those in the DV view and usually need tape around their jaw behind the mandible to position the head. The beam should be positioned as the DV view.

The lateral oblique (LO) view is taken with the animal in lateral recumbency with the head positioned parallel to the plate (Figs 3.9–3.13). The jaw should be closed and the bulla to be radiographed should be closest to the film. To avoid superimposition of the two bullae the animal's head is rotated along the long axis until the sagittal plane is 20% to the horizontal. The beam should be centred on the base of the ear.



Figure 3.6 Position of skull in DV view.



Figure 3.8 Position of skull in VD view.

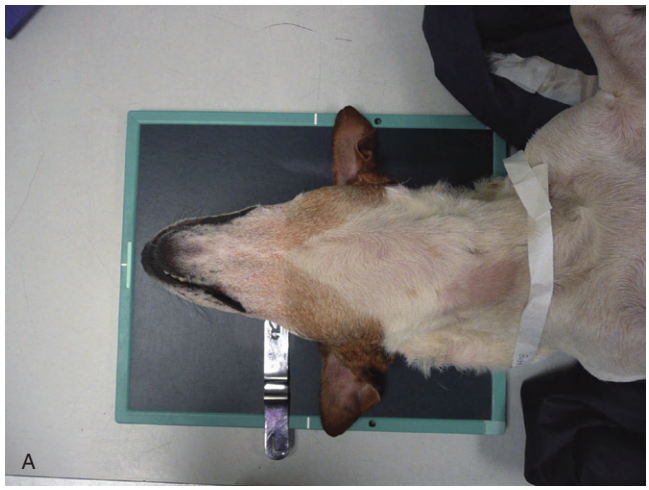




Figure 3.7 Ventrodorsal (VD) view is taken with the animal in dorsal recumbency. (**A**) View from above; (**B**) Side view.

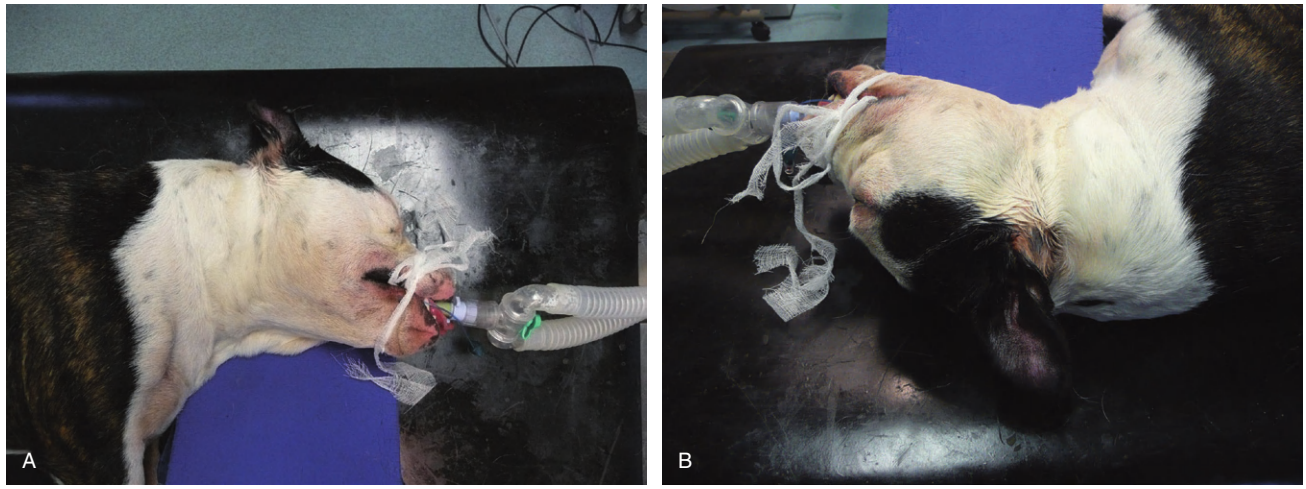


Figure 3.9 Lateral oblique (LO) view is taken with the animal in lateral recumbency with the head parallel to the plate. (**A**) and (**B**) Side views.



Figure 3.10 Position of skull in lateral view showing superimposition of bullae, demonstrating the importance of the oblique view to separate the bullae radiographically.



Figure 3.11 LO view showing bulla outline.

The rostrocaudal (RC) view is taken with the animal in dorsal recumbency (Figs 3.14–3.17). The head is positioned with the sagittal plane and hard palate vertical to the film. The tongue should be brought forward and tie to the mandible with tape. The interpupillary line should be parallel to the film. The mouth should be opened and the beam should be centred at the base of the tongue. In dolicocephalic breeds the beam should be parallel to the hard palate; in other breeds it may need to be angled up to 20° away from the vertical to avoid superimposition of the bullae on the wings of the atlas.

3.2.1 Radiography of the Canal

Plain radiographic views of the ear, especially the DV, VD, and RC, can be taken initially to try to highlight air within the canals (Fig. 3.18). In chronic cases where the canal is calcified the mineralization acts to outline the canal. If the canals are not visible then positive contrast canalography can be performed under general anaesthetic to outline them (Fig. 3.19).

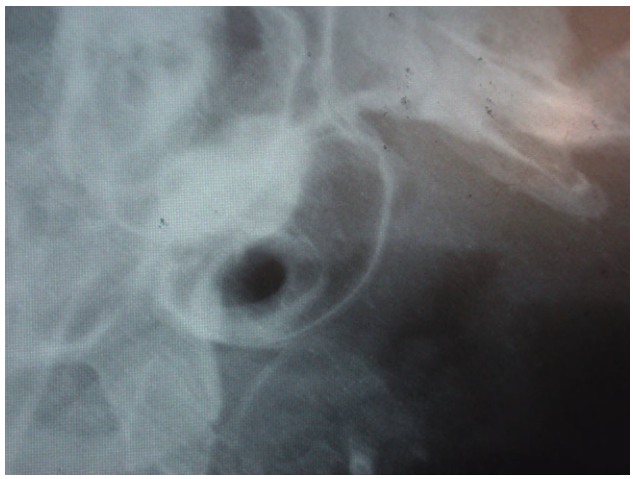


Figure 3.12 Close of bulla in LO view.

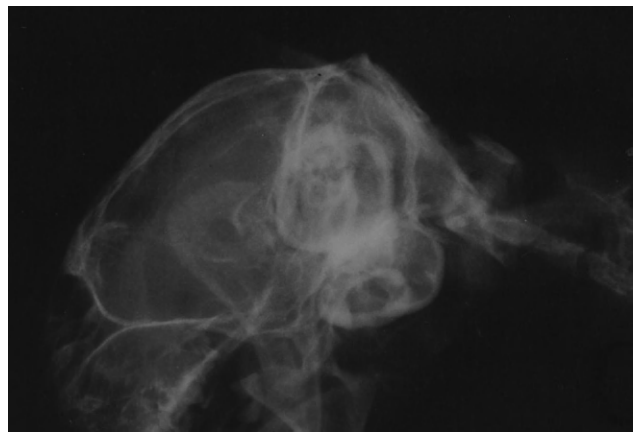


Figure 3.13 LO view showing chronic bulla changes.

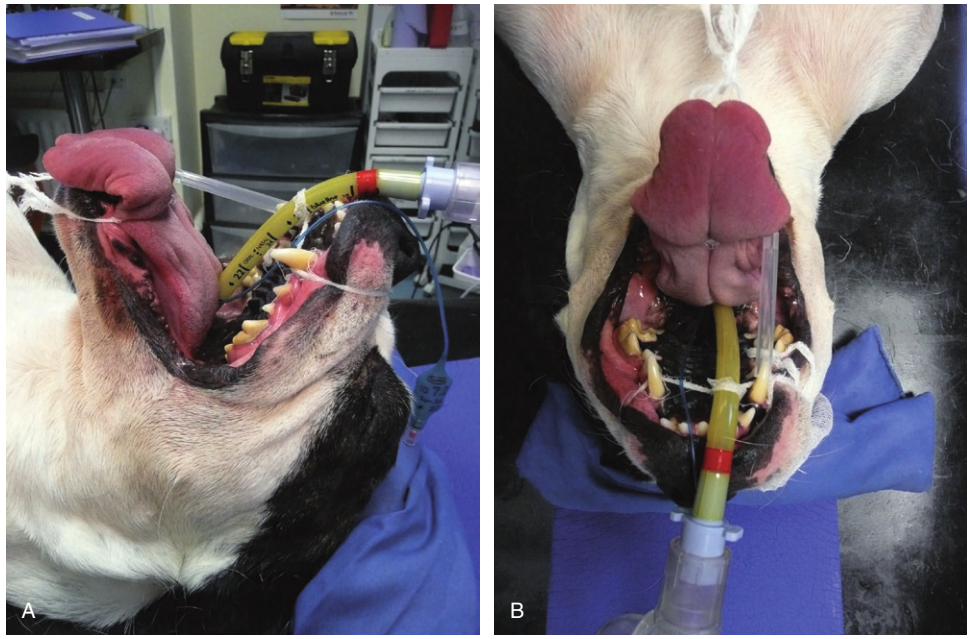


Figure 3.14 Rostrocaudal (RC) view is taken with the animal in dorsal recumbency. A, Side view; B, View from above.

In this procedure 2–6 mL of dilute iodinated contrast agent is instilled into the ear canal (0.3 mL Hypaque 50% or similar in 2.7 mL of saline for a small dog, double this amount for a large dog). If the canal is stenotic the catheter can be used to introduce the medium into the horizontal canal and then 'back fill' the canal. To prevent leakage of the contrast from the ear canal on to the surrounding tissues, a small plug of cotton wool can be placed in the ear after the contrast has been instilled. After the contrast the canal has been delivered into the ear canal it can be gently massaged to distribute the medium along the whole canal. The process may be repeated for other ear before a DV or an open mouth view is taken.

3.2.2 Radiography of the Tympanic Membrane

All four of the views described can be used in an attempt to highlight the tympanic membrane. However, the eardrum can be difficult to visualize with plain radiographs and therefore the same contrast technique as described above for radiography of the canal can be used to visualize



Figure 3.15 RC view showing outline of bullae.

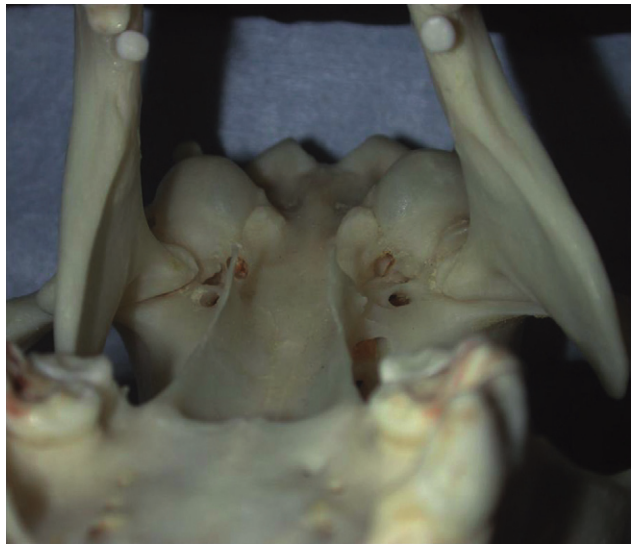


Figure 3.16 Position of skull in RC view.

the tympanic membrane. Contrast material leaking into the middle ear is a good indication that there is damage to the tympanic membrane. This is best identified on an RC view. Unfortunately, failure to detect contrast within the middle ear is not always indicative that the eardrum is intact.

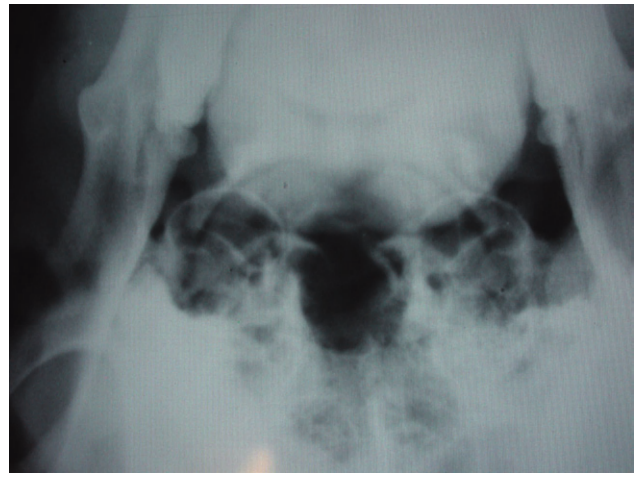


Figure 3.17 Close up of bulla in RC view.



Figure 3.18 DV radiograph showing air in ear canals.

3.3 CT

CT is based on exactly the same principals as radiography and produces a cross-sectional image of the body. The advantage over plain radiography is that the ear can be visualized without the problem of superimposition of other structures. CT images are usually acquired in the transverse plane using thin contiguous slices. Animals need to be heavily sedated or anaesthetized for imaging (Fig.



Figure 3.19 DV radiograph showing contrast medium to outline canals.



Figure 3.20 Dog needs to be sedated or anaesthetized to allow accurate positioning.

3.20) and are generally placed in sternal recumbency with their head supported in a padded trough to prevent rotation of the head and facilitate a symmetrical image (Fig. 3.21). CT differentiates well between air and bone and is an excellent modality for imaging the middle ear. The scan can be enhanced to outline areas of soft tissue inflammation using organic iodide contrast medium.

On a well-positioned CT scan of the ears both bullae should appear symmetrical. The lumina of both tympanic bullae and the external ear canals should be filled with gas (Fig. 3.22). The normal tympanic bulla has a thin, well-defined wall. In otitis media the bulla may be irregular and thickened (Fig. 3.23); occasionally there is lysis of the wall of the bulla. In chronic disease there may be soft tissue densities within the bulla suggestive of fluid or tissue. The external ear canal should be patent and of uniform thickness throughout its length. There should be no evidence of mineralization of the wall of the canal (Fig. 3.24) and no narrowing or obstruction. CT can be used to evaluate the ear canals and tympanic bullae before proceeding to surgery and is especially useful to evaluate the extent and aggressiveness of neoplastic of infectious lesions (Fig. 3.25). CT can be used to evaluate postoperative complications after middle ear surgery, e.g. abscesses, residual necrotic debris.

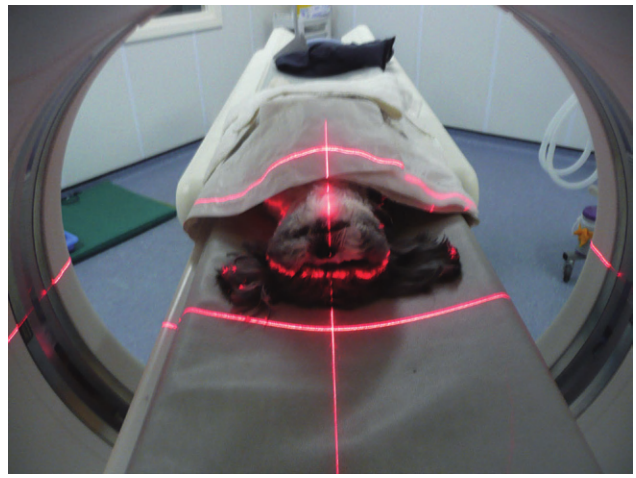


Figure 3.21 Dog positioned in CT scanner.

3.4 MRI

MRI is very different from both CT and radiography. It involves the measurement of radio signals emitted by hydrogen atoms in the body under the influence of a high-powered magnetic field. MRI is a better modality for assessing soft tissue structures that CT. Bone and air contain little hydrogen and therefore appear black on an MRI scan,



Figure 3.22 Normal CT scan of head.

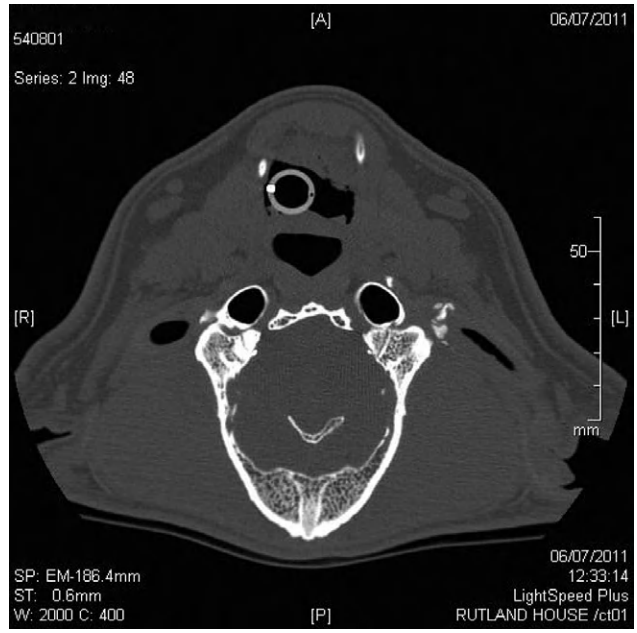


Figure 3.24 CT scan showing calcification of ear canals.

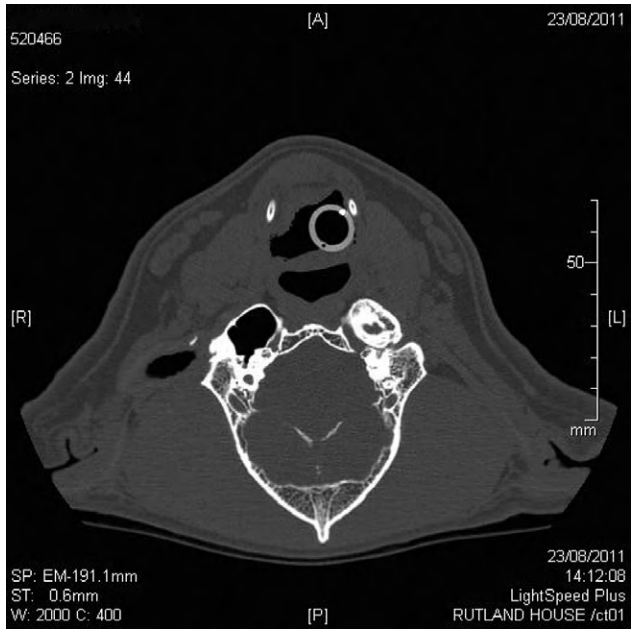


Figure 3.23 CT scan showing severe bony changes in the left tympanic bulla.

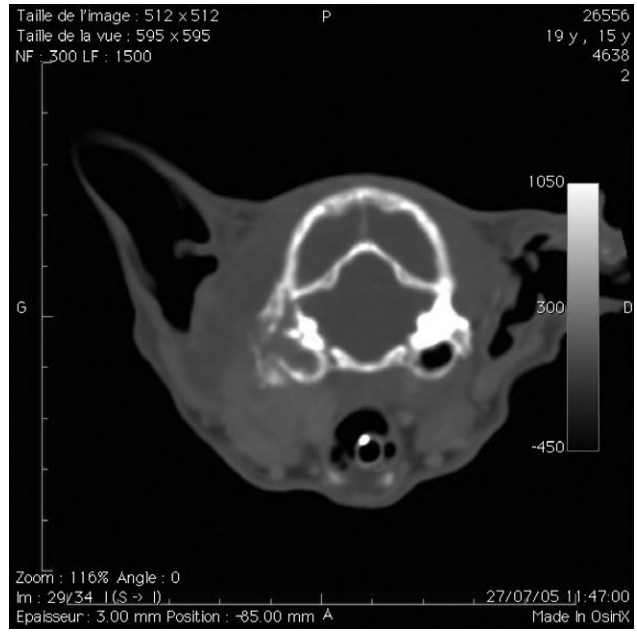


Figure 3.25 CT scan of head of cat showing para-aural abscess.

making differentiation of the two entities difficult and not allowing good visualization of such structures as the tympanic bulla. Animals are usually anaesthetized and placed in dorsal recumbency for imaging. Images are acquired in three planes (dorsal, transverse and sagittal) so that precise positioning is not as critical as it is for CT scanning (Figs 3.26, 3.27). Intravenous injection of the contrast agent gadolinium can be used to improve the sensitivity of this technique to look at areas of inflammation. MRI is not typically used to diagnose otitis externa, but narrowing of

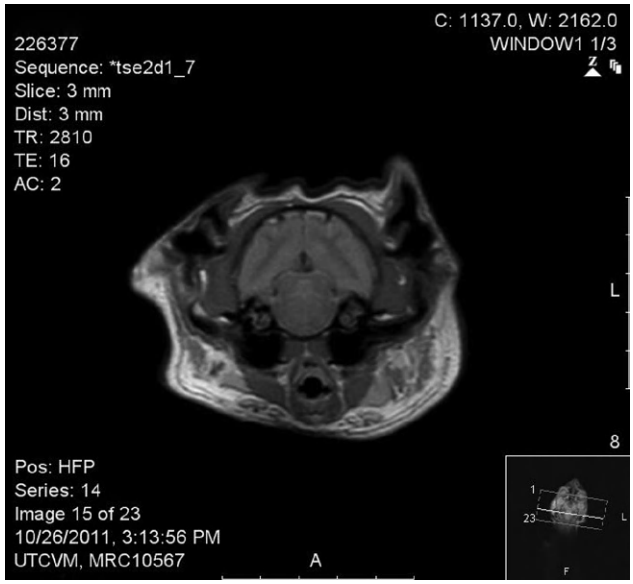


Figure 3.26 Normal MRI scan (cat).

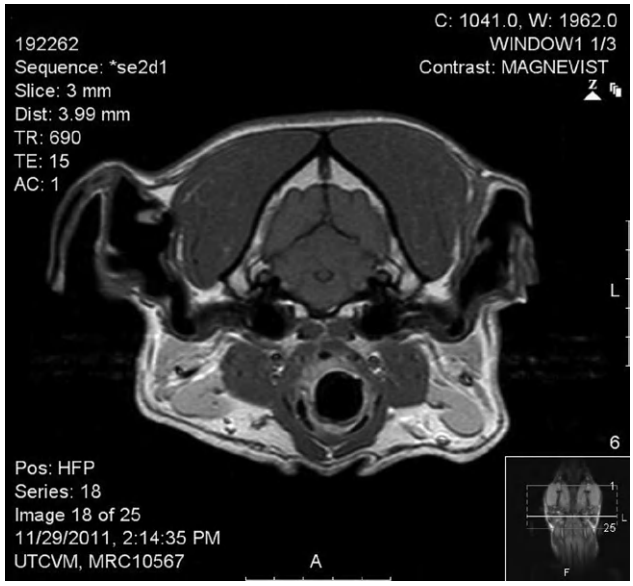


Figure 3.27 Normal MRI scan (dog).

the canal together with fibrous tissue or fluid within the canal can be identified on imaging (Fig. 3.28). One of the disadvantages of MRI, when imaging the ear, is its inability to differentiate between gas and cortical bone: both appear as signal void (black), making it difficult to differentiate the boundaries between the wall of the tympanic bulla and its air-filled lumen within the middle ear and mineralization of the external ear canals unless it is severe. Soft tissue contrast, though, is good so that fibrous tissue within the

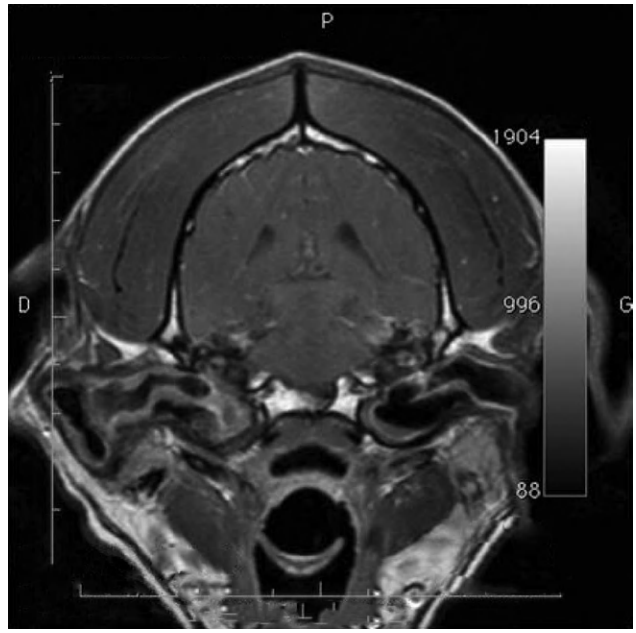


Figure 3.28 MRI scan of dog showing otitis media.

bulla can be identified, as can neoplastic lesions and those within the inner ear.

3.5 BRAINSTEM AUDITORY EVOKED RESPONSE

Electrodiagnostic testing of hearing by use of the BAER is recognized as the most accurate means of assessing hearing in dogs and cats in practice. The equipment is expensive and is therefore often only available in large centres, especially referral institutions (Fig. 3.29). It offers an objective, non-invasive assessment of the presence or absence of auditory function and is the only technique that allows the investigator to make a diagnosis of unilateral deafness. BAER is generally well tolerated by most animals, and sedation or anaesthesia is usually unnecessary. The procedure normally takes about 10–15 minutes. To record the BAER trace, three small needle electrodes are inserted subcutaneously on the animal's head. The first (1) is placed on the top of the head, the second (2) is placed in front of the ear being examined the third (3) is placed either in front of the contralateral ear or subcutaneously over the dorsal spinous process of the third thoracic vertebrae. These correspond to a recording electrode (1), a reference electrode (2), and a ground electrode (3) (Fig. 3.30). Once the needles have been positioned, they can be connected to an amplifying and signal averaging recoding system (Fig. 3.31). Clicks at various rates of delivery (clicks/second), intensity (dB) and frequencies (Hz) are fed to the dog or cat by headphones

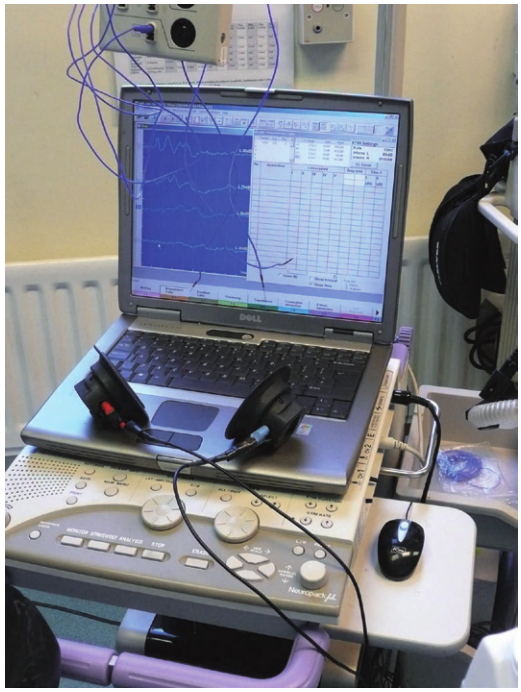


Figure 3.29 Electodiagnostic equipment used for measurement of brainstem auditory evoked response (BAER).

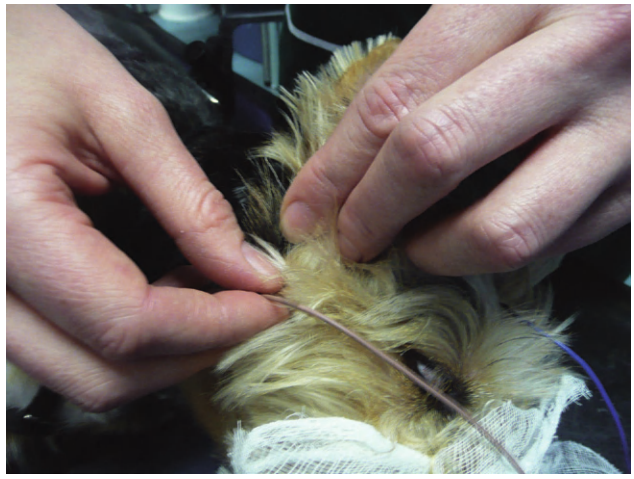


Figure 3.30 Electrodes positioned on the dog's head.

(Fig. 3.32) or ear inserts to generate a BAER trace (Fig. 3.33). The acoustic signal that results in the best response is a click with a broad frequency spectrum in the region of 2–3 kHz. The author calibrates the intensity of the clicks, as most investigators do, in decibels (dB). A typical BAER trace consists of 4–6 waveforms, each of which corresponds to a particular structure or region of the auditory pathway (Fig. 3.34). Where deafness is caused through abnormality



Figure 3.31 Electrodes connected to the amplifying and signal averaging recoding system.



Figure 3.32 Headphones positioned on the dog's head.

in the middle ear or cochlea the BAER trace will be completely flat (Fig. 3.35). Where deafness is caused by a central lesion, the trace is not completely flat: only part of the wave form is lost, depending on the site of the lesion. In such cases the BAER can be used in the neuroanatomic location of the lesion. Where hearing is present but impaired the wave form will become flat at the decibel level not registered by the animal.

Although the BAER can accurately assess the status of a dog or cat's hearing it cannot differentiate between conductive or sensorineural hearing loss. When there is reason to suspect conductive hearing loss either through damage to the ear ossicles or due to material within the middle ear, the BAER test can be repeated using a bone stimulator

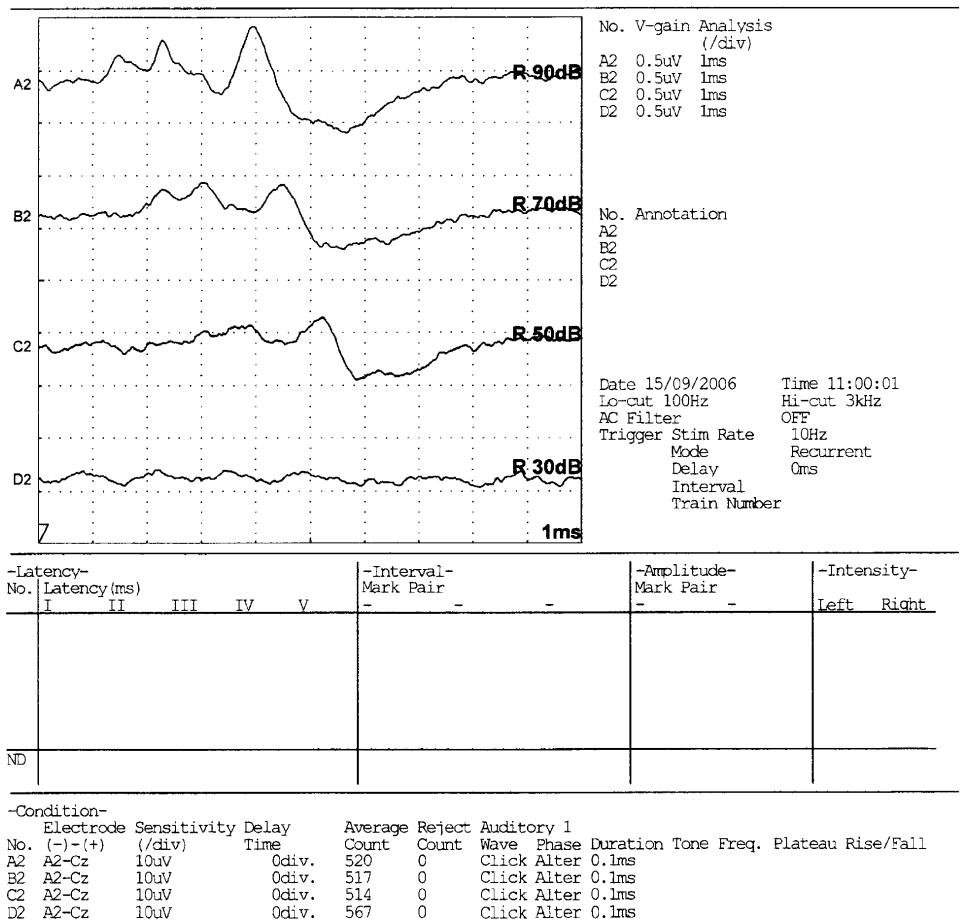


Figure 3.33 Trace generated in response to the dog's perception of the clicks from the headphones.

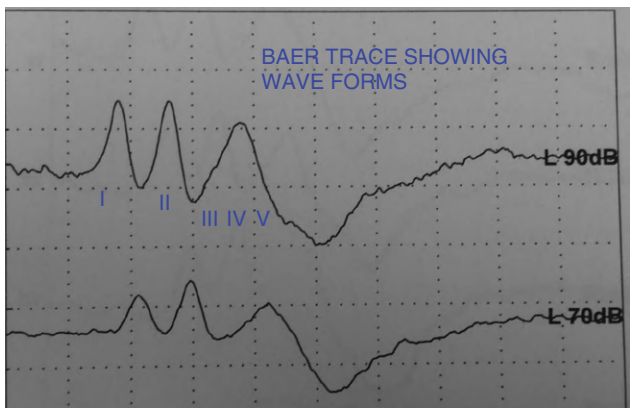


Figure 3.34 Typical BAER trace. Waves I and II are generated by the vestibulocochlear nerve. Wave III is generated by neurons in or near the cochlear nucleus. Wave IV is generated by neurons in the superior olivary complex in the pons and the cochlear nucleus and nuclei of the lateral lemniscus. Wave V is generated by neurons in the contralateral caudal colliculus of the brainstem.

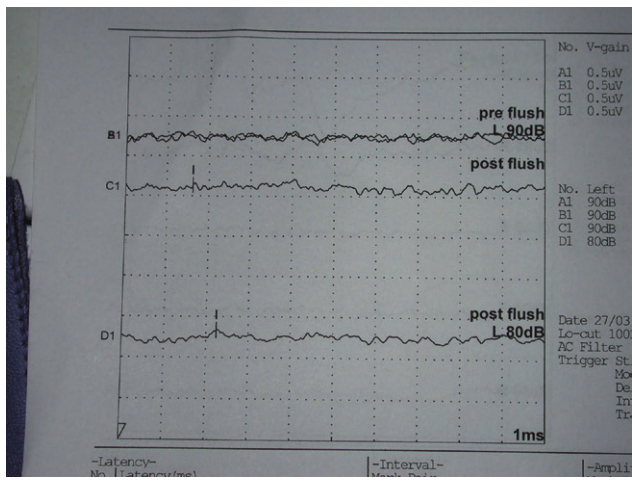


Figure 3.35 BAER trace from a deaf dog.

VetBooks.ir

instead of the earphones. The bone stimulator is held firmly against the mastoid process (although the mandible and zygomatic arch also work). The stimuli are transmitted directly through the bone of the skull to the cochlea, which is embedded in the mastoid bone, thus bypassing the external and middle ear. If sensorineural responses are intact a normal trace will be produced that is similar in form to that produced by normal air conduction. Unfortunately bone stimulation is more demanding to perform than air conduction and is more uncomfortable for the dog. It may thus be better undertaken on a sedated or anaesthetized animal. The maximum acoustic output from a bone vibrator is less than the click generated by air conduction. The mean threshold for bone conduction is only 50–60 dB whereas the air conduction threshold is said to be about 0–10 dB. This can make interpretation of bone conduction results difficult. However, because the wave forms and inter-peak latencies for both procedures are similar, it suggests both signals have the same source.

3.6 AUDITORY TYMPANOMETRY

Tympanometry is a non-invasive technique that is used to study changes in the compliance of the tympanic membrane which occurs as a result of pressure changes within the external ear canal. Tympanometry can be used to assess the integrity of the tympanic membrane and also to look for the presence of an effusion within the middle ear. The principle behind its use is that the intensity of a sound wave is dependent upon the cavity in which it is generated and the stiffness of the walls of the cavity. In a normal ear where the eardrum is intact a very different sound wave is produced compared to an ear where the tympanum is ruptured. The technique is difficult to perform effectively due to problems that can occur with lining up the equipment with the tympanum and also due the presence of a poor seal between the cuff and wall of the vertical canal. It is therefore a technique that is generally only available in referral institutions.

VetBooks.ir

VetBooks.ir

CHAPTER 4
DISEASES OF THE EAR PINNA

Sue Paterson

4.1	Erythematous Diseases	51	4.5	Nodular Diseases	70
4.2	Diseases with Crust and Scale	55	4.6	Ulcerative Diseases	72
4.3	Papular Diseases	63	4.7	Diseases with Alopecia	81
4.4	Pustular Diseases	67			

Many of the diseases of the ear start with lesions on the external ear pinna. It is important when examining the ear to look for primary lesions which can give a clue to the underlying aetiology of the disease. Primary lesions can be arranged into seven different classes (see box) and although there is some overlap among the classes, this acts as a useful starting point in defining a list of differential diagnoses. Differential diagnoses for each disease includes the other diseases presenting with the same clinical signs within the group.

CLASSES OF PRIMARY LESIONS

1. Erythematous diseases
2. Diseases with crust and scale
3. Papular diseases
4. Pustular diseases
5. Nodular diseases
6. Ulcerative diseases
7. Diseases with alopecia

A range of diagnostic tests can be employed to investigate each disease. The reader is referred to Chapter 2 for details of these.

1. Erythematous diseases
   - Allergy
   - Immune-mediated disease
2. Diseases with crust and scale
   - Infections
   - Endocrine disease
   - Environmental disease
   - Keratinization disorders
   - Neoplasia
3. Papular diseases
   - Ectoparasites
4. Pustular diseases
   - Infectious pustular disease
   - Sterile pustule disease
5. Nodular diseases
   - Infectious nodular diseases
   - Non-infectious nodular diseases
6. Ulcerative diseases
   - Autoimmune disease
   - Immune-mediated disease
   - Infections
   - Neoplasia
7. Diseases with alopecia
   - Alopecia without primary lesions
   - Alopecia with comedones

4.1 ERYTHEMATOUS DISEASES

4.1.1 Allergy

Allergic skin disease typically affects the ear pinna. Atopy, food allergy, and allergic and irritant contact dermatitis can all affect this site. It is unusual for dogs and cats with flea allergic dermatitis to present with pinnal signs although sticktight fleas can attach to this site.

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

VetBooks.ir

Atopy

Aetiology and Pathogenesis

The most recent theories on the pathogenesis of atopy suggest (to put it simply) that environmental allergens access and are processed by Langerhans cells in the skin, after percutaneous absorption. A T helper 1 (Th1) and T helper 2 (Th2) lymphocyte imbalance in atopic animals leads to amongst other things increased Th2 activity and overproduction of immunoglobulin (IgE). The resulting cascade of events triggers inflammation and pruritus. In addition, atopic animals are also thought to have epidermal barrier defects.

Clinical Signs

Clinical signs generally start between 1–3 years of age. Breed predisposition depends on the local gene pool. Where sensitivity is to pollen the otitis is likely to be seasonal. Animals allergic to house dust mites may have perennial disease but in Europe are often worse in the late autumn and early winter. In addition to otitis animals may have involvement of the skin in the periocular, feet, perianal, groin and axilla areas. In 80% of atopic dogs there are signs of otitis externa, usually with generalized skin disease. However, 20% of atopic dogs only have otitis externa, usually affecting both ears. Typical clinical signs are erythema of the pinna (Figs 4.1, 4.2) and vertical canal. Dogs will present with head shaking (Figs 4.3, 4.4)or rubbing the side of their heads. In some cases this can lead to aural haematoma formation (Fig. 4.5). Chronic changes on the ear pinna in uncontrolled disease include hyperpigmentation and lichenification (Fig. 4.6). Atopic cats may also present with otitis externa. Clinical signs include head



Figure 4.2 Erythema of ear pinna in an atopic dog.



Figure 4.3 Mild trauma to ear tip due to head shaking in an atopic dog.



Figure 4.1 Self-inflicted trauma and erythema on the ear pinna of an atopic dog.

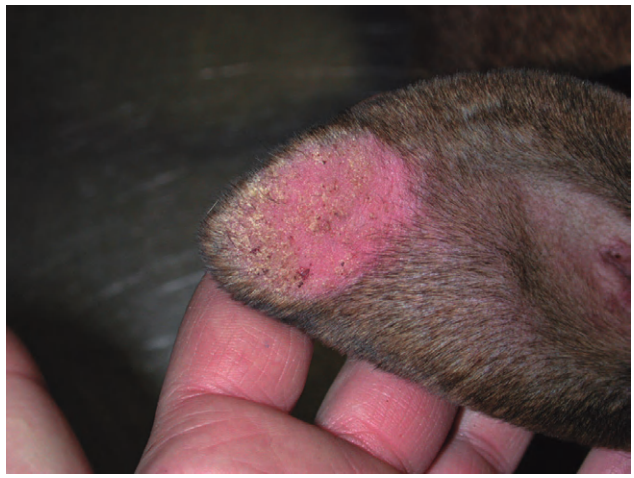


Figure 4.4 Marked trauma to ear tip due to head shaking in an atopic dog.



Figure 4.5 Aural haematoma on ear of dog due to head shaking.

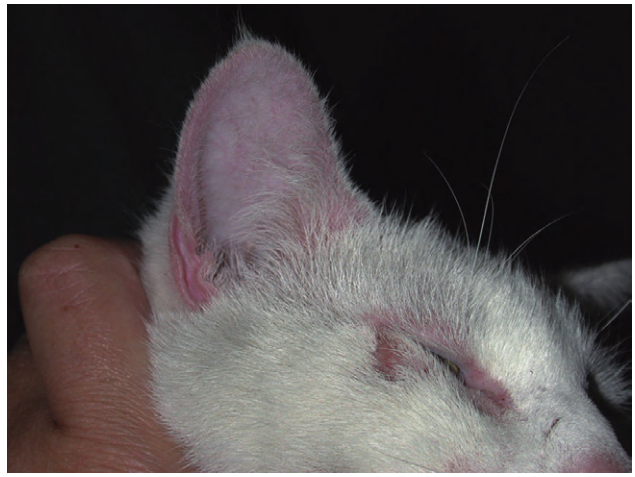


Figure 4.7 Erythema of ear pinna of an atopic cat.



Figure 4.6 Chronic change on the ear of an atopic dog.



Figure 4.8 Erythema of ear pinna of allergic Labrador with a cutaneous adverse food reaction.

shaking and erythema of the ear pinna (Fig. 4.7) or lesions of the eosinophilic granuloma syndrome.

Key Diagnostic Tests

- Rule out of other causes of pruritus, especially ectoparasites and infection.
- Rule out cutaneous adverse food reaction and allergic/irritant dermatitis.
- Specific intradermal or serologic allergy tests are useful in the management but not the diagnosis of the disease.

Food Allergy

Aetiology and Pathogenesis

Food allergy or cutaneous adverse food reaction (CAFR) is caused by an abnormal response to ingested food. Many different mechanisms exist including immunological components, metabolic reactions and food idiosyncrasies. Beef, chicken, dairy products, cereals, soya and egg have been implicated as common causes of CAFR in the dog. Similar substances are thought to be important in the cat. CAFR commonly coexists with atopy.

Clinical Signs

It is reported that 88% of food allergic dogs have signs of otitis externa. Food allergy commonly affects young dogs but it also seen in older animals with no previous history of skin disease. Labradors may be predisposed (Fig. 4.8). In dogs clinical signs commonly mimic those of atopic disease with signs of pinnal erythema and head shaking. Primary

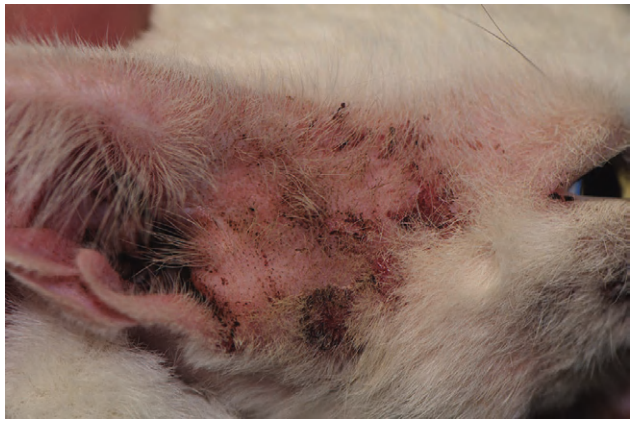


Figure 4.9 Facial and aural pruritus in a cat with food allergy.

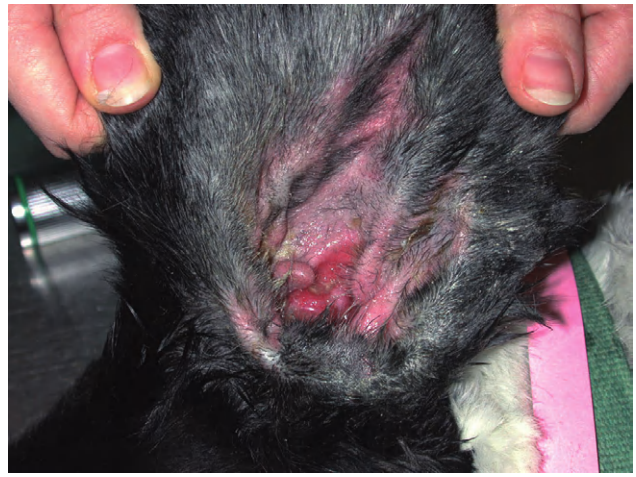


Figure 4.10 Erythema and ulceration of ear canal in a dog with irritant contact dermatitis due to ear drops.



Figure 4.11 Close-up of Figure 4.10.

lesions are uncommon. Unlike many cases of atopic disease, CAFR is not seasonal. Cats with food allergy typically present with facial pruritus, crusting, and secondary excoriation; the ear may be involved (Fig. 4.9).

Key Diagnostic Tests

- Diet trial using novel ingredients to which the dog or cat has had no or minimal exposure, or a hydrolysed diet, should be fed for up to 8 weeks.
- Dietary challenge should be performed once resolution has been achieved.

Allergic and Irritant Contact Dermatitis

Aetiology and Pathogenesis

These two conditions are very similar and are caused when an environmental substance has direct contact with the skin. Substances that affect the ear pinna and canal are generally liquids (creams, gels, lotions, etc.). Allergic contact dermatitis is a type IV hypersensitivity whereas irritant contact dermatitis is caused by noxious substances. Any topical medication may cause a reaction. However, neomycin and propylene glycol, which is a common component of many different ear drops and cleaners, have been implicated as causal agents.

Clinical Signs

Both of these conditions are less common than both food allergy and atopy as a cause of otitis externa. Allergic contact dermatitis takes multiple exposures to induce a reaction and affects individual animals. Irritant contact dermatitis causes a reaction on the first exposure and affects all in-contact animals. Typically dogs and cats will fail to respond to rational topical drugs and their disease deteriorates as therapy continues. Clinical signs usually start 1–3 days after the start of therapy. Lesions consist of erythema, oedema, erosions and in severe cases ulceration (Figs 4.10, 4.11).

Key Diagnostic Tests

- Ruling out of other allergic triggers, removal of topical therapy with subsequent improvement.
- Rechallenge should be avoided.

4.1.2 Immune-Mediated Disease

Feline Relapsing Polychondritis (Auricular Chondrosis)

Aetiology and Pathogenesis

Very rare, poorly understood disease thought to be an immune-mediated attack against collagen. Cats are often

VetBooks.ir



Figure 4.12 Feline relapsing polychondritis.

feline leukaemia virus (FeLV) or feline immunodeficiency virus (FIV) positive. It has also been recorded in dogs.

Clinical Signs

The ear pinna is swollen, erythematous to violaceous and painful (Fig. 4.12). In more chronic cases the pinna may be curled and deformed. Involvement of other organs, e.g. the joints, heart and eyes, have been described. Some cats may show signs of pyrexia and lethargy.

Key Diagnostic Tests

- Bloods show neutrophilia, lymphocytosis, hyperglobulinaemia.
- Biopsy of the pinna reveals a lymphoplasmacytic inflammation with loss of cartilage, basophilia and cartilage necrosis.

4.2 DISEASES WITH CRUST AND SCALE

4.2.1 Infections

The most common infectious diseases to present with crusting and scaling are dermatophytosis, yeast infections and leishmaniasis.

Dermatophytosis

Aetiology and Pathogenesis

In dogs the most common causes for dermatophytosis are *Microsporum canis* and *M. gypseum*. Other less frequent species include *Trichophyton mentagrophytes*, *M. persicolor* and *T. erinacei*. The Yorkshire Terrier is predisposed to *M.canis* infection, the Jack Russell Terrier is predisposed to *T. mentagrophytes* and *T. erinacei*. In the cat dermatophytosis is principally caused by *M. canis*; the Persian appears

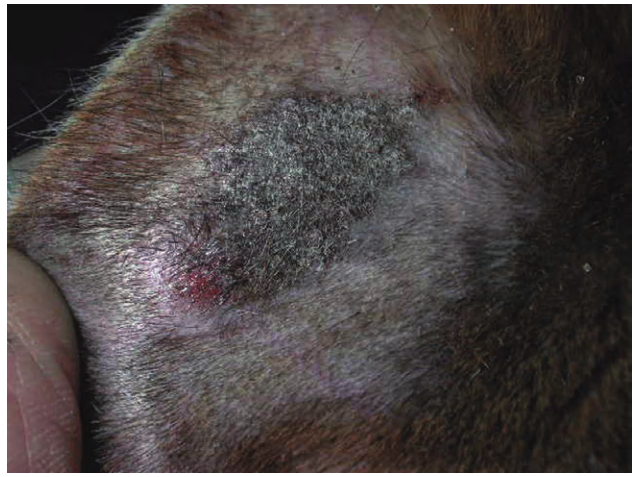


Figure 4.13 Well-circumscribed grey scaly lesions on the ear of a dog with ringworm.

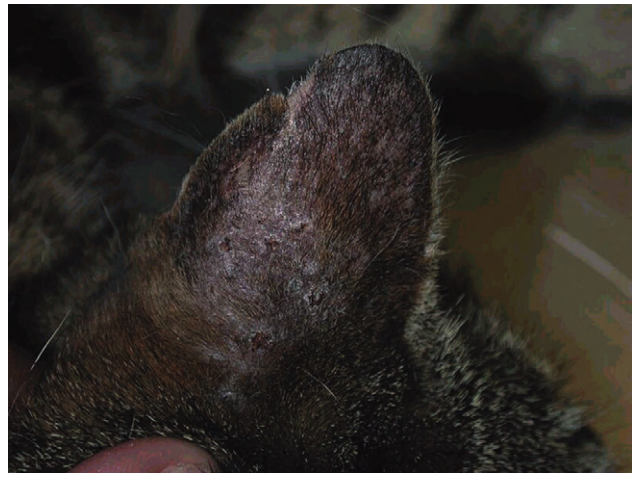


Figure 4.14 Ringworm on ear of kitten.

to be at increased risk. Infection is by contact with infected animals or a contaminated environment. The incubation period is 1–3 weeks. Immunocompromised individuals are predisposed, including young and elderly animals.

Clinical Signs

Infection with *M. canis* presents with fine grey 'ash-like' alopecic lesions (Fig. 4.13); pruritus is minimal (Fig. 4.14). *T. mentagrophtyes* infection produces more severe inflammation with alopecia, crusting and in severe cases furunculosis. Lesions are usually well demarcated.

Key Diagnostic Tests

- Trichography with hairs mounted in potassium hydroxide or lactophenol cotton blue.

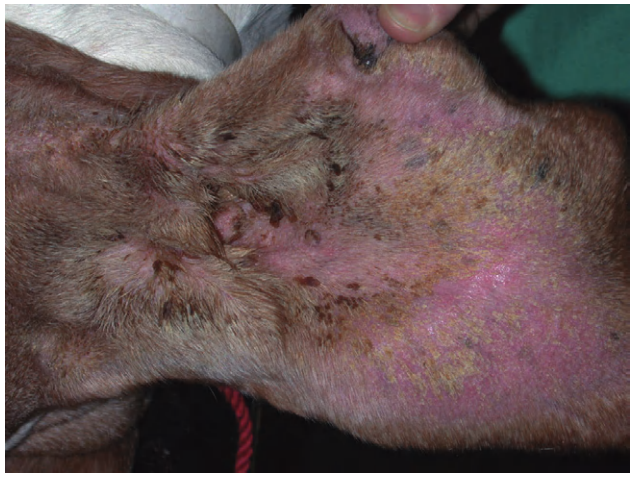


Figure 4.15 Malassezia infection on the ear of a Bassett.



Figure 4.16 Malassezia on the ear flap of an allergic dog.

- Fungal cultures taken with sterile toothbrush or carpet squares.

Yeast Infection

Aetiology and Pathogenesis

Malassezia yeasts are normal commensals of the canine and feline skin. Malassezia dermatitis is therefore usually secondary to an underlying condition. The West Highland White Terrier and Bassett (Fig. 4.15) appear to be overrepresented. Young dogs with malassezia infection frequently have underlying allergy. Endocrine disease is more common in older dogs as an inciting factor. In cats malassezia is usually seen secondary to endocrine disease, especially hyperthyroidism. In the dog the majority of infections are caused by *Malassezia pachydermatis*. This yeast has also been implicated in the cat as well as *M. sympodialis* and *M. furfur*. Candida yeast is a rare cause of skin disease in the dog or cat.

Clinical Signs

The ear pinna is erythematous and greasy with a yellow seborrhoeic appearance (Fig. 4.16), usually with a rancid, yeasty odour. In chronic disease the pinna becomes lichenified and hyperpigmented. Animals usually have a ceruminous otitis externa associated with signs on the ear pinna.

Key Diagnostic Tests

- Tape strippings, impression smears, cultures.

Leishmaniasis

Aetiology and Pathogenesis

Leishmaniasis is a rare disease in the United Kingdom but is common in Europe in dogs that live in or visit the Mediterranean basin. It is also found in southern Russia, India, China and eastern Africa. In the United States it has been identified in Oklahoma, Ohio and Texas. At least 30 different species have been identified in 5 different groups: *L. donovani, L. major, L. tropica, L. aethiopica and L. mexicana.* Leishmania is transmitted by blood-sucking sandflies (*Phlebotomus* spp. in Europe and Asia; *Lutzomyia* spp. in the Americas). Clinical signs can become apparent anything from 1 month to 7 years after exposure. It is rare in cats.

Clinical Signs

Clinical signs are very variable. Systemic signs can affect many different organs, and a full description is beyond the remit of this book. Cutaneous lesions affected 80% of dogs. The most common presentation on the ear pinnae is an exfoliative dermatitis with small adherent silvery scale (Fig. 4.17). Similar signs can be seen around the eyes, often referred to as periocular lunettes (Fig. 4.18). Dogs may also have nasodigital hyperkeratosis, nail disease, ulceration over pressure points, pustules, papules and nodules.

Key Diagnostic Tests

- Routine bloods: non-regenerative normochromic, normocytic anaemia, hypergammaglobulinaemia, hypoalbuminaemia.
- Organisms can be identified on cytology of scrapes or lymph node aspirates or from bone marrow or spleen.

VetBooks.ir

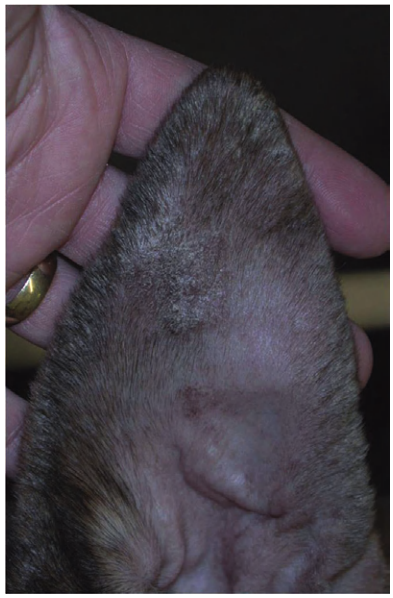


Figure 4.17 Scale on the ear flap of a dog with leishmaniasis.



Figure 4.18 Leishmaniasis in a dog showing generalized silvery scale. Courtesy of Kevin Camilleri.

- Histopathology: very variable pattern, organisms seen in 50% of cases with Giemsa stain.
- Leishmania-specific IgG detects presence of the organism but not necessary active disease.
- Polymerase chain reaction (PCR) highly sensitive.

4.2.2 Endocrine Disease

Hypothyroidism

Aetiology and Pathogenesis

In dogs approximately 90% of cases are due to primary hypothyroidism caused by the destruction of the gland.



Figure 4.19 Scaling on the periphery of an ear flap in a hypothyroid dog.

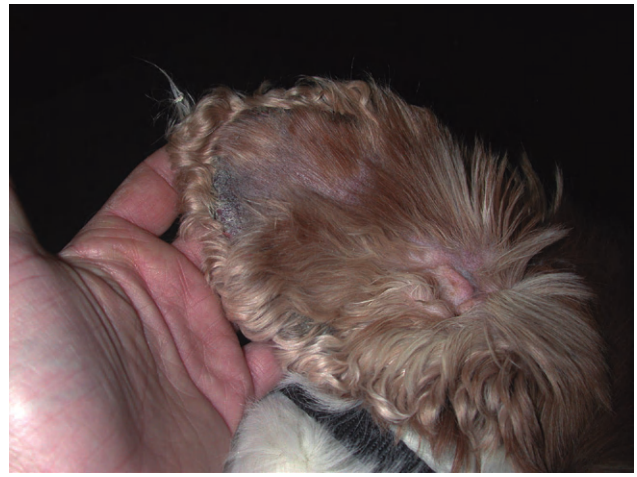


Figure 4.20 Alopecia and scaling on the ear flap of a hypothyroid dog.

This is caused by lymphocytic thyroiditis or idiopathic thyroid necrosis and atrophy. Predisposed breeds include Labrador and Golden Retrievers, Dobermanns, Bearded Collies and Beagles.

Clinical Signs

Clinical signs are variable, affecting many different organ systems. Common cutaneous signs include bilateral symmetrical truncal alopecia, 'rat tail', seborrhoea, secondary bacterial pyoderma. Signs affecting the ear pinnae include none pruritic peripheral scaling (Fig. 4.19), alopecia (Fig. 4.20) or in some cases hypertrichosis.

Key Diagnostic Tests

- Blood samples: mild non-regenerative anaemia, elevations in cholesterol, ALT, AP and creatinine kinase.

- Thyroid function tests used in combination, as a single test is rarely diagnostic. A panel should include total T_4, freeT_4, thyroid stimulating hormone and thyroglogulin autoantibodies.

4.2.3 Environmental Disease

Actinic Change

Aetiology and Pathogenesis

Actinic skin damage is caused by prolonged exposure to ultraviolet light (UV). Lesions can affect the periocular, nasal and pinnal skin. Typically it is seen in elderly dogs and cats with white pinnae. Chronic damage can lead to the development of oncogenic change such as squamous cell carcinoma and haemangio/sarcoma.

Clinical Signs

Initial signs are of erythema and thickening of the pinnae with fine scaling (Fig. 4.21). Chronically the tips of the pinnae can become curled and scarred and cutaneous horn may develop. As neoplastic transformation occurs the ear pinna become ulcerated with marked proliferative changes.

Key Diagnostic Tests

- Biopsy reveals signs of superficial fibrosis and follicular keratosis typical of actinic change

4.2.4 Keratinization Disorders

Primary keratinization disorders are very uncommon causes of skin disease in the dog and rare in the cat. However, when they occur the pinna is commonly affected and in some cases only the ear pinna is affected.

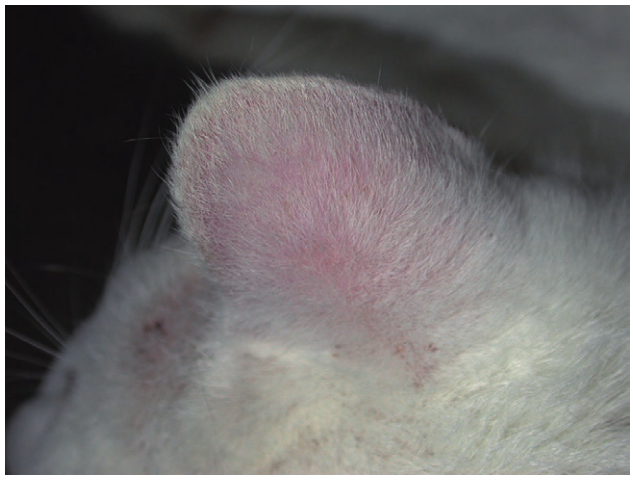


Figure 4.21 Actinic change on a cat's ear pinnae.

Primary Idiopathic Seborrhoea

Aetiology and Pathogenesis

Most commonly, but not exclusively, recognized in the Cocker Spaniel (Fig. 4.22). It is caused by an idiopathic increase in epidermal turnover. The transit time of cells from the basal cell layer to the stratum corneum is shortened to 7–8 days (normal dog 21 days).

Clinical Signs

Affected dogs have thick greasy, adherent, yellowy/orange scale around the external ear canal extending on to the ear pinna. Follicular casts may be present on hairs. Animals tend to be malodorous and pruritic often with secondary infection with malassezia or pyoderma. Otitis externa is common. Chronically the ear canal can become lichenified (Fig. 4.23). Other areas that are involved include nipples, lip folds, ventral neck, trunk and feet.

Key Diagnostic Tests

- Predisposed breed.
- Biopsy only supportive, not diagnostic.
- Non-specific changes include a hyperplastic superficial perivascular dermatitis with ortho/parakeratosis, follicular keratosis and variable dyskeratosis.
- Other causes of seborrhoea must be eliminated.



Figure 4.22 Primary idiopathic seborrhoea in a Cocker Spaniel.

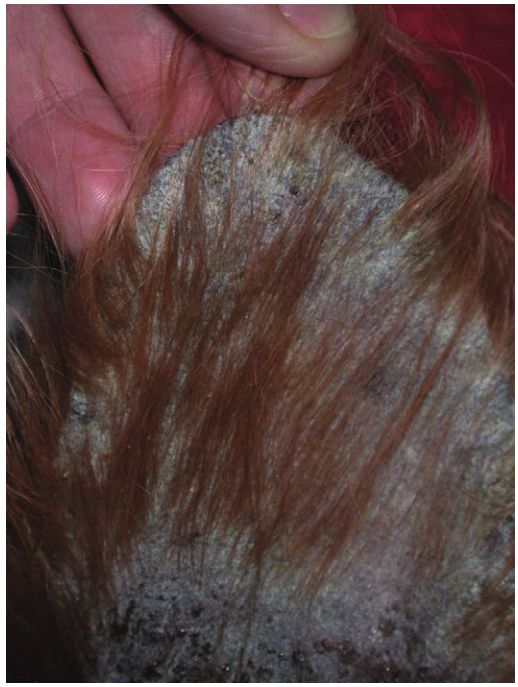


Figure 4.23 Primary idiopathic seborrhoea in a Cavalier King Charles Spaniel.

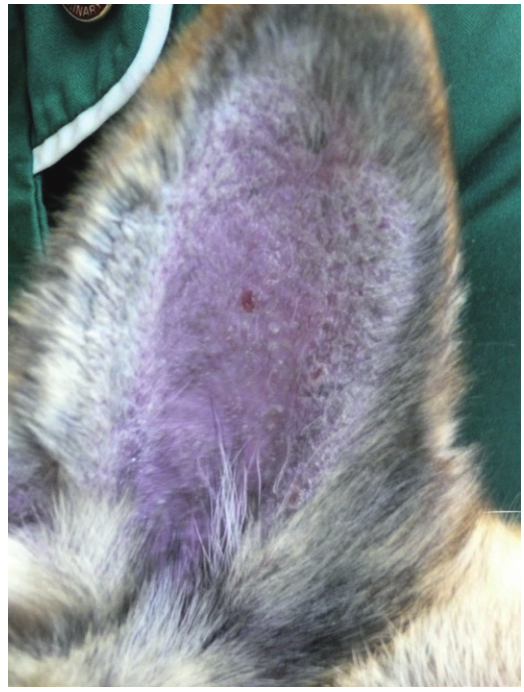


Figure 4.24 Sebaceous adenitis on the ear of an Akita.

Sebaceous Adenitis

Aetiology and Pathogenesis

Uncommon disease of unknown aetiology caused by loss of sebaceous glands. Theories include an autoimmune reaction against the glands or a primary structural defect of the gland leading to leakage of sebum and a foreign body reaction. Predisposed breeds include Standard Poodles, Akitas (Fig. 4.24), Vizslas and Samoyeds.

Clinical Signs

Signs vary between breeds but lesions on the ear pinna usually take the form early on of scaling and thinning of the hair. There are usually prominent follicular casts around the base of the hairs (Fig. 4.25). Chronically the skin becomes more involved, with tightly adherent silver-white scale and tufted, matted hair. A dry ceruminous otitis externa with secondary infection is common.



Figure 4.25 Chronic sebaceous adenitis on the ear flap of a Labrador.

Key Diagnostic Tests

- Trichography reveals typical follicular casts. Multiple biopsies are needed.
- Early signs show a multifocal mixed inflammatory infiltrate (granulomatous–pyogranulomatous) around the sebaceous glands.
- Advanced cases have moderate acanthosis, hyperkeratosis and lack of sebaceous glands. Perifollicular granulomas are often found in the areas normally occupied by the sebaceous glands.

Ear Margin Seborrhoea

Aetiology and Pathogenesis

This is thought to be a primary keratinization defect of unknown aetiology that affects the margins of the ear. Dogs with pendulous ear pinnae appear to be predisposed, especially Dachshunds, Springer Spaniels and Cocker Spaniels.



Figure 4.26 Ear margin seborrhoea in a Cocker Spaniel.



Figure 4.27 Exfoliative cutaneous lupus erythematosus in a German Short-Haired Pointer.

Clinical Signs

Early signs are of excessive adherent keratinous debris accumulating along the margins of the ear (Fig. 4.26). Chronically, material can build up along the margins of the ears leading to fissuring.

Key Diagnostic Tests

- Clinical signs in a predisposed breed.
- Biopsy is supportive rather than diagnostic, showing signs of surface and follicular ortho/parakeratotic hyperkeratosis.
- Other differentials should be ruled out.

Exfoliative Cutaneous Lupus Erythematosus of the German Short-Haired Pointer

Aetiology and Pathogenesis

A possible hereditary disease of the German Short-Haired Pointer caused by an immune-mediated reaction against basal epithelial cells.

Clinical Signs

Signs are first recognized in young dogs of 5–8 months of age. Initially signs consist of scaling and thinning of the hair coat on the face, back and ears (Figs 4.27, 4.28). The disease can become generalized. Prominent follicular casting is seen with more diffuse scaling.

Key Diagnostic Tests

- Clinical signs in a predisposed breed.
- Biopsy reveals signs of ortho/parakeratotic hyperkeratosis, hydropic degeneration of basal cells and individual keratinocyte necrosis throughout the stratum corneum and a mixed cellular mild to moderate interface dermatitis.



Figure 4.28 Exfoliative cutaneous lupoid erythematosus on the ear pinna of a German Short-Haired Pointer.

Idiopathic Facial Dermatitis in Persian and Himalayan Cats

Aetiology and Pathogenesis

A disease of unknown aetiology. Some authorities suggest it may be a keratinization disorder involving the sebaceous glands. Age of onset is variable, ranging from 10 months to 6 years.

Clinical Signs

Accumulations of thick dark waxy debris is seen around the eyes, facial fold, chin and ears (Fig. 4.29). Cats commonly have a bilateral erythematous otitis with accumulations of black/brown debris on the ear pinna (Fig. 4.30).



Figure 4.29 Idiopathic facial dermatitis in a Persian cat.

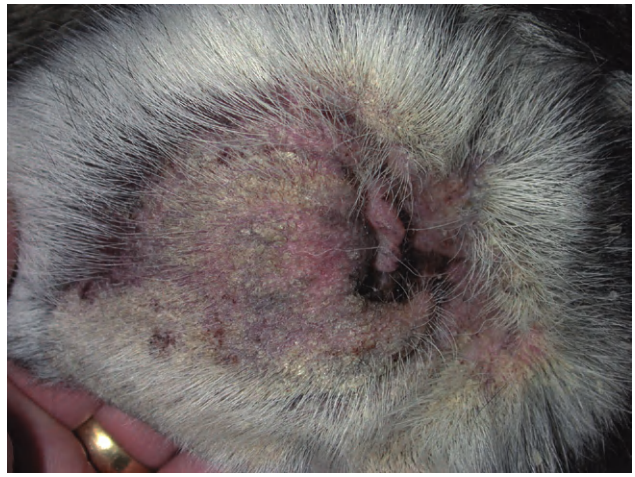


Figure 4.31 Ear flap of a Husky with zinc-responsive dermatosis.

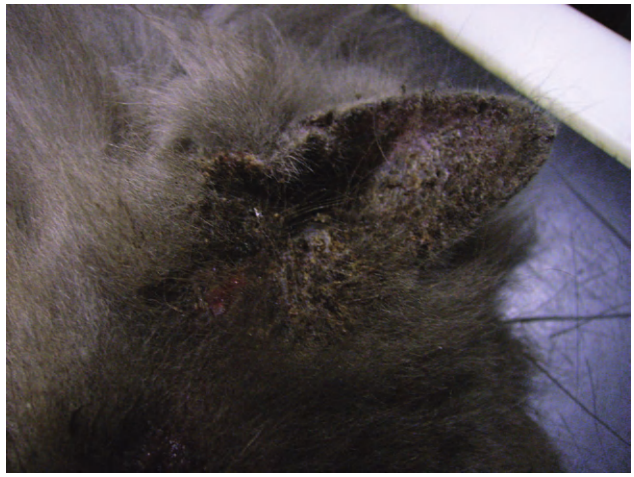


Figure 4.30 Ceruminous otitis in a Persian cat with idiopathic facial dermatitis.



Figure 4.32 Husky with zinc-responsive dermatosis showing typical periocular lesions.

Secondary infection with yeast and bacteria is common and often leads to intense pruritus.

Key Diagnostic Tests

- Clinical signs in a predisposed breed.
- Biopsy reveals acanthosis with superficial crusting. Hydropic degeneration of basal cells and occasional dyskeratotic keratinocytes especially in the follicular epithelium is seen, with sebaceous gland hyperplasia.

Zinc-Responsive Dermatoses

Aetiology and Pathogenesis

Zinc-responsive dermatosis occurs as a result of an impaired ability to utilize zinc. In type I disease, seen in the Siberian Husky and Alaskan Malamute, disease is caused by an inadequate absorption of zinc from the bowel. Type II disease is seen in fast-growing giant breed puppies which are fed an unbalanced diet. A relative deficiency of zinc occurs due to competition for absorption with zinc by high levels of cereal, calcium, copper or iron. Intestinal phytate and inorganic phosphate also bind zinc and hinder absorption.

Clinical Signs

Typically the ear pinna is erythematous and covered with fine silvery scale (Fig. 4.31). Dogs usually also have footpad hyperkeratosis and similar cutaneous lesions around the eyes (Fig. 4.32) and over pressure points. Pruritus is variable; secondary pyoderma is common.

Key Diagnostic Tests

- Type I predisposed breeds, type II history of unbalanced diet.
- Tape stripping of skin reveals nucleated keratinocytes consistent with parakeratosis.
- Biopsy confirms acanthosis with diffuse parakeratosis.
- Response to therapy often diagnostic.

4.2.5 Miscellaneous

Psoriasiform–Lichenoid Dermatosis of the English Springer Spaniel

Aetiology and Pathogenesis

A scaling disease of the ear pinna of young English springer spaniels of unknown aetiology. The complete response that has been reported to cephalexin therapy suggest it may be an immune-mediated reaction to staphylococcus.

Clinical Signs

Characterized by erythematous papules and crusts with hyperkeratotic plaques on the concave aspect of the ear pinna. The external ear canal, head and ventral trunk may also be affected. Lesions may wax and wane with time, becoming more hyperkeratotic over several years, often assuming a papillomatous appearance chronically.

Key Diagnostic Tests

- Clinical signs and predisposed breed.
- Histopathology reveals a superficial perivascular to interstitial dermatitis with psoriasiform epidermal hyperplasia and areas of lichenoid interface dermatitis.

4.2.6 Neoplasia

Epitheliotropic Lymphoma

Aetiology and Pathogenesis

Most neoplastic diseases that affect the ear flap produce nodules, plaques or ulcers. However epitheliotropic lymphoma (EL) in the early stages of the disease can present with diffuse scaling. EL is caused by malignant transformation of T lymphocytes; atopic dermatitis is thought to be a risk factor in dogs. The neoplastic lymphocytes infiltrate the upper epidermis and dermis.

Clinical Signs

Dogs are usually elderly and scaling can affect the whole body. EL is a very pleomorphic disease : commonly dogs and cats present with a pruritic erythroderma with plaques of silver-white scale. The ear pinna in these cases is thickened and hyperkeratotic with plaques and ulceration (Fig. 4.33).



Figure 4.33 Silvery scale on the face and ears of a dog with epitheliotropic lymphoma.

Key Diagnostic Tests

- Impression smear shows abundant neoplastic lymphoid cells with basophilic cytoplasm and pleomorphic indented to lobular nuclei.
- Biopsy reveals a lichenoid band of pleomorphic neoplastic lymphocytes that infiltrate superficial dermis. Neoplastic cells may form within small vesicles in the epidermis (Pautrier's microabscesses).

Paraneoplastic Exfoliative Dermatitis (PED)

Aetiology and Pathogenesis

PED is a paraneoplastic syndrome associated with the development of thymoma in cats. Skin lesions regress if the tumour is removed. The pathogenesis is poorly understood; it is possibly an autoimmune cell-mediated process.

Clinical Signs

Affects older cats (>10 years) Initial lesions are non-pruritic and erythematous becoming exfoliative in the first instance on the head, neck and pinnae (Fig. 4.34) then generalizing. Cats often show signs of coughing, dyspnoea, lethargy and anorexia.

Key Diagnostic Tests

- Clinical appearance and identification of a thoracic tumour.
- Skin biopsies reveal an interface dermatitis with hydropic degeneration of basal cells, apoptosis of keratinocytes, satellitosis, and a lymphocytic dermal infiltrate.

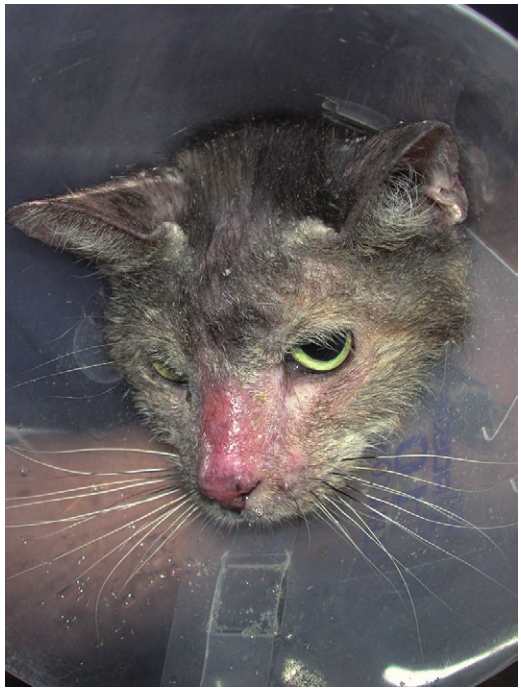


Figure 4.34 Fine scale on the face and ears on a cat with a thymoma.

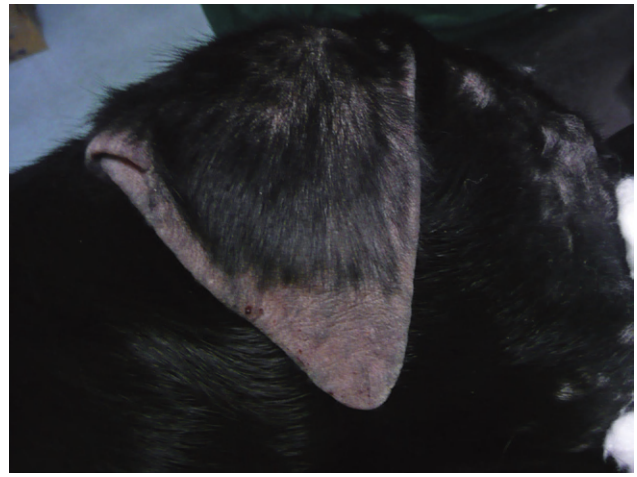


Figure 4.35 *Sarcoptes scabiei* affecting the pinnal margins.



Figure 4.36 Crust and scale on the ear pinna of a puppy with sarcoptes.

4.3 PAPULAR DISEASES

4.3.1 Ectoparasites

Ectoparasitic conditions of the ear pinna present with papular eruptions. Many of these diseases do not cause otitis externa but can lead to head shaking and self-inflicted trauma directed to the ear pinna and the side of the face.

Sarcoptes Scabiei

Aetiology and Pathogenesis

A common, highly contagious disease of dogs, rare in cats caused by *Sarcoptes scabiei*. Adult mites burrow through the epidermis, particularly in sparsely haired areas, laying eggs. Life cycle 14–21 days. Adult females typically survive 2–6 days off the host.

Clinical Signs

Typically dogs exhibit acute onset pruritus. Predilection sites include the pinnal margins (Fig. 4.35) and extensor aspects of elbows and hocks. Lesions appear as yellowy/grey crust along the periphery of the ear pinna (Fig. 4.36) and papular lesions on the pinna, often with marked secondary self-inflicted trauma. It is commonly seen in gun dogs and often associated with contact with foxes. Otitis externa is rare.

Key Diagnostic Tests

- Most dogs have a strong pedal pinnal reflex when the tip of the ear is scratched.
- Deep skin scrapings to identify mites (Fig. 4.37), faecal pellets, or eggs but this has poor sensitivity.
- Serology for sarcoptes specific IgG 4 weeks after infestation. False positives can occur in atopic dogs due to cross-reactivity with *Dermatophagoides* spp. house dust mites.
- Response to appropriate scabicidal therapy.

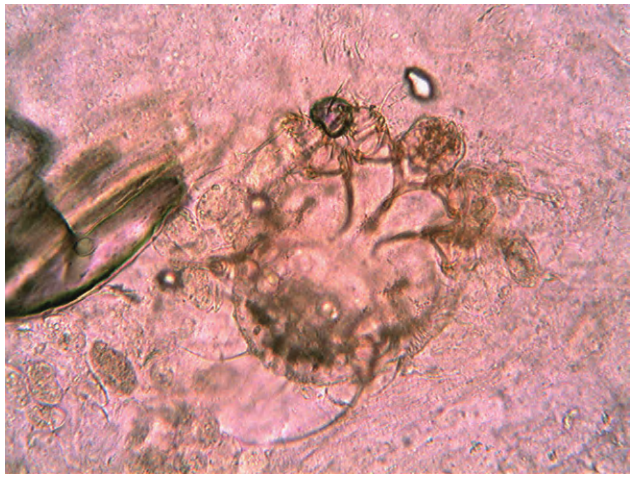


Figure 4.37 *Sarcoptes scabiei* mite.

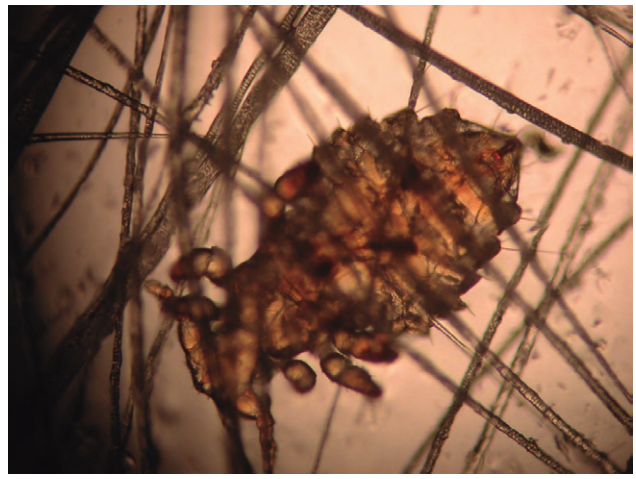


Figure 4.39 *Trichodectes canis.*

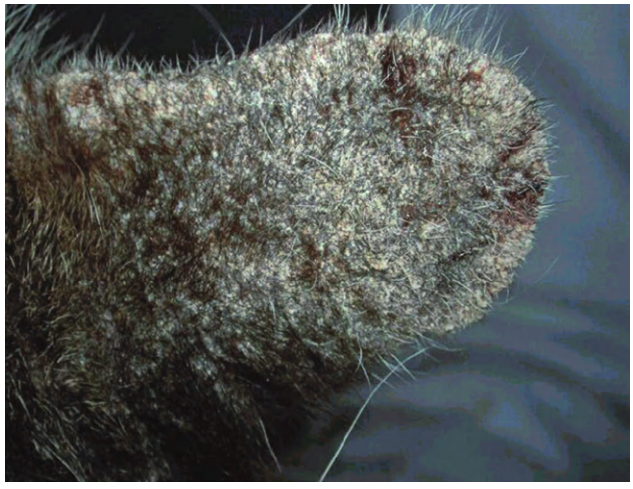


Figure 4.38 Ear pinna of a cat with notoedres.



Figure 4.40 *Felicola subrostratus.*

Notoedres Cati

Aetiology and Pathogenesis

A highly contagious ectoparasite infestation of cats is caused by the sarcoptiform mite *Notoedres cati*. Principally a disease of cats, it will also affect dogs and people. The periphery of the ear pinna is a predilection site.

Clinical Signs

A highly pruritic disease affecting the head and ear pinnae, often referred to as 'head mange'. Lesions typically appear as thick crusted papules which can coalesce to form thick crust, often with excoriation and alopecia of the convex surface of the ear pinna (Fig. 4.38).

Key Diagnostic Tests

- Deep skin scrapings reveal adult mites, immature larvae, and eggs.
- Response to appropriate therapy.

Pediculosis

Aetiology and Pathogenesis

Commonly seen in young and debilitated animals, especially those housed in groups. Lice are host specific but can transiently infest other hosts. Canine lice include the biting louse *Trichodectes canis* (Fig. 4.39) and the sucking lice *Linognathus setosus* and *Heterodoxus spineger*. Cats are only affected by the biting louse *Felicola subrostratus* (Fig. 4.40). The life cycle takes 21 days and is completed on the host.

Eggs are laid on hairs. Transmission is by direct contact or by fomites.

Clinical Signs

The tips of the ears are common sites for infestation with lice. Lesions are variable but usually pruritic consisting of scaling, pruritus, alopecia, papules, and crusting. Gun dogs, especially spaniels, are commonly affected and may show signs of head shaking. Lice can also found at other mucocutaneous junctions or may be generalized.

Key Diagnostic Tests

- Tape strippings, hair plucks for operculated eggs cemented to hair shafts.
- Superficial skin scrapings for parasites.
- Biting lice have broad heads, sucking lice have longer, tapered heads.

Fleas

Aetiology and Pathogenesis

The rabbit flea *Spilopsyllus cuniculi* will feed around the tips of the ear of cats and occasionally dogs. It is particularly seen on cats that hunt rabbits. Fleas are transient parasites and are often tolerated by cats without producing any clinical signs. Fleas are most active during the summer.

Clinical Signs

Lesions appear as crusted papules around the periphery of the ear tip. Crusts often overlie thickened ulcerated ear tips (Figs 4.41, 4.42).

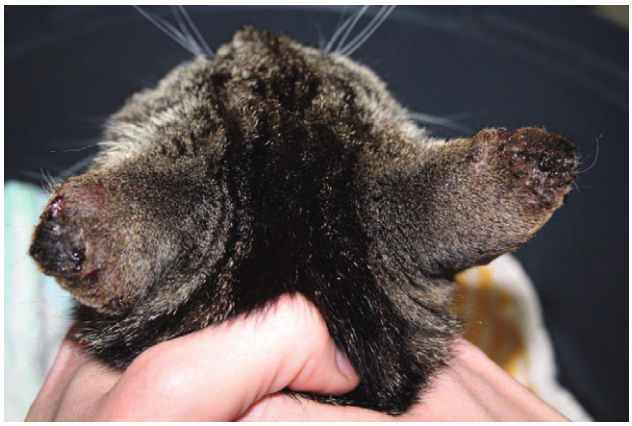


Figure 4.41 Papular lesions on the ear tips due to *Spilopsyllus cuniculi* (Picture courtesy of Jan Declercq).

Key Diagnostic Tests

- Fleas can be seen feeding on the ear tips.
- Differentiation between *Spilopsyllus cuniculi* and *Ctenocephalides felis* cannot be made on the basis of their pronotal and genal combs. Both are present in both species. However, the head of the *Spilopsyllus* is broader (Fig. 4.43) and the abdomen more rounded than *Ctenocephalides*.

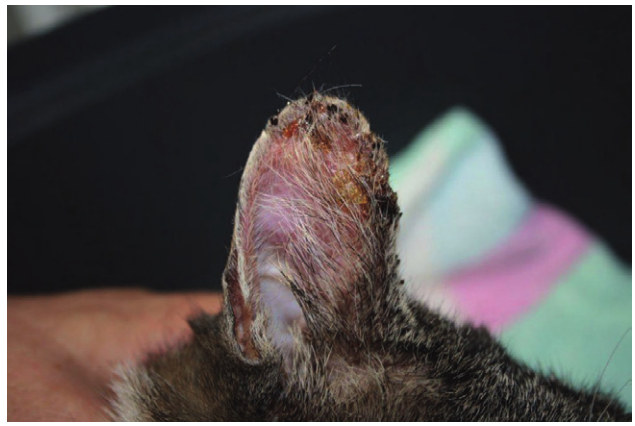


Figure 4.42 Papular lesion on ear tip of cat due to *Spilopsyllus cuniculi* (Picture courtesy of Jan Declercq).

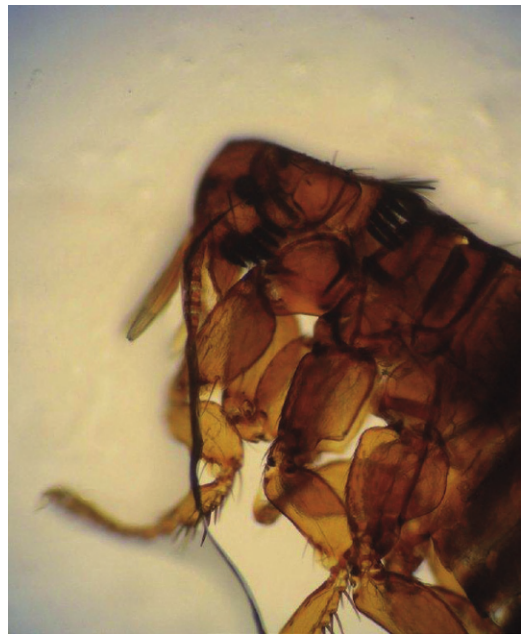


Figure 4.43 Broad-headed flea *Spilopsyllus cuniculi* (Picture courtesy of Jan Declercq).

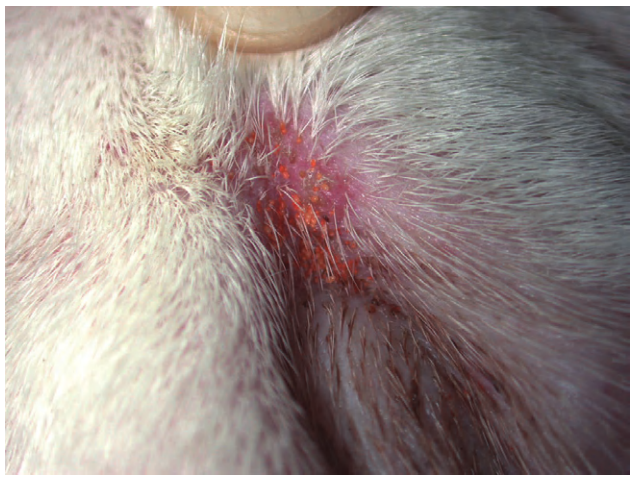


Figure 4.44 *Neotrombicula autumnalis* on the skin of a dog.



Figure 4.45 The parasitic six-legged larvae of *Neotrombicula autumnalis*.

- A wet paper test and blood samples are of minimal value for *S. cuniculi.*

Harvest Mites

Aetiology and Pathogenesis

The parasitic larvae of harvest mites *Neotrombicula autumnalis* and *Eutrombicula alfredugesi* can infest dogs and cats during the autumn months. Their feeding activity causes a papular rash on contact sites. Infestation is usually transient, lasting only a few days, but larvae can cause intense pruritus. Free-roaming animals are predisposed, especially where they have access to areas of rough vegetation.

Clinical Signs

Mites are seen on head, ears (Henri's pocket, small fold of skin at the base of the ear pinna), feet (interdigital spaces; Fig. 4.44) and ventrum. Examination of affected areas reveals clusters of orange/red larvae which have the appearance of red pepper grains to the naked eye. Lesions consist of papules, crust, scale, and alopecia.

Key Diagnostic Tests

- Bright red or orange six-legged larvae can be seen with the naked eye or collected on tape strippings or superficial skin scrapings and identified microscopically (Fig. 4.45).

Ticks

Aetiology and Pathogenesis

Dogs and cats can be affected by both hard ticks (ixodic) and soft ticks (argasid). Ixodic ticks are most commonly implicated as a cause of disease in domestic pets.

Clinical Signs

All stages of the life cycle after hatching (larva, nymph, adult) can attach to the dog or cat's skin. They enlarge in size as they engorge with blood. The head and the pinna are common sites of attachment. Commonly they fail to produce signs of cutaneous irritation but once they detach can leave a small papular/nodular lesion at the point of attachment.

Key Diagnostic Tests

- Ticks are large parasites and are visible with the naked eye.
- Biopsy of tick lesions reveals a central nodule of lymphoplasmacytic inflammation with a peripheral eosinophilia.

Fly and Mosquito-Bite Dermatosis

Aetiology and Pathogenesis

Biting flies such as *Stomoxys calcitrans*, mosquitos, or sand flies can feed on the ear pinnae of dogs and cats to produce a papulocrustous reaction. In cats, mosquito-bite hypersensitivity (MBH) is a type I reaction to mosquito saliva rather than being an irritant reaction to fly-bites as it is in the dog. It is particularly seen in short-haired cats with outdoor access.

Clinical Signs

In fly-bite dermatitis in dogs lesions appear as crusted papular eruptions, especially in animals with erect ears on the tips of the pinnae and those with drooping ears on the fold. They are particularly seen in the summer months. In cats with MBH lesions also occur in the summer but are



Figure 4.46 Lesions of mosquito-bite hypersensitivity on the head of a cat (Picture courtesy of Daniel Morris).



Figure 4.47 Methicillin-resistant staphylococcal infection on the ear pinnae of a dog.

more extensive, with papules on both the outer and inner aspect of the pinna (Fig. 4.46). Lesions are often generalized, involving the bridge of the nose, eyelids, chin, lips, and paw pads.

Key Diagnostic Tests

- A history of exposure to biting flies.
- A resolution of lesions with fly avoidance measures.
- In MBH there is in addition a peripheral eosinophilia and hypergammaglobulinaemia.
- Histopathology will differentiate between a MBH and autoimmune skin disease, the former reveals signs of superficial and deep eosinophilic dermatitis, eosinophilic folliculitis and furunculosis, and collagen degeneration.

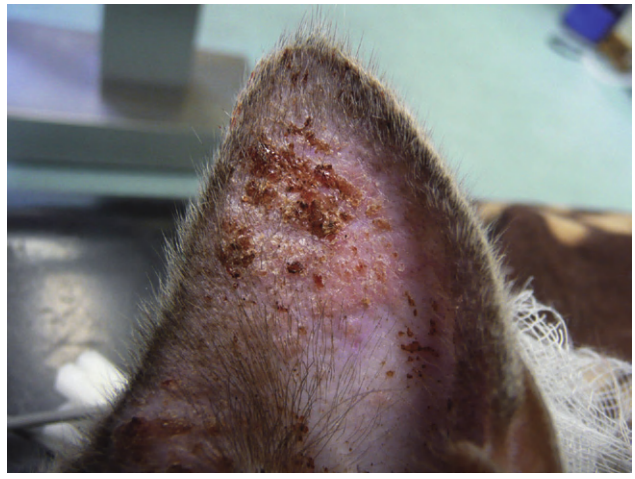


Figure 4.48 Staphylococcal infection on the ear pinnae of an allergic cat.

4.4 PUSTULAR DISEASES

4.4.1 Infectious Pustular Disease

Aetiology and Pathogenesis

Bacterial disease will cause pustular lesions on the ear pinnae of both dogs and cats. The most common pathogens are coagulase-positive staphylococcus, especially *Staphylococcus pseudintermedius* although *S. aureus*, *S. hyicus*, and *S.scheiferi* have also been implicated. Meticillin-resistant staphylococcal infections have also been identified as a cause of pyoderma including otitis in dogs and cats (Fig. 4.47). Staphylococcal infection is in most cases associated with an underlying disease and infection commonly also extends into the ear canal. In young dogs and cats parasites, allergy (Fig. 4.48) and keratinization disorders are frequent underlying triggers. In middle-aged and older animals endocrinopathies (Fig. 4.49) and metabolic disease may be an inciting factor.

Clinical Signs

Pustules are a less common finding on the ear pinna in dogs and cats with infectious disease than with autoimmune disease. Most commonly dogs with infection of their ear pinnae present with erythema, scale, and crusting with occasional papules and pustules. In severe cases the ear pinna may be ulcerated, which can mimic autoimmune skin disease (Fig. 4.50). The periphery of the pinna is usually spared.



Figure 4.49 Severe staphylococcal infection on the ear of a dog with hypothyroidism.



Figure 4.50 Severe ulceration of ear pinnae in an undiagnosed staphylococcal infection.

Key Diagnostic Tests

- Stained impression smears, pustule cytology (degenerate neutrophils and cocci), culture and sensitivity.

4.4.2 Sterile Pustule Disease

Primary pustular lesions are more commonly seen in sterile pustular disease than in infectious disease.

Pemphigus Foliaceus

Aetiology and Pathogenesis

Pemphigus is an autoimmune disease of dogs and cats where autoantibodies are thought to be directed against desmosomal components (part of the intercellular bridge between epidermal cells). Cell–cell cohesion is lost, resulting in acantholysis and pustule formation.

Clinical Signs

Predisposed breeds include Akita (Fig. 4.51), Chow Chow, Dobermann Pinscher, and Bearded Collie. Lesions commonly start on the face, especially the dorsal aspect of the nose and the ear pinna. Footpad hyperkeratosis is common in dogs, whereas a sterile purulent paronychia is seen in cats. Mucocutaneous lesions are rare. On occasions lesions can stay confined to the ear pinna. In dogs pustular lesions are common on the concave surface of the ear pinna, often with accompanying scale, alopecia, and erosions (Figs 4.52, 4.53). In cats pustules are less common but thick crusting is seen, especially around the periphery of the ear pinna which can lead to distortion of its outline (Figs 4.54, 4.55).

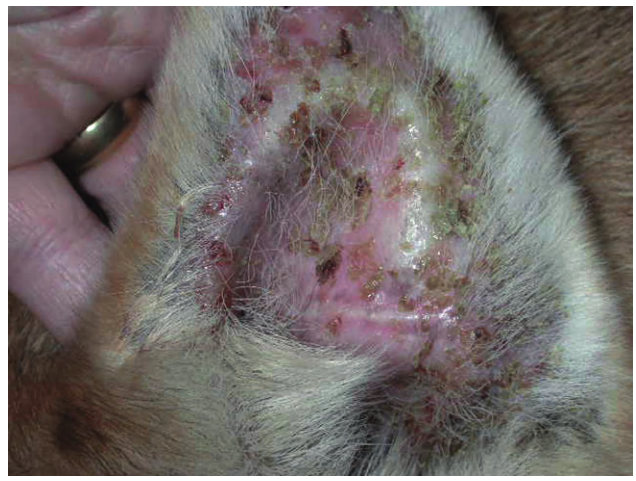


Figure 4.51 Crusts and pustules on the ear pinnae of an Akita with pemphigus foliaceus.

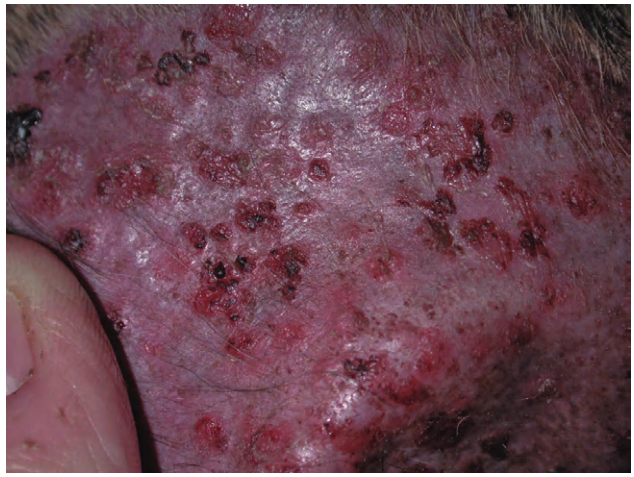


Figure 4.52 Pustules and crusts on the ear pinnae of a dog with pemphigus foliaceus.



Figure 4.53 Severe pemphigus foliaceus on the ear pinna of a Yorkshire Terrier.

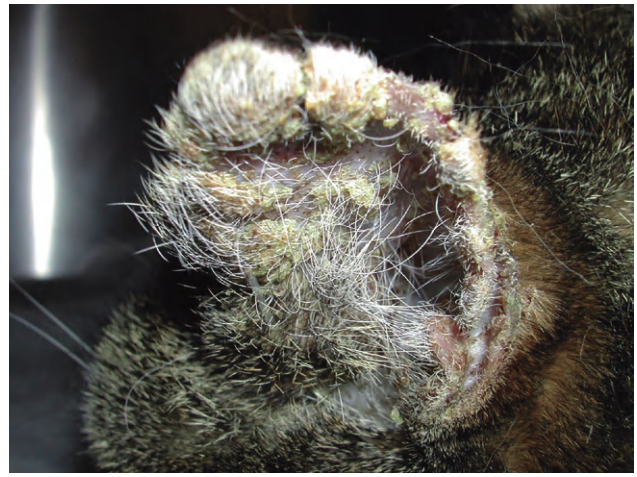


Figure 4.54 Thick crust on the ear pinna of a cat with pemphigus foliaceus.

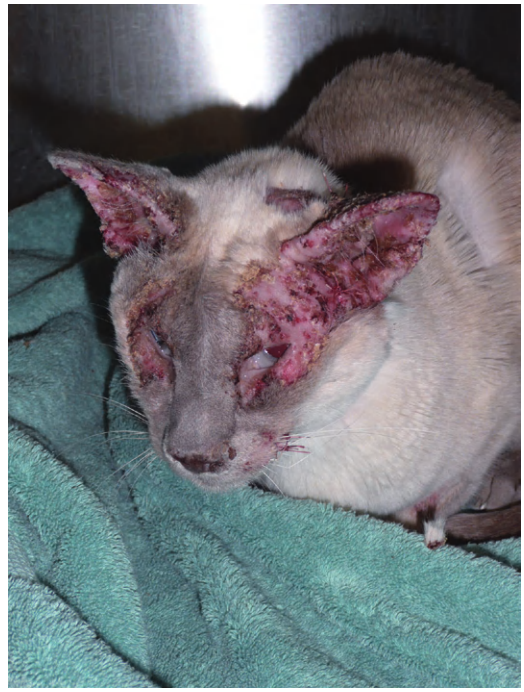


Figure 4.55 Pustules, crusting, and erosions on the face of a cat with pemphigus foliaceus.

Key Diagnostic Tests

- Stained impression smears from lesions or impression smears of the underside of crusts.
- Pustule cytology (non-degenerate neutrophils, rare bacteria, and acanthocytes).
- Biopsy of primary lesions reveals intragranular or subcorneal acantholysis with resultant cleft, vesicle, or pustule formation.
- Pustules contain non-degenerate neutrophils or eosinophils.

Canine Juvenile Cellulitis (Puppy Pyoderma, Puppy Strangles)

Aetiology and Pathogenesis

Immune-mediated disease of unknown aetiology which leads to the development of a pustular/granulomatous

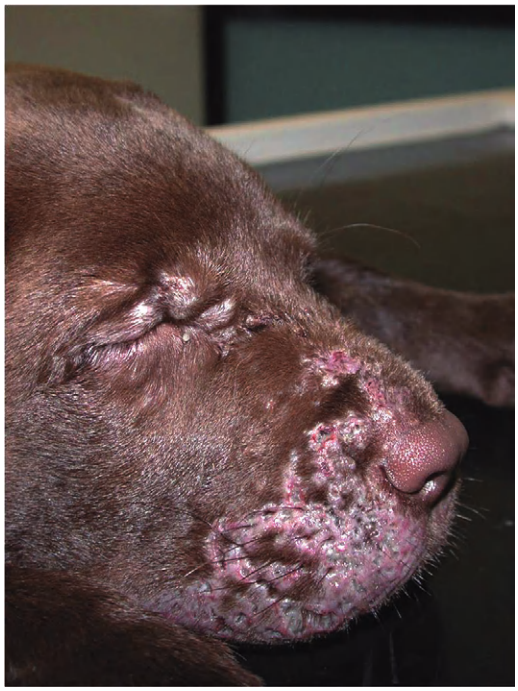


Figure 4.56 Swelling of the lips and muzzle in a case of juvenile cellulitis.

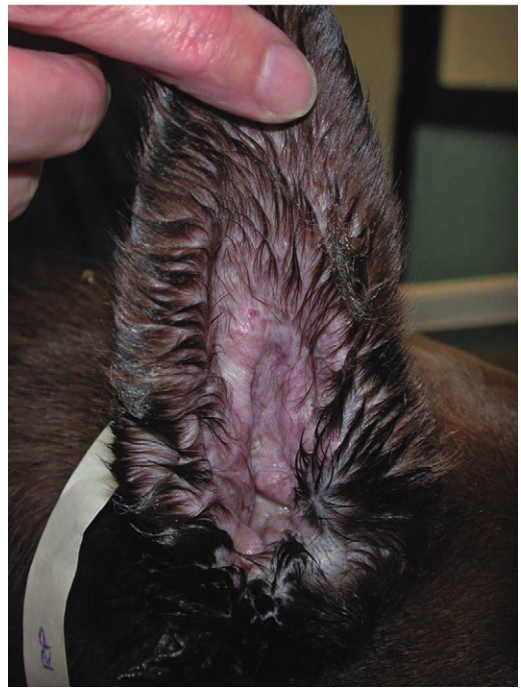


Figure 4.57 Sterile ulcerative otitis in canine juvenile cellulitis.

lesions on the face and ears of young dogs, usually 3 weeks to 4 months of age.

Clinical Signs

Predisposed breeds include Labrador and Golden Retriever, Dachshunds, and Gordon Setter. Dogs present with acute swelling of lips, muzzle (Fig. 4.56), eyelids, and ear pinnae. Sterile pustules are common, especially on the concave surface of the ear flap. These rupture to form ulcers with secondary exudation and crusting. A sterile ulcerative otitis externa is common (Fig. 4.57). Submandibular lymphadenopathy is usually present. Rarely, lesions can be seen on other areas as well as a sterile arthritis.

Key Diagnostic Tests

- Clinical signs.
- Pustule cytology (neutrophils, mononuclear cells, rare mixed secondary bacterial infection).
- Biopsies of early lesions reveal multiple discrete or confluent granulomas and pyogranulomas consisting of clusters of large epithelioid macrophages and variably sized cores of neutrophils.

Epithelitropic Lymphoma

See section 4.2.6.

EL can present as a pustular disease that can affect the ear pinnae.

4.5 NODULAR DISEASES

4.5.1 Infectious Nodular Diseases

Infectious disease rarely causes signs of nodular lesions on the ear pinna.

Bacterial Pyogranulomas

These can be caused by a range of organisms including staphylococcus, streptococcus (botryomycosis), actinomyces, and nocardia. The ear pinna is rarely a site of infection.

Fungal Granulomas

These can be caused by a range of organisms including *Cryptococcus neoformans* (cryptococcosis); alternaria, cladosporium, and others (phaeohyphomycosis); and *Sporothrix schenkii* (sporothrichosis). Of these, cryptococcus is most commonly found on the ear pinna.

Cryptococcus Neoformans

Aetiology and Pathogenesis

C. neoformans is a saprophytic budding yeast. It is mostly associated with pigeon droppings and eucalyptus leaves. Cats are more commonly affected than dogs. The route of infection is thought to be inhalation, which may account for the high incidence of lesions on the nasal planum. Involvement of the rest of the skin is thought to be by haematogenous or lymphatic spread. Immunosuppressed

animals are thought to be at an increased risk of developing the disease. In cats it is usually associated with FeLV and FIV infections.

Clinical Signs

Approximately 55% of cats have upper respiratory disease with a nasal discharge, with polyploid lesion forming masses in the nostril or granulomatous lesions on the bridge of their nose. When lesions affect the pinna they form papules and nodules which can be up to 10 mm in diameter.

Key Diagnostic Tests

- Impression smears, fine needle aspirate.
- Excisional biopsy reveals a cystic degeneration or vacuolation of the dermis and subcutis or a nodular to diffuse pyogranulomatous to granulomatous dermatitis with organisms which appear as round–elliptical bodies with a thick clear capsule.
- Serology may be useful in the form of a latex cryptococcal antigen test.

Viral Infection of the Ear Pinna

This generally results in ulcerative lesions (see section 4.6). Papillomavirus, however, will cause wart-like verrucose lesions that can affect the ear pinna.

Papillomatosis

Aetiology and Pathogenesis

Most lesions are caused by a papovavirus virus that can be transmitted by direct or indirect contact. At least three different types of papovavirus have been identified in the dog, with site specificity. Canine viral papillomatosis is common and principally affects young dogs although it can occur in animals on immune-modulating therapy with drugs such as ciclosporin (Fig. 4.58). Lesions occur around the mouth and on the skin including the ear pinna.

Clinical Signs

Lesions present as well demarcated superficial flesh-coloured cauliflower-like masses (Fig. 4.59) that can range in size from 2 mm to 3 cm. They are non-pruritic.

Key Diagnostic Tests

- History and excisional biopsy.
- Biopsy most commonly reveals signs of papillated or plaque-like epidermal hyperplasia and papillomatosis.

4.5.2 Non-Infectious Nodular Diseases

Most of the nodular diseases that affect the ear pinna are non-infectious.

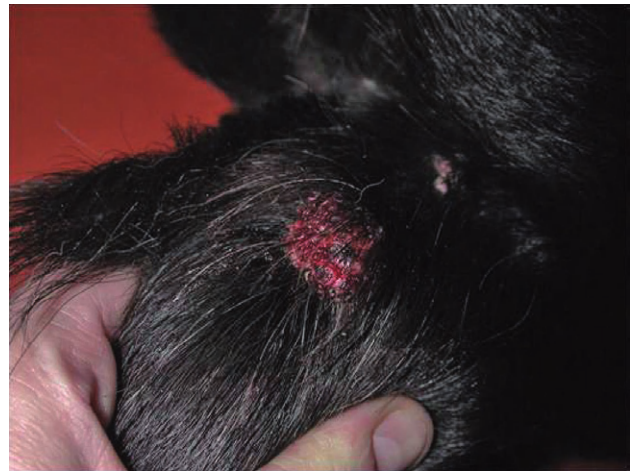


Figure 4.58 Papillomas on the ear of a dog on ciclosporin therapy.



Figure 4.59 Papillomas on the ear of a young dog.

Sterile Granuloma and Pyogranulomatous Disease

Aetiology and Pathogenesis

The pathogenesis of this disease is unknown. However, as no causal infectious organisms have been identified and the lesions respond to glucocorticoids and other immune-modulating drugs, it is suspected to be an immune-mediated process. It is an uncommon disease in dogs and a rare disease in cats.

Clinical Signs

The Great Dane, Boxer, Golden Retriever, Weimaraner, and Collie are predisposed. No feline breed is at increased risk. Lesions consist of firm painless papules and nodules that can be variably alopecic occasionally with secondary ulceration and infection (Fig. 4.60). They are most commonly



Figure 4.60 Nodular lesions on the ear pinnae of a dog with sterile granulomatous/pyogranulomatous disease.

seen on the bridge of the nose and muzzle but can also be seen on the ear pinna.

Key Diagnostic Tests

- Fine needle aspirates reveal a pyogranulomatous to granulomatous inflammation without microorganisms.
- Excisional biopsy of a lesion reveals nodular to diffuse pyogranulomatous to granulomatous dermatitis.
- Early lesions show a vertical orientation of oblong perifollicular granulomas or pyogranulomas that track the hair follicle.
- Tissue culture for aerobic and anaerobic bacteria, mycobacteria, and fungi is negative, as are histopathological special stains.

Histiocytic Disease

Several different cutaneous histiocytic diseases have been identified in the dog. These are reactive (cutaneous and systemic histiocytosis) or neoplastic (cutaneous histiocytoma [see below] and histiocytic sarcoma). In addition disseminated histiocytic sarcoma is recognized especially in the Bernese Mountain Dog, but this rarely affects the skin.

Cutaneous and Systemic Histiocytosis

Aetiology and Pathogenesis

The precise aetiology is unknown but is thought to be due to a dysregulation of the immune system.

Clinical Signs

The Bernese Mountain Dog, Golden and Labrador Retriever, Boxer, and Rottweiler are predisposed to systemic histiocytosis (SH). Cutaneous lesions in both diseases are similar, but SH also has multiple organ involvement. Typical lesions are erythematous plaques and nodules often with associated alopecia. They are usually non-pruritic and non-painful.

Key Diagnostic Tests

- Fine needle aspirates shows signs of cytological histiocytes.
- Excisional biopsy of a nodule reveals superficial and deep perivascular nodular or diffuse dermal and subcutaneous infiltrates of cytologically normal histiocytes.

Neoplasia

Aetiology and Pathogenesis

Most common tumours of the ear pinna in the cat are listed in Table 4.1. Most tumours are locally invasive with a low metastatic potential. Squamous cell carcinomas are the most common pinnal tumours in the cat.

The most common tumours of the ear pinna of dogs are listed in Table 4.2. As in the cat, most tumours are locally invasive with low metastatic potential. All appear with about equal frequency.

Clinical Signs

Variable clinical signs dependent on type of tumour.

Key Diagnostic Tests

- Fine needle aspirate, and excisional biopsy, except in canine hstiocytoma which is a self-resolving lesion.

4.6 ULCERATIVE DISEASES

4.6.1 Autoimmune Disease

Many of the autoimmune diseases will present with ulceration of the ear pinna.

Cutaneous Lupus Erythematosus (Discoid Lupus Erythematosus)

Aetiology and Pathogenesis

Also known as discoid lupus erythematosus (DLE), this is thought to be an actinically induced immune-mediated disease that induces damage at the level of the dermoepidemal junction. There may be a genetic predisposition to develop disease.

Clinical Signs

Predisposed breeds include Shetland sheepdog, Rough Collie, German Shepherd dog, and Siberian Husky (Figs

VetBooks.ir

Table 4.1 Most common tumours of the ear pinna in the cat

Tumour	Comments
Squamous cell carcinoma	Most common tumour, usually sunlight induced, found in elderly white cat (>9 years of age). Two forms exist: proliferative ulcerated vegetative form and erosive ulcerated form. Although this is a malignant and invasive tumour it is slow to metastasize (Fig. 4.61)
Fibrosarcoma	Common cutaneous tumour in the cat, it is irregular and nodular, poorly circumscribed, 1–15 cm in diameter. Often alopecic and ulcerated. In young cats (<5 years of age) it is a virally induced tumour caused by FeSV. In older cats it is not associated with FeSV. Locally invasive tumour with low metastatic potential (Fig. 4.62)
Haemangioma/ sarcoma	Tumours may be sunlight induced, often found in white cats. Haemangiomas are small (0.5–4.0 cm) usually solitary, benign, slow growing alopecic raised blue-purple dermal to subcutaneous masses. Haemangiosarcomas are fast-growing, poorly circumscribed, invasive, large tumours with a high metastatic rate. Commonly seen in male cats >10 years of age, tumours are usually red–dark blue plaques and nodules up to 2 cm in diameter (Fig. 4.63)
Melanoma	Uncommon pinnal tumours found in cats with average age of 10 years. Most appear to be benign and are usually solitary; 1–3 cm in size; polyploidy or plaque-like; grey, brown, or black
Basal cell tumour/ epithelioma/ carcinoma	Well-circumscribed, indurated, white to pigmented, plaques or umbilicated nodules with variable alopecia and ulceration. Usually found in adult cats. Tumours tend to be slow growing and benign. Usually small (0.5–3.0 cm in diameter). Himalayans, Siamese, and Persian cats may be predisposed
Mast cell tumour	Common pinnal tumour seen in older cats (mean 10 years). Siamese cats and male cats are predisposed. Tumours have a very variable appearance and size, 0.5–5 cm in diameter. They may be papular or nodular or plaques; pink, yellow, or white. They are usually well circumscribed and attached to underlying skin. Tumours are usually benign (Fig. 4.64)

FeSV, feline sarcoma virus.



Figure 4.61 Squamous cell carcinoma on the ear pinna of a cat.



Figure 4.62 Fibrosarcoma on the face of a cat.



Figure 4.63 Haemangiosarcoma on the ear of a cat.



Figure 4.64 Multiple mast cell tumours on the head of a cat.

Table 4.2 Most common tumours of the ear pinna in the dog

Tumour	Comments
Squamous cell carcinoma	Sunlight-induced tumour commonly found on the ears; predisposed breeds include Dalmatian, Bull Terrier, and Boxer. Two forms exist: a proliferative, ulcerated, vegetative form and an erosive, ulcerated form, both of which are of variable size. Tumours are locally invasive and slow to metastasize
Haemangiosarcoma	Most commonly seen in dogs >10 years, especially German Shepherd, Golden Retriever, Bernese Mountain Dog, and Boxer. White-haired breeds are at increased risk of sunlight-induced disease. Tumours are poorly circumscribed red–dark blue plaques or nodules <2 cm in diameter. Locally invasive with high metastatic potential
Papilloma	See viral disease, section 4.5.1
Mast cell tumour	Boxer, Boston Terrier, Golden and Labrador Retrievers, Bulldogs, Staffordshire Bull Terriers, and Shar Peis predisposed. Average age 8 years. Variable size (few mm–several cm in diameter)and appearance. May be single or multiple dermal or subcutaneous papules to pedunculated lesions, except in the Shar Pei where lesions are diffuse. Can be erythematous to hyperpigmented. Malignancy and metastatic potential depends on the grade of the tumour
Cutaneous histiocytoma	Young dogs <3 years. Usually solitary elevated domed none pruritic painless nodules up to 2 cm in diameter. They are usually rapidly growing. Often ulcerate before spontaneous regression. Predisposed breeds include Boxer, Bulldog, and Scottish Terrier
Sebaceous gland tumour (nodular hyperplasia, epithelioma, SA, SAC)	All lesions seen in elderly dogs 9–10 years. NSH most commonly seen in Beagles, Cocker Spaniels (Fig. 4.65), and Poodles. Variable sized (3 mm–5 cm) solitary, well-circumscribed, raised smooth, greasy, wart-like or cauliflower like in appearance. SE most commonly seen in Shih tzu, Lhasa Apso, and Irish Setters, similar in appearance to NSH but can be ulcerated and melanotic (Fig. 4.66). SA and SAC are similar in appearance to the other sebaceous tumours. Only SAC is invasive and metastatic (Fig. 4.67)
Plasmacytoma	Common tumour of the ear pinna and top of the vertical ear canal. Average age of onset 10 years. Cocker Spaniels appear to be predisposed. Lesions are well circumscribed (Fig. 4.68), raised firm to soft, pink to red, dermal in location, usually 1–2 cm in diameter

NSH, nodular sebaceous hyperplasia; SA, sebaceous gland adenoma; SAC, sebaceous gland adenocarcinoma; SE, sebaceous epithelioma.



Figure 4.65 Nodular sebaceous hyperplasia on the ear pinnae of a Cocker Spaniel.



Figure 4.66 Extensive nodular sebaceous hyperplasia on the ear of a Yorkshire Terrier.



Figure 4.67 Adenocarcinoma on the ear flap of an elderly West Highland White Terrier.



Figure 4.68 Plasmacytoma on the ear flap of a dog.

4.69, 4.70). DLE is a rare disease in cats (Fig. 4.71). The most common site for lesions is the planum nasale but the ear pinna can be involved especially in the cat. Acutely lesions are alopecic and erythematous but chronically ulceration with crusting is seen.

Key Diagnostic Tests

- History.
- Cytology of lesions is of limited value and non-diagnostic.
- Biopsy reveals an interface dermatitis (hydropic, lichenoid or both), focal hydropic degeneration of basal epidermal cells, pigment incontinence, and apoptotic keratinocytes.

Bullous Pemphigoid

Aetiology and Pathogenesis

Rare vesiculobullous disease associated with autoantibody attack of the hemidesmosomes and possible basement membrane zone. Triggers for the disease are unclear but susceptibility to environmental factors such as ultraviolet light and drug reactions may be important.

Clinical Signs

Dogs and cats can both be affected and are usually systemically unwell, anorectic, depressed, and febrile. Most commonly affects Collie breeds. Onset of lesions is typically acute, producing erosions and ulceration. Affected areas include mucocutaneous junctions (Fig. 4.72), axial regions, and groin. The ear pinna can be affected, presenting with severe ulceration and exudation (Fig. 4.73). Bullous



Figure 4.69 Discoid lupus erythematosus in a Husky showing crusts and scale on the periphery of the ear pinnae.



Figure 4.70 Chronic ulcerative lesions due to discoid lupus erythematosus on the ear of a German Shepherd dog.

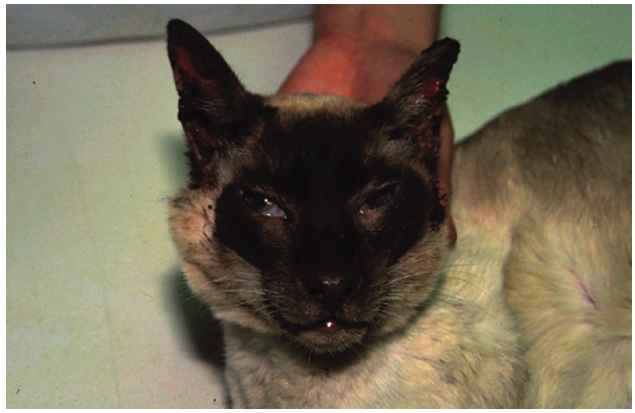


Figure 4.71 Discoid lupus erythematosus in a cat showing ulceration of ear pinnae.

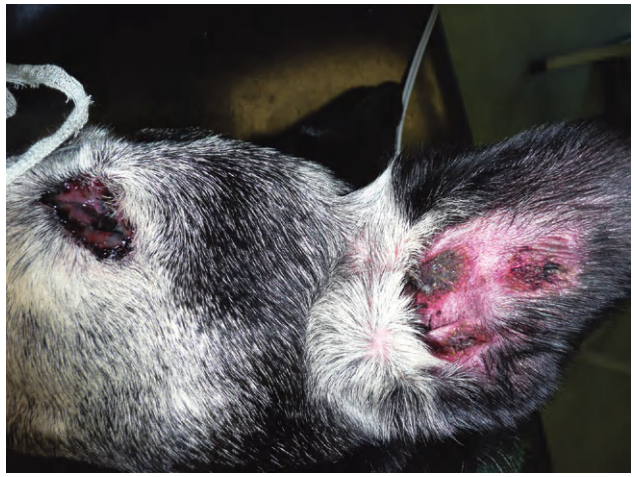


Figure 4.72 Ulceration around the eyes and on the ear pinnae in a Collie with bullous pemphigoid.



Figure 4.73 Ulceration of the ear pinna in a dog with bullous pemphigoid.

pemphigoid will more commonly present with intact vesicles that other autoimmune diseases.

Key Diagnostic Tests

- Cytology of lesions is of limited value and non-diagnostic.
- Biopsy reveals subepidermal clefts and vesicle formation. Acantholysis is not seen. Inflammatory infiltrates vary from mild and perivascular to marked and lichenoid.

4.6.2 Environmental Disease

Cold-induced damage usually causes ulceration of the ear pinna.

Frostbite

Aetiology and Pathogenesis

Tissue damage occurs due to prolonged exposure to freezing weather, especially in wet and windy conditions. Damage occurs due to direct cold injury to the cells and also due to circulatory problems leading to tissue hypoxia.

Clinical Signs

Lesions occur on sparsely haired areas especially tips of tail and the ears. Footpads can also be affected. Acutely the skin is erythematous, painful, and oedematous (Fig. 4.74). After 7–14 days a line of demarcation may become evident between normal and damaged tissue; the latter area may then slough.

Key Diagnostic Tests

- History and clinical signs.

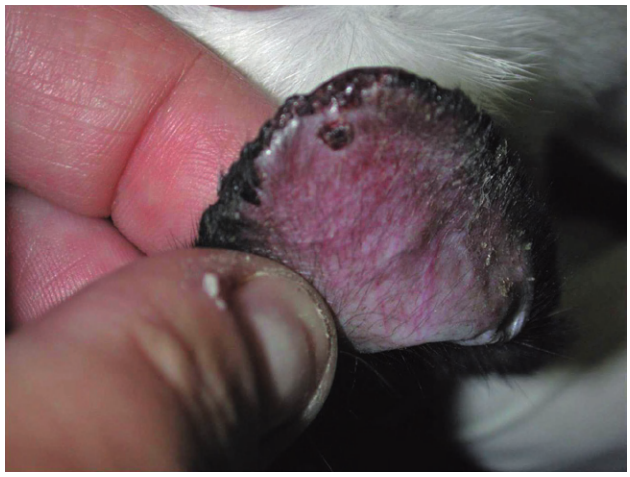


Figure 4.74 Frostbite lesion on the ear tip of a dog.

4.6.3 Immune-Mediated Disease

Idiopathic Ear Margin Vasculitis

Aetiology and Pathogenesis

Thought to be a type III immune complex mediated disease of unknown aetiology, but can be associated with drug administration.

Clinical Signs

Dachshunds seem to be predisposed. Lesions present as punched-out ulcerative lesions that remain confined to the margins of the ear pinna (Fig. 4.75). The ulcers appear to be non-pruritic and painless. The disease is usually bilateral (Fig. 4.76) and each ear may have numerous lesions which will expand if left untreated along the concave surface of the ear pinna.



Figure 4.75 Punched-out ulcers on the ear tip of a dog.



Figure 4.76 Bilateral ulceration of the periphery of the ear pinna in a case of vasculitis.

Key Diagnostic Tests

- History.
- Biopsy of lesion which demonstrates varying degrees of a leukocytoclastic vasculitis.

Cold Agglutinin Disease

Aetiology and Pathogenesis

Cold agglutinin disease (CAD) is an autoimmune disorder associated with cold-reacting erythrocyte autoantibodies. Antibodies lead to autoagglutination of erythrocytes in the extremities, especially the ear pinna, once the temperature drops below a critical level. Microthromboses form in the small end arterioles and capillaries within the ear pinna, which leads to varying degrees of ischaemic necrosis.

Clinical Signs

Early lesions consist of oedema and erythema. Later purpura and acrocyanosis develop, followed by crusting, ulceration, and necrosis which can then lead to loss of the ear tip (Fig. 4.77). Other affected sites include the tail and footpads.

Key Diagnostic Tests

- History, physical examination.
- Demonstration of significant titres of cold agglutinins. Autoagglutination occurs at 0–4 °C and is reversed at 37 °C.



Figure 4.77 Loss of ear tip in cold agglutinin disease.

Drug Eruption

Aetiology and Pathogenesis

Skin disease is caused by exposure to a particular drug which may be administered systemically or topically. Many different immune-mediated patterns can be create by drugs including oedema, pemphigus, pemphigoid, and vasculitis. Common groups of drugs that induce reactions include antibiotics, non-steroidal anti-inflammatories, vaccines, and anthelmintics.

Clinical Signs

Lesions commonly affect the ears when a vascular insult occurs. Vasculitis produces severe punched-out, or in severe cases confluent, ulceration of the periphery of the ear pinna (Fig. 4.78).

Key Diagnostic Tests

- History.
- Biopsy is rarely diagnostic only supportive.
- Provocative challenge should be avoided.

Canine Familial Dermatomyositis

Aetiology and Pathogenesis

Thought to be an immune-mediated disease that leads to vascular damage and as a result damage to the skin and muscle. The condition is recognized as being familial in the Shetland sheepdog and Rough Collie but other breeds can be affected.

Clinical Signs

Affects dogs more than 6 months of age. Lesions affect the bridge of the nose, periocular skin, and ear tips (Fig. 4.79). In some cases the tail tip and nails may also be involved.



Figure 4.78 Vasculitis on the ear tip of a cat due to a drug eruption.



Figure 4.79 Dermatomyositis in a dog.

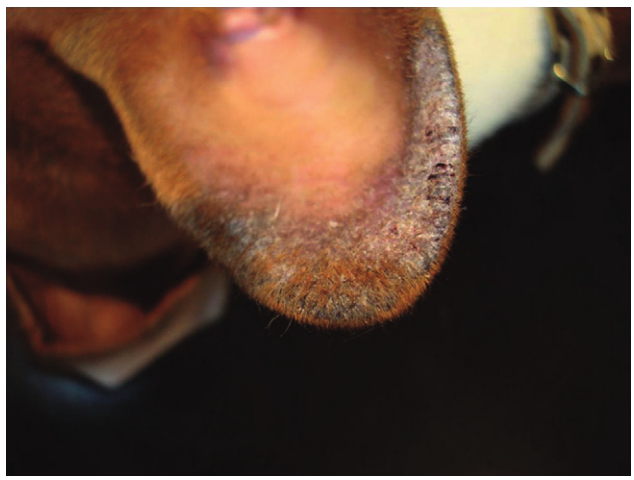


Figure 4.80 Scale and erythema on the ear tip of a dog with dermatomyositis.

Severely affected dogs have masticatory muscle damage and have problems eating. Early skin lesions are alopecic with erythema and some scale (Fig. 4.80). Chronic ear lesions can produce deformation of the ear pinna occasionally with ulceration and necrosis (Fig. 4.81).

Key Diagnostic Tests

- History, predisposed breed.
- Biopsy of lesions reveals scattered vacuolar changes of surface and follicular basal cells. Occasional apoptotic cells are seen. Follicular atrophy and fibrosis is common. Vasculitis can be seen in the skin.
- EMG abnormalities can be demonstrated in some dogs.

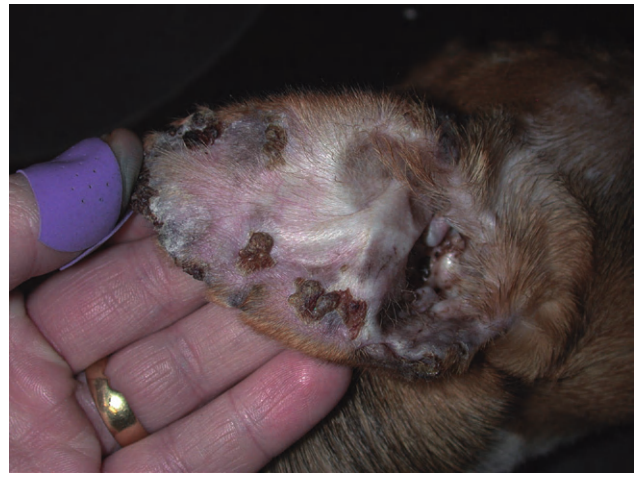


Figure 4.81 Ulceration and necrosis of ear pinna in a case of dermatomyositis.

Erythema Multiforme Complex

Aetiology and Pathogenesis

The erythema multiforme complex (EM) is a very rare group of immune-mediated skin diseases that has been divided into a range of progressively more severe forms: EM minor, EM major, Stephen–Johnson syndrome (SJS), SJS–toxic epidermal necrolysis overlap syndrome, toxic epidermal necrolysis (TEN). EM minor and EM major are commonly associated with infection, especially postviral infection. The other forms are usually drug induced.

Clinical Signs

The more severe forms of the group have the most rapid onset of signs, and lesions become progressively more widespread with the more aggressive disease variants. The ear pinna is commonly affected in all forms of the disease complex. EM lesions are generally erythematous macules and papules (Fig. 4.82) often presenting as 'target lesions'. Blistering, ulceration, and necrosis of the skin is seen in SJS/TEN (Fig. 4.83). As the disease becomes more severe and widespread, so the animal's general health deteriorates.

Key Diagnostic Tests

- History and clinical signs.
- Biopsy reveals signs of a hydropic interface dermatitis with single-cell necrosis of keratonocytes and satellitosis of lymphocytes and macrophages. However, the

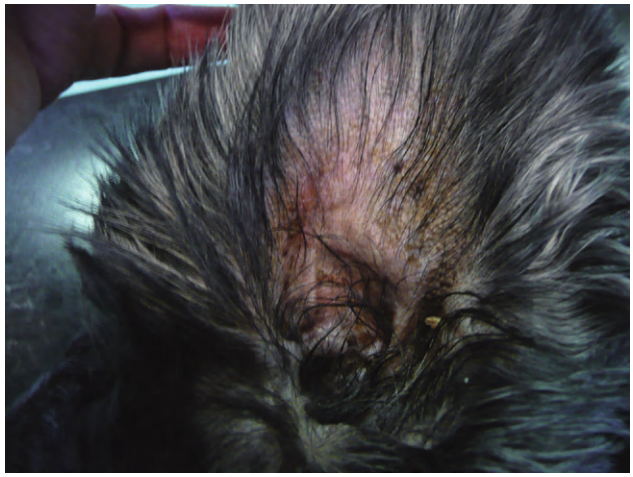


Figure 4.82 Early signs of erythema multiforme in a dog.



Figure 4.83 Severe signs of erythema multiforme due to a hepatic tumour.

distribution and severity of the clinical signs contributes to the diagnosis as well as the histopathology.

Puppy Pyoderma
See section 4.4.2.

4.6.4 Infections

Staphycoccal Infection
See section 4.5.1.



Figure 4.84 Lesions of cat pox on the face of a cat.

Fungal Infection
See section 4.5.1.

Viral Infection—Feline Pox Virus (Cowpox)

Aetiology and Pathogenesis

Caused by an orthopoxvirus thought to be transmitted by the bites of small rodents. The disease appears to be most common in the autumn.

Clinical Signs

The primary lesion usually occurs at the site of the bite and present as transient erythematous papules or erosions that resolve. These may be on the face (Fig. 4.84), ear, or leg. After 10–14 days the cat will usually develop more generalized large crusted papules.

Key Diagnostic Tests

- Virus can be isolated from scabs or via viral transport medium.
- Biopsy reveals hyperplastic balloon degeneration, reticular degeneration, microvesicle formation, and necrosis of the affected epidermis and outer root sheath of the hair follicle. Eosinophilic intracytoplasmic viral inclusions are found in keratinocytes.

4.6.5 Neoplasia

Epitheliotropic Lymphoma
See section 4.2.6 (Fig. 4.85).

Squamous Cell Carcinoma
See section 4.5.2.

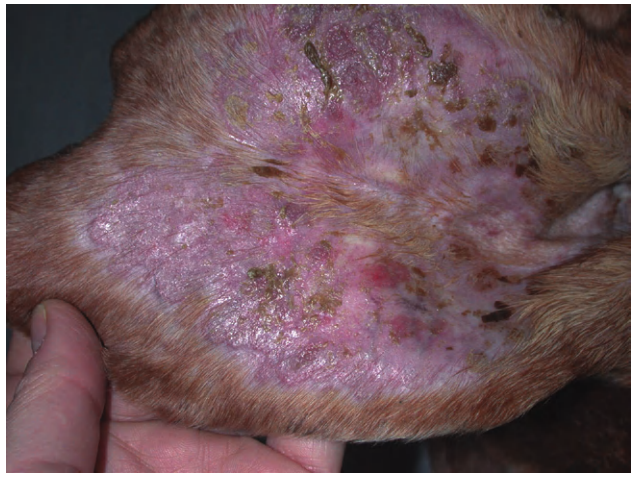


Figure 4.85 Ulceration of the ears of a dog with epitheliotropic lymphoma.



Figure 4.86 Pattern baldness on the ear of a young Dachshund.

4.7 DISEASES WITH ALOPECIA

4.7.1 Alopecia Without Primary Lesions

Hypothyroidism

See section 4.2.2.

Pattern Baldness

Aetiology and Pathogenesis

Uncommon disease of unknown aetiology. A genetic predisposition is thought to exist.

Clinical Signs

Predisposed breeds include the Dachshund, Boston Terrier, Chihuahua, Greyhound, Whippet, and miniature Pinscher. Hair loss is non-pruritic but insidious and slowly progressive (Fig. 4.86). In the Dachshund clinical signs typically start at 6–12 months of age. Hair coat on the ear pinna initially thins due to hairs becoming short and fine before being lost completely (Fig. 4.87). Other areas of the body are normally spared. Alopecic areas become hyperpigmented.

Key Diagnostic Tests

- History, predisposed breeds.
- Biopsy reveals miniaturisation of the hair follicles with normal adnexal structures. Follicles are shorter and thinner with small hair bulbs and fine hair shafts.



Figure 4.87 Pattern baldness on the ear of a Dachshund.

Follicular Dysplasia

Aetiology and Pathogenesis

Follicular dysplasia occurs as a result of abnormal formation of the hair shaft. This can be associated with colour dilute alopecia where abnormalities in pigmentation within the follicular shaft leads to hair loss. Predisposed breeds include the Dobermann and Dashshund.

Clinical Signs

Alopecia commonly affects the dorsal and lateral trunk but will also affect the convex aspect of the ear pinnae. Dogs with light blue (grey) coats are affected at 6 months of age,



Figure 4.88 Colour dilute alopecia showing thinning of the hair on the body and the ear pinnae.

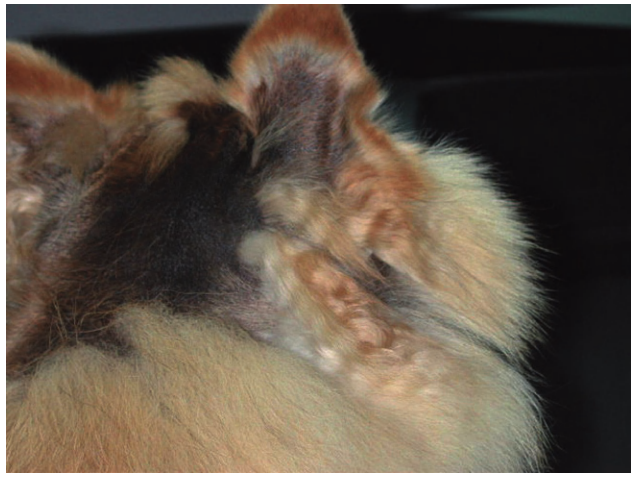


Figure 4.89 Alopecia areata on the head and ear of a Chow Chow.

less dilute colours (steel blue) may not develop signs until 2–3 years of age (Fig. 4.88).

Key Diagnostic Tests

- Hair plucks will reveal numerous macromelanosomes of irregular size and shape unevenly distributed along the hair shaft.
- Histopathology shows signs of melanin clumping in epidermal and follicular basal cells and hair maitrix cells with numerous peribulbar melanophages.

Alopecia Areata

Aetiology and Pathogenesis

Alopecia areata is caused by an immune-mediated attack against the anagen hair follicle. It is most commonly seen in dogs.

Clinical Signs

Alopecia may be focal or multifocal and is none inflammatory. Alopecic areas are well circumscribed and exposed skin appears normal. It commonly affects the ear pinna but signs may also be found on the head, neck, and trunk (Fig. 4.89).

Key Diagnostic Tests

- Hair plucks show signs of typical 'exclamation point' hairs, i.e. short stubby hairs with frayed, fractured distal ends.
- Biopsy reveals a peribulbar infiltrate of lymphocytes, histiocytes, and plasma cells often referred to as a 'swarm of bees'. Chronically, follicular atrophy occurs.



Figure 4.90 Early signs of melanoderma in a Yorkshire Terrier.

Melanoderma and Alopecia in Yorkshire Terriers

Aetiology and Pathogenesis

A common hereditary disorder of unknown aetiology.

Clinical Signs

Animals exhibit signs of alopecia and hyperpigmentation of the ear pinna, bridge of nose, tail, and feet at 6 months to 3 years of age. Affected skin is smooth and shiny, not pruritic or painful (Fig. 4.90).

Key Diagnostic Tests

- Clinical signs.
- Biopsy reveals orthokeratotic hyperkeratosis of epidermal surface and of hair follicle with epidermal melanosis.

Congenital Alopecia

Aetiology and Pathogenesis

Congenital alopecia is seen in alopecic breeds which include Mexican hairless dogs, Chinese crested dogs, and Sphinx cats and their crosses. Alopecia is present at birth.

Clinical Signs

Generalized truncal alopecia is present at birth; in some cases the animals may retain some hair on the ear pinnae (Figs 4.91, 4.92).

Key Diagnostic Tests

- Clinical signs in an affected breed.
- Biopsies reveal atrophic, decreased numbers or complete absence of hair follicles. Follicular dilatation and hyperkeratosis are common.



Figure 4.91 Congenital hypotrichosis affecting the ear in a cat.



Figure 4.92 Congenital whole-body hypotrichosis.

4.7.2 Alopecia with Comedones

Comedones can be associated with a range of different diseases, the most important of which are demodicosis and hyperadrenocorticism.

Demodicosis

Aetiology and Pathogenesis

Follicular mites cause alopecia and comedone formation on the ear pinna of both the dog and the cat. In the dog this is *Demodex canis* and in the cat *Demodex cati*. Demodex is an asymptomatic skin commensal in healthy immunocompetant dogs and cats. Its life cycle is 20–35 days. In young dogs less than 3 years (juvenile onset demodicosis), disease may be associated with intrinsic hereditary factors that allow multiplication of the mites. In dogs more than 3 years old immunosuppresive factors such as endocrine disease or neoplasia may trigger disease (adult-onset demodicosis).In cats *D. cati* is usually associated with immunosuppression with systemic disease such as diabetes mellitus, natural or iatrogenic hyperadrenocorticism, FeLV or FIV infection. Demodicosis can be localized or generalized and squamous (Fig. 4.93) or pustular in nature. All of the forms can affect the ear pinna, but the non-follicular forms of the mites *D. gatoi* and *D. corniei* usually presents with erythema and scale, not comedones.

Clinical Signs

Ear pinna lesions present as alopecia, with scale (Fig. 4.94). The skin can have a blue-grey appearance due to confluent comedone formation. Secondary infection with malassezia or bacteria additionally causes papular/pustular lesions (Fig. 4.95). Other commonly affected areas include face, neck, head, forelimbs, and trunk (Fig. 4.96).



Figure 4.93 Squamous demodicosis in a dog.



Figure 4.94 Early lesions of demodicosis with comedone formation.



Figure 4.96 Demodicosis in a young dog affecting the face.



Figure 4.95 Pustular demodicosis affecting a dog's face.



Figure 4.97 Naturally occurring Cushing's disease showing comedones and alopecia of the ear pinnae.

Key Diagnostic Tests

- Deep skin scrapes and hair plucking.
- Squeezing the skin to try to express follicular contents may be useful.
- In thickened skin and in the Shar Pei biopsy may be necessary.

Hyperadrenocorticism

Aetiology and Pathogenesis

Naturally occurring cases (80–85%) of canine hyperadrenocorticism (HAC) are caused by a functional pituitary adenoma leading to excessive production of adrenocorticotropic hormone (ACTH) and bilateral adrenal hyperpla-

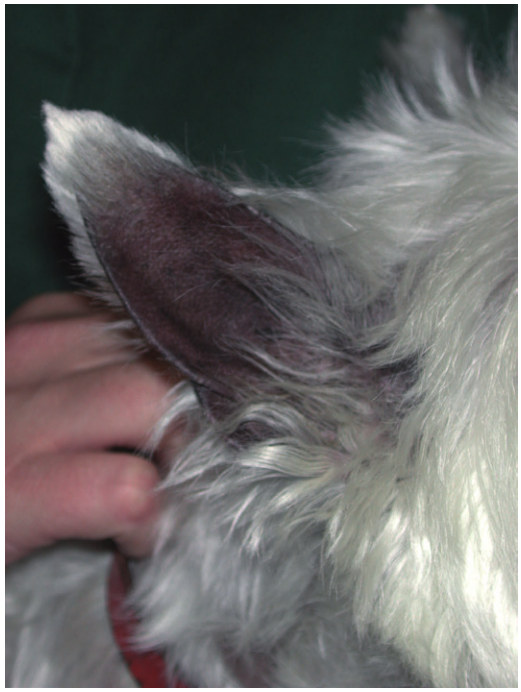


Figure 4.98 Iatrogenic Cushing's disease caused by excessive topical glucocorticoid therapy.

sia. Approximately 15–20% of cases are caused by an adrenal tumour. HAC is rare in the cat. Clinical signs are associated with excessive cortisol production.

Iatrogenic HAC affecting the ear pinna is caused by excessive topical or systemic glucocorticoids. Topical glucocorticoids may be steroid sprays, creams or gels applied to the ear pinna or potent otic steroid drops that are carried out of the ear by epithelial migration and deposited on the ear pinna. Systemic steroids create iatrogenic HAC when they are given for prolonged periods without appropriate measures to spare the adrenal/pituitary axis, i.e. daily dosage or continued use of long-acting injectable products.



Figure 4.99 Alopecia and curling of the ear tip in a dog with iatrogenic Cushing's disease.

Clinical Signs

Naturally occurring HAC: terriers, Dachshunds (adrenal tumours), Boxers (pituitary tumours) are predisposed. Non-cutaneous signs are protean and include polyuria, polydipsia, polyphagia. Cutaneous signs include symmetrical to generalized non-inflammatory alopecia, cutaneous atrophy and comedones. The ear pinna typically reveals atrophic, alopecia skin with comedone formation (Fig. 4.97). Where secondary infection is present there will be some peripheral crusting.

Iatrogenic HAC produces similar signs to the natural disease (Figs 4.98, 4.99). However in cats the ear pinnae will show typical signs of atrophy with inward curling of the tips of the ear pinna.

Key Diagnostic Tests

- Routine haematology and biochemistry, dynamic function tests (ACTH stimulation test, low-dose dexamethasone suppression tests).
- Biopsy rarely indicated.

VetBooks.ir

VetBooks.ir

CHAPTER 5
CONDITIONS OF THE EAR CANAL

Sue Paterson

5.1	Allergy	87	5.7	Foreign Bodies	91
5.2	Endocrine Disease	87	5.8	Neoplastic and Hyperplastic Conditions of Ear Canal	93
5.3	Ectoparasites	88			
5.4	Keratinization Disorders	90	5.9	Predisposing Factors in Otitis Externa	97
5.5	Autoimmune Disease	90	5.10	Perpetuating Factors	100
5.6	Idiopathic Disease	90			

The ear canal is lined by skin and as such any skin problem can also affect the ears. A range of primary triggers have been identified as causing otitis externa. These are listed in Table 5.1.

Table 5.1 Primary triggers for otitis externa

Primary trigger	Comments
Allergy	Atopy, food allergy, contact hypersensitivity
Endocrine disease	Hypothyroidism, hyperadrenocorticism
Ectoparasites	*Otodectes cynotis*, *Demodex* (*canis*, *cati*)
Keratinization disorders	Sebaceous adenitis, primary idiopathic seborrhoea
Autoimmune skin disease	Pemphigus foliaceus, discoid lupus erythematosus
Idiopathic	Juvenile cellulitis, proliferative and necrotizing otitis (cat)
Foreign bodies	Grass awns, medication, ceruminoliths
Neoplastic disease	(see Table 5.2)
Hyperplasic disease	Polyps, glandular hyperplasia

5.1 ALLERGY (SEE ALSO CHAPTER 4, SECTION 4.1.1)

5.1.1 Atopy, Food Allergy, Contact Allergy/Irritancy

Clinical Signs

In acute allergy the canal appears erythematous and hyperplastic (Fig. 5.1) with minimal exudation. The pars flaccida may appear swollen and oedematous (Fig. 5.2). In more chronic disease the canal can become narrowed (Fig. 5.3) with increased erosion and ulceration, exudation and secondary infection (see later, section 5.10). Food allergy and atopy cannot be distinguished purely on the grounds of the appearance of the canals. Disease can be unilateral. In cases of contact irritancy/allergy where topical medication is not discontinued the canal can become severely ulcerated.

5.2 ENDOCRINE DISEASE

5.2.1 Hypothyroidism (See Also Chapter 4, Section 4.2.2)

Clinical Signs

Changes in the canal are none specific with mild hyperplasia and a thick ceruminous discharge (Fig. 5.4). Chronic cases develop bacterial and/or yeast infection and on occasion aural demodicosis.

5.2.2 Hyperadrenocorticism (See Also Chapter 4, Section 4.2.2)

Clinical Signs

Glandular secretions are often reduced in uncomplicated cases, but the immunosuppressive effects of the disease can

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

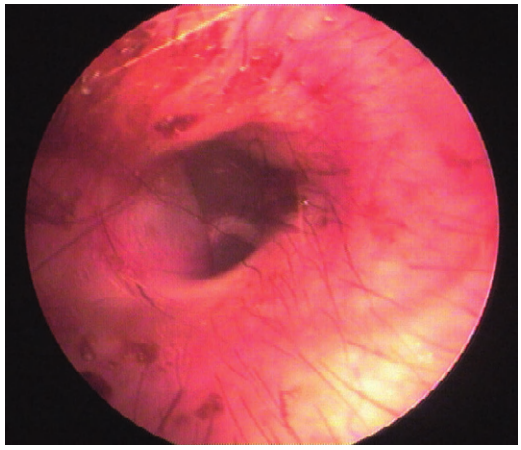


Figure 5.1 Erythema of the ear canal in early allergy.

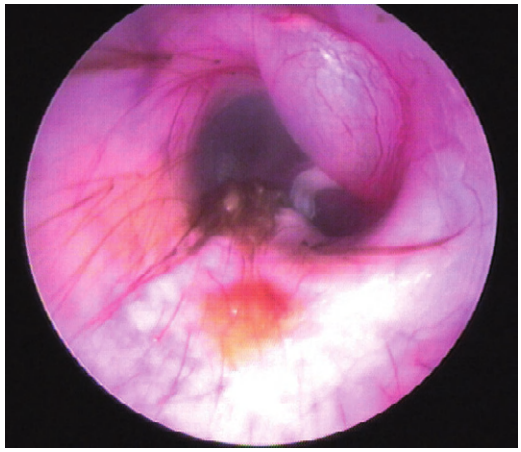


Figure 5.2 Oedema of the pars flaccida in an allergic ear.

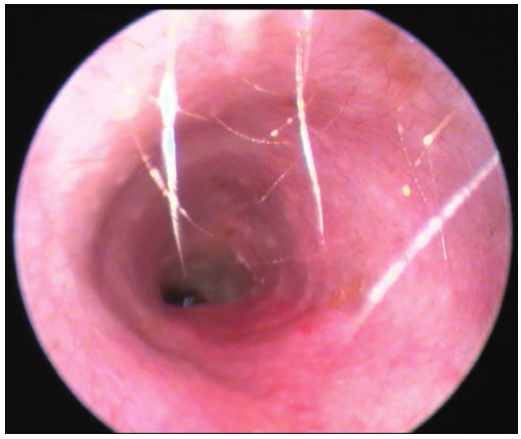


Figure 5.3 Narrowed hyperplastic allergic ear canal.

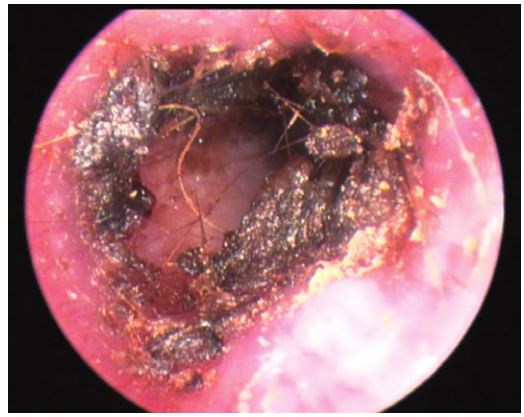


Figure 5.4 Ceruminous discharge in the ear canal of a hyporthyroid dog.

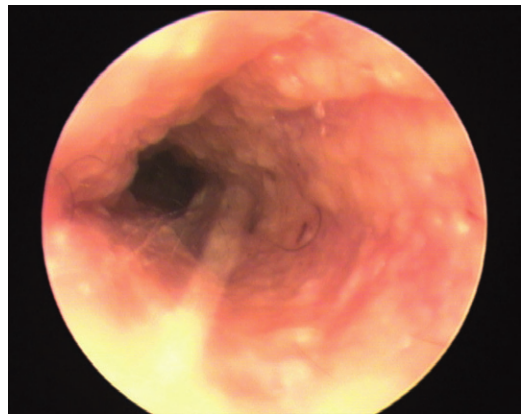


Figure 5.5 Pseudomonas infection in a dog with hyperadrenocorticism.

predispose to aural demodicosis or infection with bacterial or yeast (Fig. 5.5).

5.3 ECTOPARASITES

5.3.1 Otodectes Cynotis

Aetiology and Pathogenesis

Otodectes cynotis is a relatively large mite (0.3–0.4 mm) that lives predominantly in the ear canal of the dog and cat although it can be found occasionally on other areas of the body, especially around the tail base on cats where they sleep curled up. It is a non-burrowing mite and lives by browsing on surface on skin debris and tissue fluid. It is thought to be able to live off the host for several weeks. Mites have zoonotic potential and have been recorded to cause a popular eruption in contact people.

Clinical Signs

Young dogs appear to be predisposed. Otodectes infestation leads to the production of copious amounts of dry,

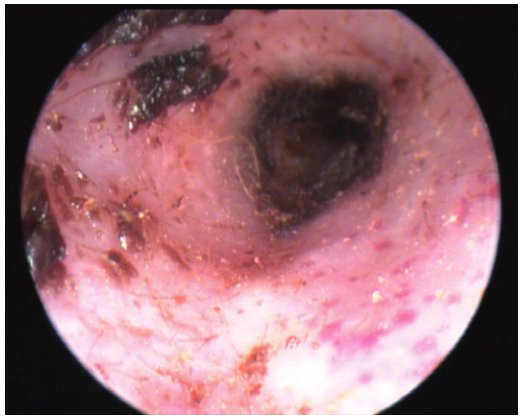


Figure 5.6 Otodectes mites in an ear canal.

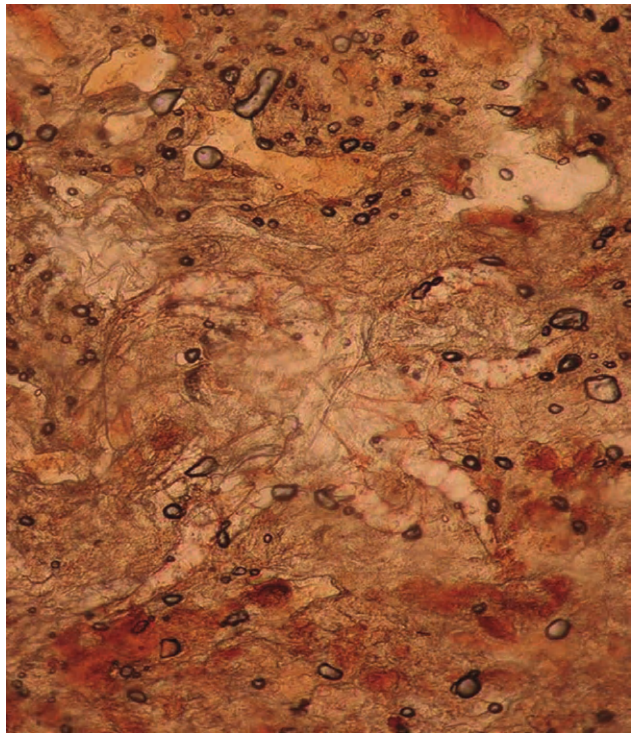


Figure 5.7 *Otodectes cynotis* in ear wax.

dark brown, crumbly waxy debris. Some authors describe it as having a 'coffee grounds' appearance. Mites can be seen down an otoscope with the naked eye as small, white, moving dots (Fig. 5.6). The degree of erythema and pruritus within the canal is variable, as some animals are thought to mount an allergic reaction to the mites leading to a disproportionate amount of inflammation for the number of mites that are present.

Key Diagnostic Tests

- Clinical signs.
- Microscopic examination of ear wax (which can be mounted in 10% potassium hydroxide) reveals typical oval mites with short pedicles and suckers on the front four legs (Fig. 5.7).

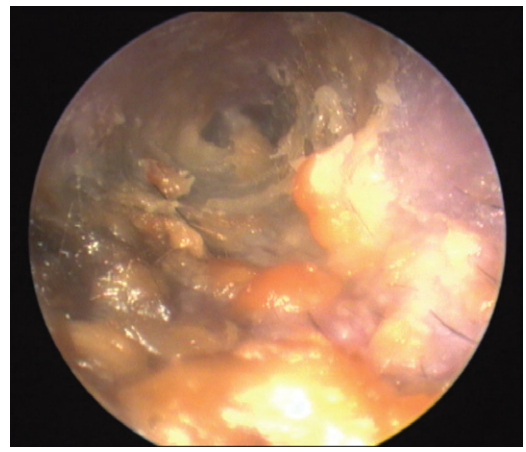


Figure 5.8 Hyperplastic waxy canal in a dog with demodicosis.

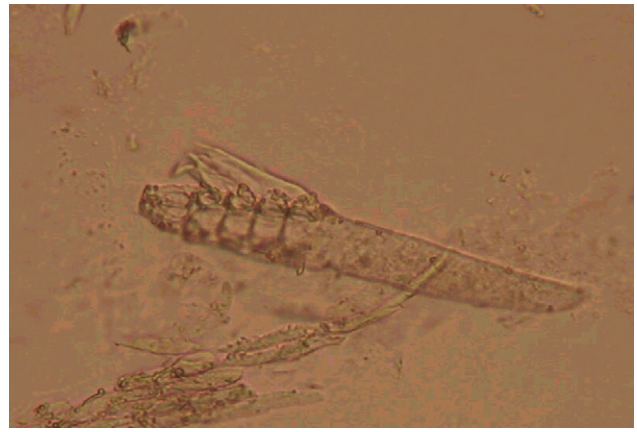


Figure 5.9 Demodex mites in ear wax.

5.3.2 Demodex Mites

Aetiology and Pathogenesis

See Chapter 4, section 4.7.2.

Clinical Signs

Demodex canis, the follicular demodex mite, is reported as a rare cause of otitis externa in the dog. Clinical signs within the canal are none specific which is usually hyperplastic and waxy (Fig. 5.8). In cats *D. gatoi* is more commonly associated with a ceruminous otitis externa. Demodectic mites can be recovered from the ear canal either by examination of wax or by skin scrapings taken from the wall of the canal (Figs 5.9, 5.10).

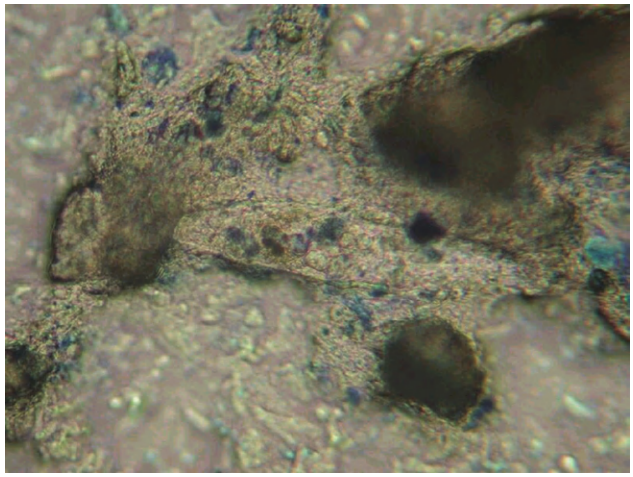


Figure 5.10 Demodex partially obscured by debris on a tape stripping.

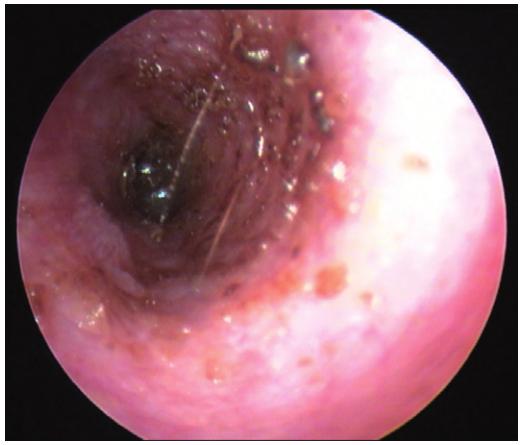


Figure 5.11 Ear canal of dog with sebaceous adenitis.

5.4 KERATINIZATION DISORDERS

5.4.1 Sebaceous Adenitis

Aetiology and Pathogenesis
See Chapter 4, section 4.2.4.

Clinical Signs
Destruction of sebaceous glands in the skin also leads to damage to glandular tissue within the ear canal. Dogs typically have an erythematous, often hyperplastic, canal containing large amounts of dry, crusty material (Fig. 5.11). Over-cleaning of these ears with acidic solutions or potent ceruminolytics can lead to severe Gram-negative infection.

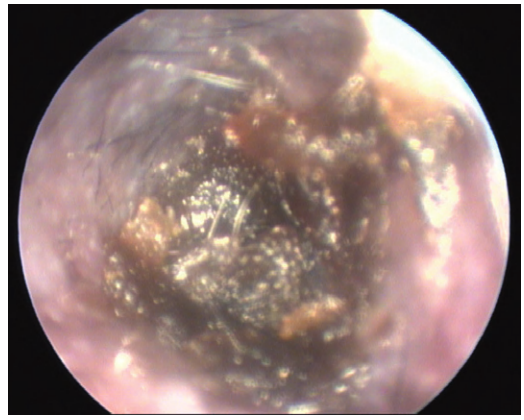


Figure 5.12 Primary idiopathic seborrhoea in the ear canal of a dog.

5.4.2 Primary Idiopathic Seborrhoea

Aetiology and Pathogenesis
See Chapter 4, section 4.2.4.

Clinical Signs
Can be seen in many different breeds, but the Cocker Spaniel appears to be over-represented. Increased wax production in the ear leads to a thick, malodorous, ceruminous discharge (Fig. 5.12) often with secondary yeast infection. In chronic cases the ear canal can develop signs of glandular hyperplasia.

5.5 AUTOIMMUNE DISEASE

5.5.1 Pemphigus Foliaceus

Aetiology and Pathogenesis
See Chapter 4, section 4.4.2.

Clinical Signs
Can affect any breed but the Akita is predisposed and can develop severe otitis externa due to pemphigus with minimal involvement of the rest of the skin. In the early stages the ear is painful and the canal is hyperplastic, erythematous and ulcerated, primary pustules can be seen in some cases (Fig. 5.13). In more chronic disease secondary infection can occur with Gram-negative infection.

5.6 IDIOPATHIC DISEASE

5.6.1 Juvenile Cellulitis

Aetiology and Pathogenesis
See Chapter 4, section 4.4.2.

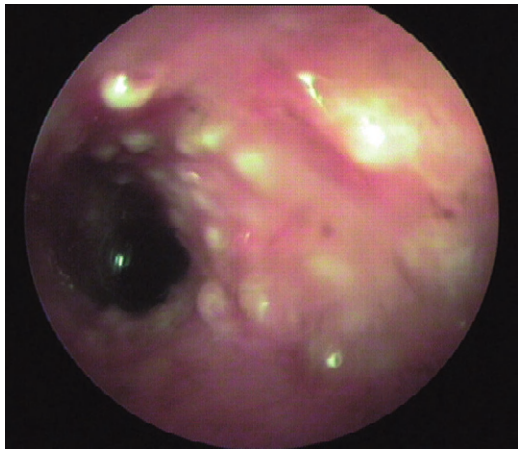


Figure 5.13 Pustules in the ear canal of a dog with pemphigus foliaceus.

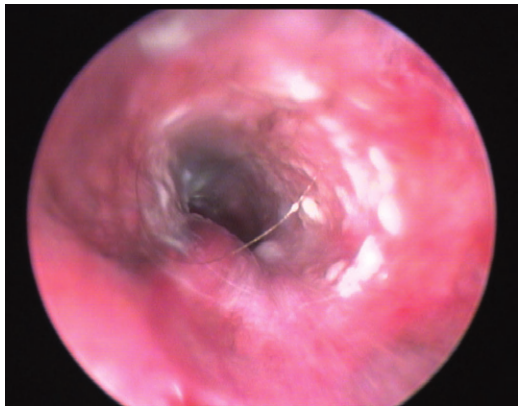


Figure 5.14 Ear canal in a dog with juvenile cellulitis.

Clinical Signs

Ear canal is swollen, erythematous, ulcerated and painful (Fig. 5.14). The discharge is often sero-purulent in nature but in the early stages of the disease is sterile. In more chronic disease Gram-negative infection can occur.

5.6.2 Proliferative and Necrotizing Otitis

Aetiology and Pathogenesis

Rare disorder of unknown aetiology, which has only been reported in a small number of cats. All cats have been young (<5 years of age). Its response to immune-modulating drugs may suggest a possible immune-mediated cause.

Clinical Signs

Affected cats have large tan to dark brown-black coalescing plaques (Fig. 5.15) covering the concave surface of the ear pinnae and external ear canals. Friable material from the plaques and thick dark exudates occludes the ears. Secondary infection with bacteria and yeast is common.

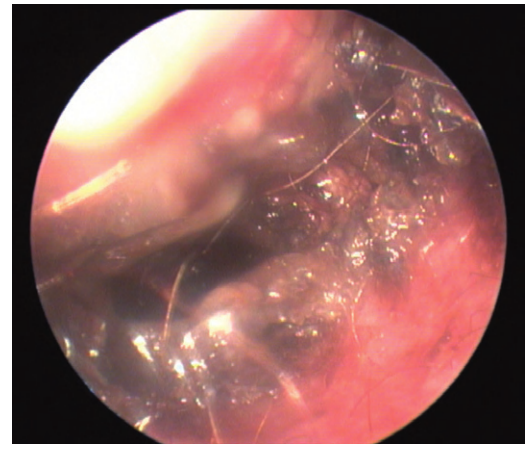


Figure 5.15 Ear canal in a cat with proliferative and necrotizing otitis.

Key Diagnostic Tests

- History and clinical signs.
- Biopsy reveals acanthosis with pronounced hair follicle root sheath hyperplasia and neutrophilic luminal folliculitis, follicular hyperkeratosis. Necrotic keratinocytes are found in the outer root sheath.

5.7 FOREIGN BODIES

5.7.1 Naturally Occurring – Grass Awns, Dirt, Sand, Broken or Loose Hairs

Clinical Signs

Most commonly seen in young hunting type dogs who present with an acute onset, painful, unilateral otitis externa, often with no previous history of ear disease. Palpation of the vertical canal usually elicits pain. The foreign body can usually be seen on otoscopic examination (Fig. 5.16a). Some foreign bodies are completely asymptomatic (Fig. 5.16b). However, material can migrate into the deepest portions of the vertical canal (Fig. 5.16c), rupturing the tympanic membrane and causing otitis media.

5.7.2 Iatrogenic – Fluid or Powder Medication

Clinical Signs

Ear disease is acute in onset and is often associated with a history of a visit to the groomer or vet. Medication can accumulate due to poor epithelial migration or due to excessive application without removal by an owner. Powder can become impacted at the base of the canal (Fig. 5.17) and may need mechanical removal, or the overuse of ear

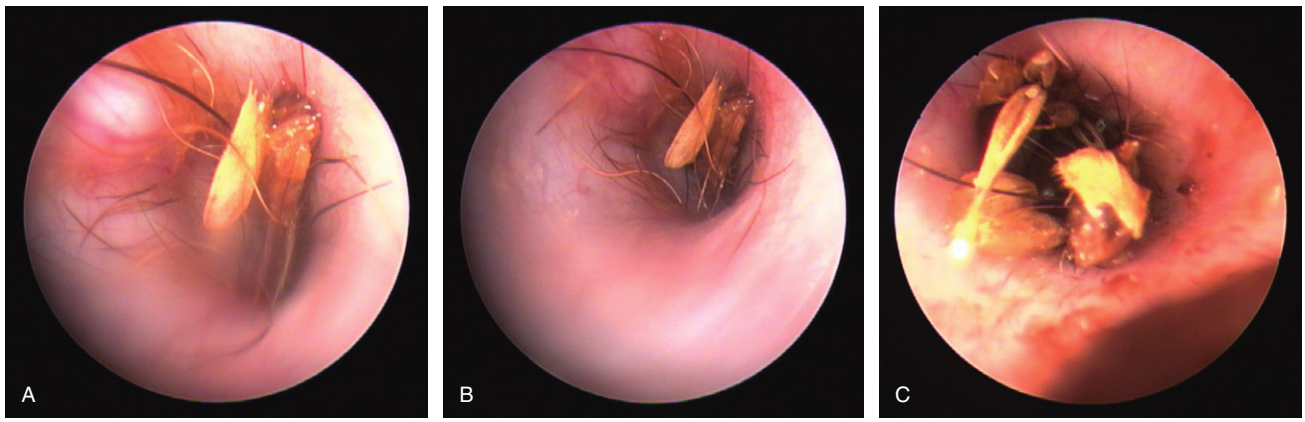


Figure 5.16 Foreign bodies within the ear canals of dogs (**A–C**).

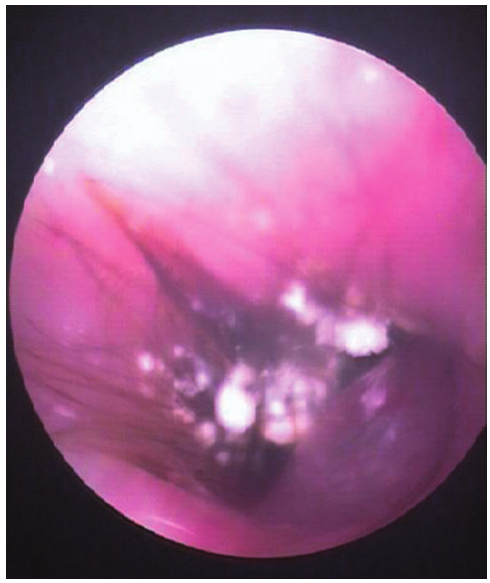


Figure 5.17 Canker powder within the ear canal of a dog.

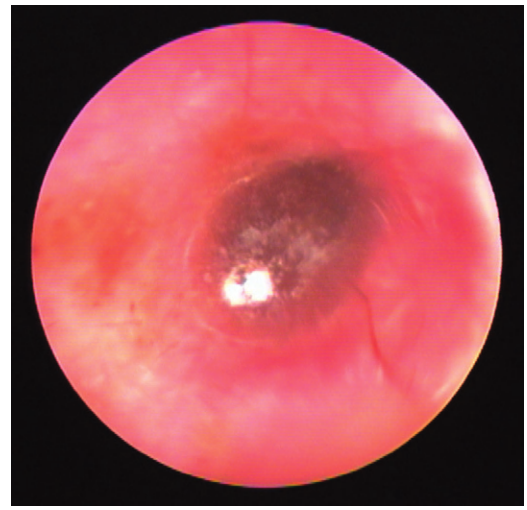


Figure 5.18 Fluid within the ear canal.

drops or flush solution can accumulate in the horizontal canal (Fig. 5.18). Where material accumulates in the horizontal canal the dog can present with head shaking or with a reduction in hearing.

5.7.3 Failure of Epithelial Migration – Ceruminolith

Aetiology and Pathogenesis

When damage to the keratinocytes on the surface of the tympanic membrane occurs due to infection or ear mite infestation, healing of the tympanum can be accompanied by fibrosis. Damage to the germinal epithelium in this way leads to a loss in normal migration which normally emanates from the central depression of the tympanum, the umbo. Wax and keratin accumulate at the base of the eardrum and form either soft wax plugs (Fig. 5.19) or large hard concretions (2–4 cm) or ceruminoliths (Fig. 5.20). Ceruminoliths may become attached to the short hairs that originate from around the annulus of the tympanic membrane or to the eardrum itself.

Clinical Signs

When ceruminoliths become impacted in the canal they cause discomfort that leads to head shaking or rubbing. Some ceruminoliths are quite large (Fig. 5.21). If the ceruminolith becomes attached to the hairs in the horizontal canal, head shaking leads to acute discomfort (Fig. 5.22). Where the ceruminolith is attached to the tympanum, head shaking can lead to eardrum rupture when material is released into the middle ear (Fig. 5.23), leading to otitis media. Compacted material in the horizontal canal can

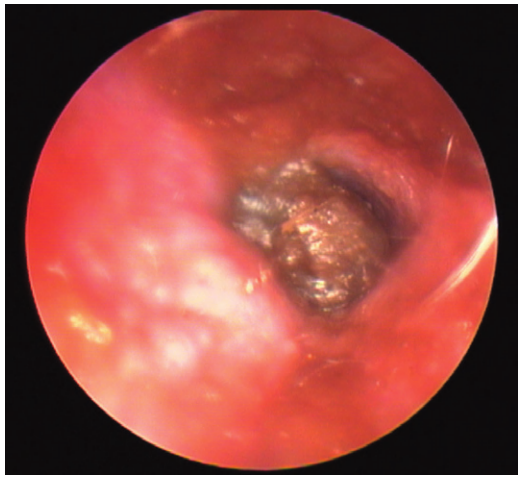


Figure 5.19 A soft ceruminolith impacted in the ear canal.

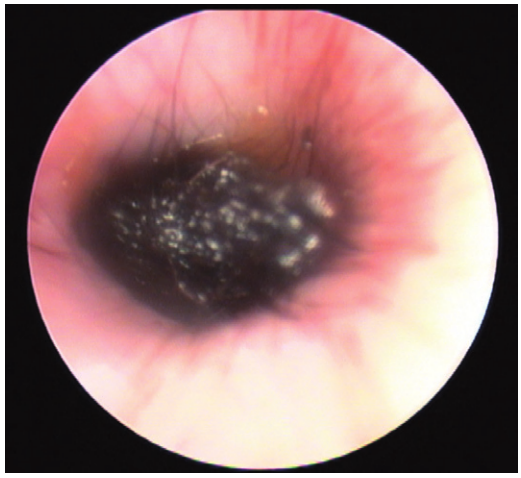


Figure 5.20 A hard ceruminolith impacted in the ear canal.



Figure 5.21 Ceruminolith after removal showing the size of the impaction compared to a needle.

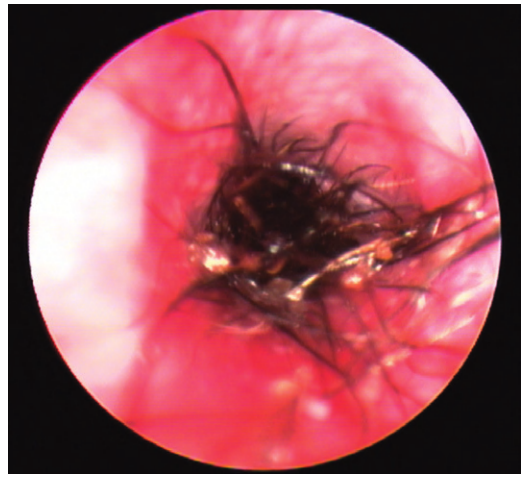


Figure 5.22 Ceruminolith attached to hairs in the horizontal canal.

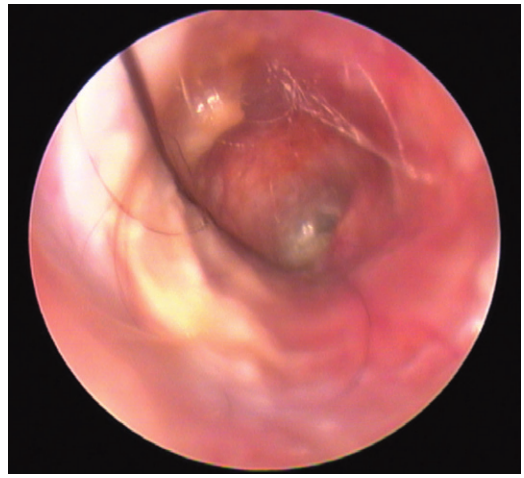


Figure 5.23 Ceruminolith that has penetrated the tympanic membrane. The picture shows hairs sticking out from the tympanic membrane.

lead to a reduction in hearing. Where the weight of the ceruminolith causes an increase in pressure within the middle ear cavity, vestibular disease can ensue.

5.8 NEOPLASTIC AND HYPERPLASTIC CONDITIONS OF EAR CANAL

Tumours of the ear canal in dogs are uncommon. In dogs the ratio between benign and malignant tumours is about equal. In the dog the most common tumours are those arising from ceruminous glands (Table 5.2).

Most tumours of the ear canal in the cat are malignant often being carcinomas derived from the epithelial and

Table 5.2 Neoplastic and hyperplastic conditions of the canine ear canal

Benign neoplasms	Malignant neoplasms
Ceruminous gland adenoma/hyperplasia (Fig. 5.24)	Ceruminous gland adenocarcinoma
Basal cell tumour	Squamous cell carcinoma
Papilloma (Fig. 5.25)	Soft tissue sarcoma (Fig. 5.27)
Sebaceous gland adenoma (Fig. 5.26)	Malignant melanoma
Histiocytoma	
Plasmacytoma	



Figure 5.24 Ceruminous gland adenomas in a dog.

adnexal structures of the auditory meatus. The most common tumours are those arising from the ceruminous glands. Table 5.3 lists the most common neoplastic and hyperplastic conditions of the feline ear canal.

5.8.1 Benign Tumours

Ceruminous Gland Adenoma

Aetiology and Pathogenesis

These are common benign tumours of the external ear canal. They are most frequently seen in middle aged to elderly animals.

Table 5.3 Neoplastic and hyperplastic conditions of the feline ear canal

Benign neoplasms	Malignant neoplasms
Ceruminous gland adenoma (Fig. 5.28)	Ceruminous gland adenocarcinoma
Sebaceous gland adenoma	Squamous cell carcinoma
Fibroma	Sebaceous gland adenocarcinoma



Figure 5.25 Papillomas in the ear canal.

Clinical Signs

These tumours are raised and occasionally pedunculated (Fig. 5.29), may be solitary (Fig. 5.30) or multiple (Fig. 5.31), pink (Fig. 5.32), or melanotic (Fig. 5.33). Tumours usually manifest clinical signs once they occlude the canal, when animals will present with pruritus and head shaking. Secondary infection is common often with Gram-negative bacteria which produce a malodorous discharge. Broad-based ceruminous gland adenomas are recognized in dogs as a subset of these types of tumours (Fig. 5.34). These lesions appear as broad-based polyps attached to the wall.

Key Diagnostic Tests

- Pinch biopsies are often not diagnostic. In the case of broad-based tumours they can be misleading, being suggestive of polyps as biopsies reveal inflammatory stroma with inflammatory cells covered by epithelium.



Figure 5.26 Sebaceous gland adenoma in a dog.



Figure 5.27 Malignant soft tissue sarcoma.

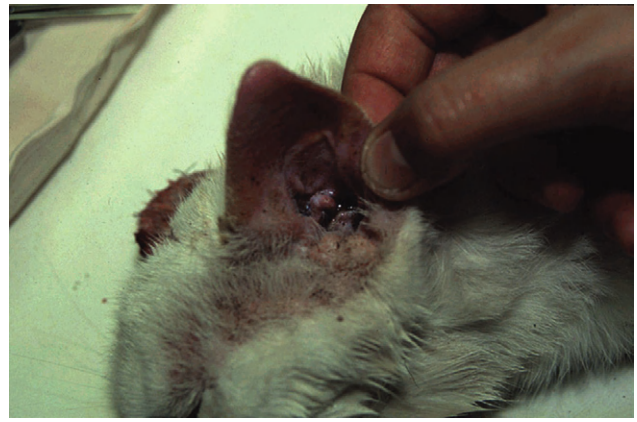


Figure 5.28 Ceruminous gland adenoma in a cat.

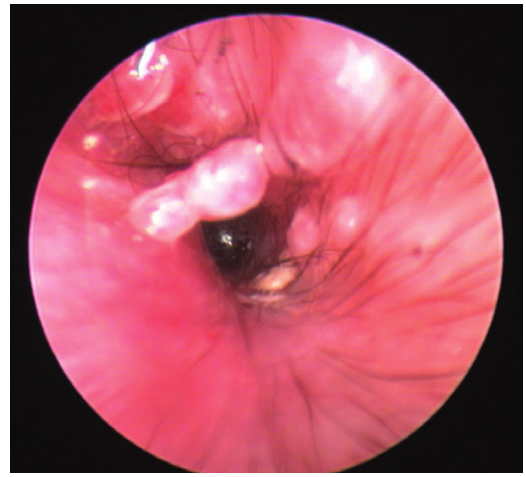


Figure 5.29 Pedunculated ceruminous gland adenomas.

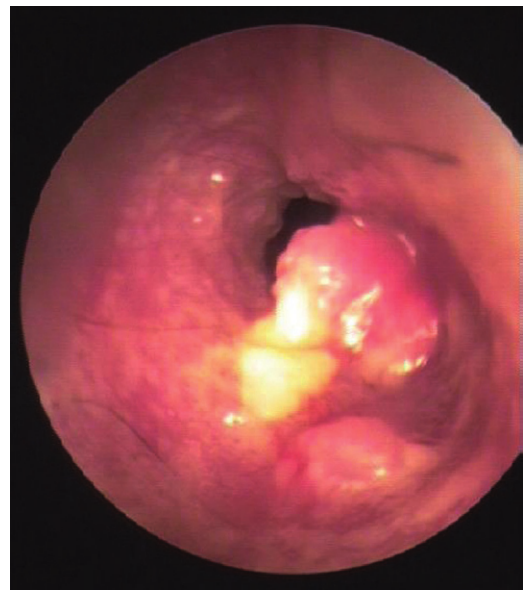


Figure 5.30 A solitary ceruminous gland adenoma.

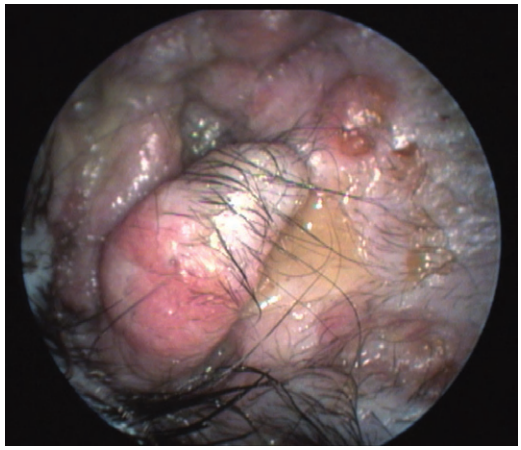


Figure 5.31 Multiple ceruminous gland adenomas.

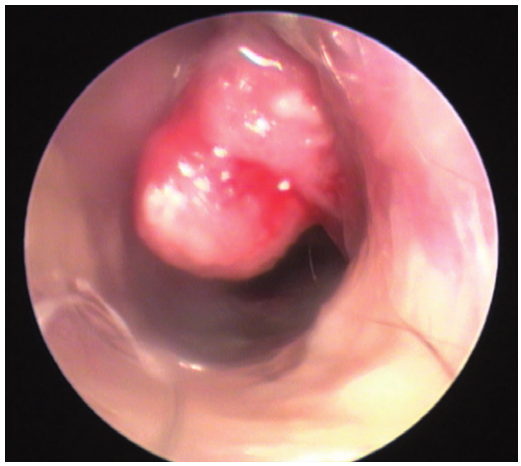


Figure 5.32 Pink ceruminous gland adenomas.

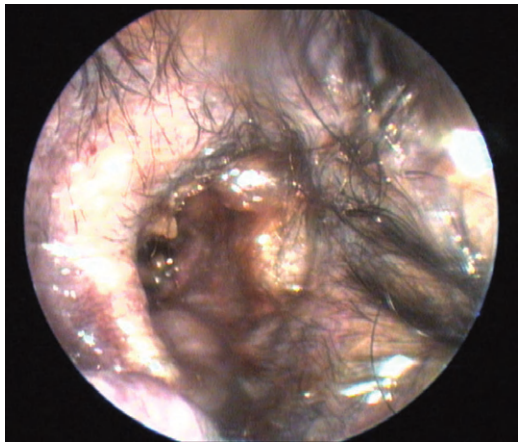


Figure 5.33 Melanotic ceruminous gland adenomas.

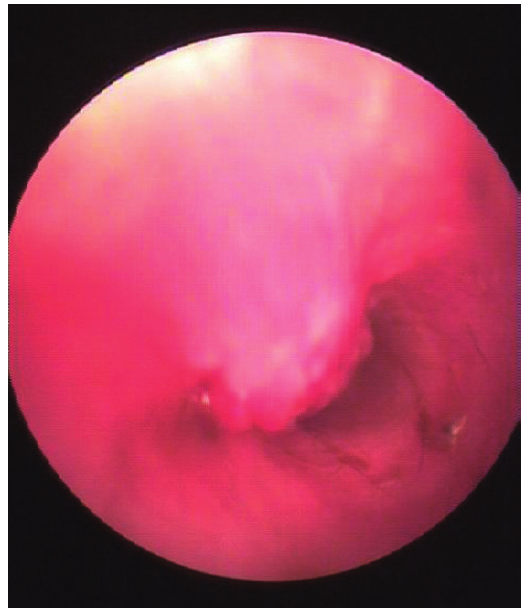


Figure 5.34 Wide-based atypical ceruminous gland adenomas.

- Excisional biopsy of the affected area of the canal is recommended to confirm a diagnosis; histopathology reveals a lobulated tumour subdivided by fine connective tissue trabeculae. The lobules may show central aggregations of green/brown cerumen.

5.8.2 Malignant Tumours

Ceruminous Gland Adenocarcinoma

Aetiology and Pathogenesis

A malignant tumour derived from ceruminous glands. This tends to be invasive and ulcerative rather than occlusive. Most cases occur in old animals with an average age in cats of 12 years and dogs of 9 years.

Clinical Signs

Usually presents as a unilateral recurrent infection, with a haemorrhagic, malodorous, purulent discharge, in older dogs and cats (Fig. 5.35). The symptoms respond poorly to topical or systemic medication. The lesion in the canal is most often pink, ulcerated, and friable (Fig. 5.36). Ipsilateral mandibular lymphadenopathy is common. Extension of the lesion into the bulla is said to occur in about 50% of cases.

Key Diagnostic Tests

- Radical excisional biopsy including the bulla is recommended; histopathology reveals a similar picture to the ceruminous gland adenoma, but the cell types demonstrate more malignant criteria.

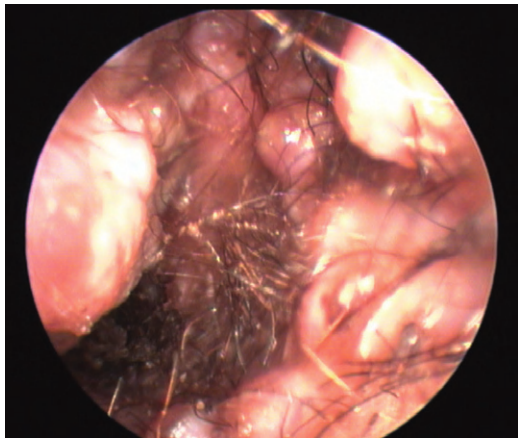


Figure 5.35 Malignant ceruminous gland adenocarcinoma in a dog.

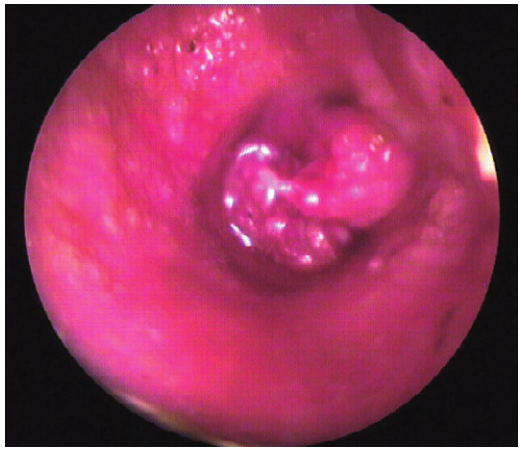


Figure 5.36 Malignant ceruminous gland adenocarcinoma in a cat.

Squamous Cell Carcinoma

Aetiology and Pathogenesis

A malignant, usually fast-growing, tumour derived from the epidermis. Cases tend to be more common in cats than in dogs. The mean age in cats is 11 years and dogs 10 years.

Clinical Signs

Animals usually present with a painful unilateral otitis externa. The aural discharge is usually haemorrhagic, malodorous, and purulent. Tumours are proliferative and ulcerative with a tendency to infiltrate locally (Fig. 5.37).

Key Diagnostic Tests

- Radical excisional biopsy including the bulla is recommended; histopathology reveals cords and nests of cells within the dermis which can infiltrate deep tissues. Laminated masses of keratin, 'keratin pearls', are seen in well and moderately differentiated tumours.

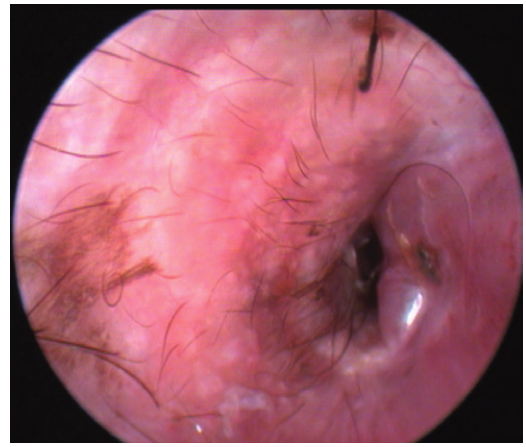


Figure 5.37 Infiltrative malignant squamous cell carcinoma in the horizontal canal of a dog.



Figure 5.38 Pendulous pinnae in a Bassett.

5.9 PREDISPOSING FACTORS IN OTITIS EXTERNA

Predisposing factors do not cause disease in their own right but contribute to disease. These include such things as the conformation of the ear flap and also the lifestyle of the dog or cat. This is particularly important in working dogs if they swim and the ear canals are constantly being wetted, or in show dogs where the canal may be traumatized by over-exuberant plucking of hairs. Treatment effects are also important; otic drops or cleaners can either lead to irritant reactions in the canal, due for instance to the fact they are astringent or have a low pH or where they modify the flora within the ear canal leading to secondary infection.

5.9.1 Conformation Effects on Otitis Externa

The presence of large, pendulous ear pinnae (Fig. 5.38) or ear pinnae that have a hairy concave surface can predispose

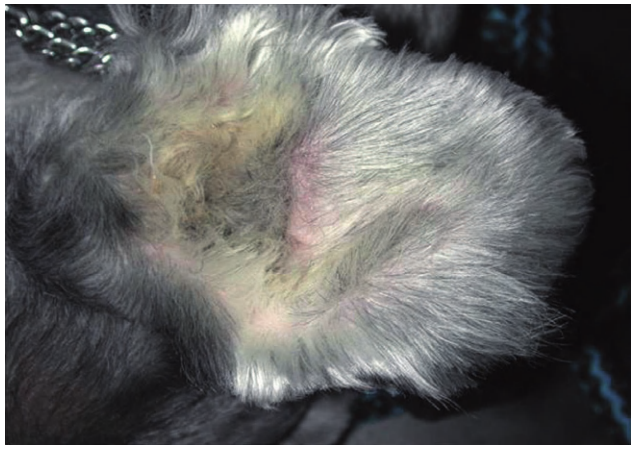


Figure 5.39 Hairy ear canal in a Spaniel.

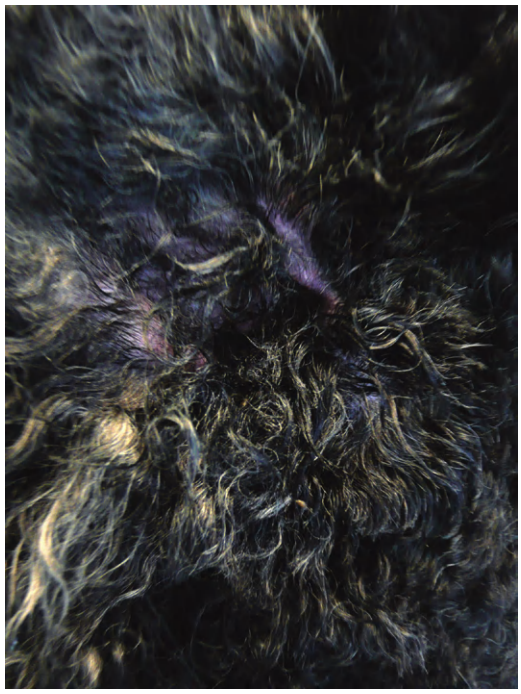


Figure 5.40 Hairy ear canal in a Poodle.

to disease. Within the canals the presence of large amounts of hair as recognized in the Spaniel (Fig. 5.39) or Poodle (Figs 5.40, 5.41), or a very narrow canal which can be seen in such breeds as the Shar Pei (Figs 5.42, 5.43) may increase the risk of development of disease.

5.9.2 Treatment Effects

The use of inappropriate topical therapy can lead to disease within the ear canal. Cleaners should be used routinely as part of the normal therapeutic regime employed to treat otitis externa. However, when the ear canal is ulcerated and painful, especially in diseases complicated by Gram-negative infection, the use of cleaners with a low pH or potent ceruminolytic cleaners can damage the canal (Figs 5.44, 5.45). Long-term use of occlusive oil-based cleaners can lead to the development of infection. Chronic antibiotic usage can lead to the overgrowth of resistant bacteria such as methicillin-resistant staphylococcal infection, *Ente-*



Figure 5.42 Shar Pei showing narrow ear canal.

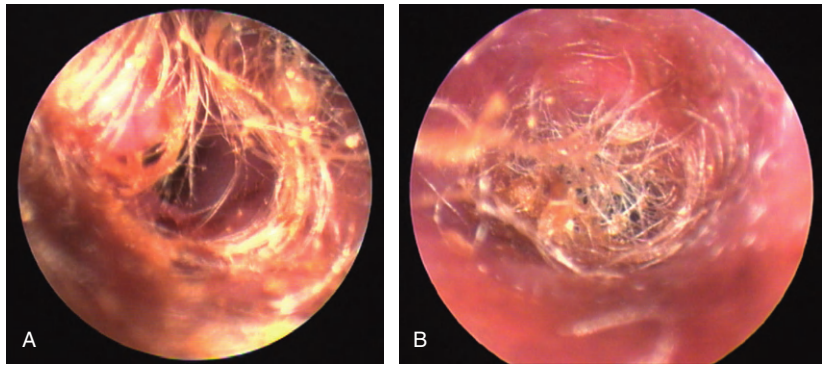


Figure 5.41 Hair within the ear canal (**A, B**).

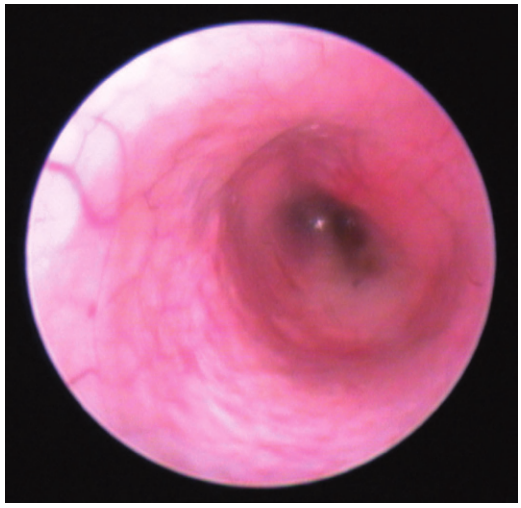


Figure 5.43 Video otoscopic picture of narrowed ear canal in a Shar Pei.

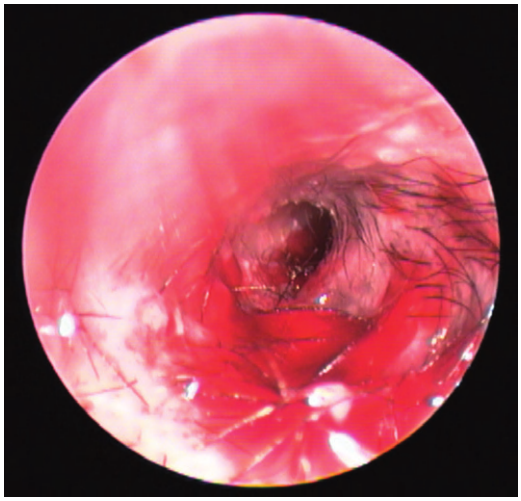


Figure 5.44 Ulcerated ear canal. Further damage will be produced by use of inappropriate cleansers.

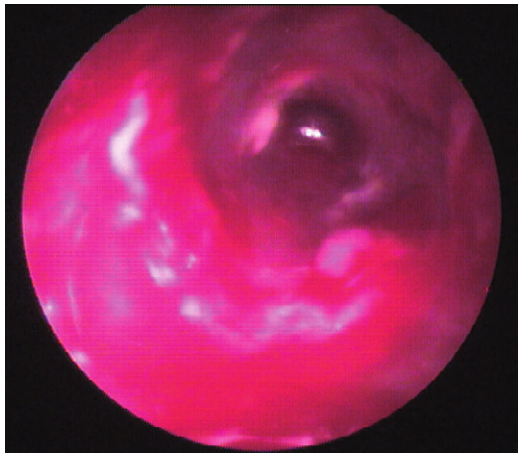


Figure 5.45 Severely ulcerated ear canal caused by overuse of an acidic ear cleaner.

rococcus faecalis, or anaerobic infection. The application of large volumes of topical drugs to the ear can lead to pooling of material of medication in the horizontal canal. Some topical drugs are known to be common triggers for contact hypersensitivity: these include neomycin and silver sulphadiazine as well as propylene glycol, which is a common base for many different ear products.

5.9.3 Excessive Wetting of the Ear Canals

When water is allowed to stand for prolonged periods of time inside the canal it leads to maceration of the ear canal and overgrowth of yeast, leading to infection. This is commonly seen in gun dogs that swim and submerge their heads to fill their ears with water. Water-based medication such as aqueous solutions of Tris–EDTA and antibiotics in water will also lead to canal maceration and yeast infection (Fig. 5.46).

5.9.4 Inappropriate Use of Cotton Tip Applicators and Dry Cotton Wool

Cotton tip applicators are best employed to remove fluid from an ear canal. Repeated insertions of the tip into the ear canal while restricting its movement can absorb fluid on to the tip; this may be repeated until the tip comes out of the ear dry. If an infected, inflamed, friable, oedematous ear canal wall is scraped with an applicator as part of an over-exuberant cleaning process this action may lead to ulceration of the canal wall (Fig. 5.47) and subsequent infection. Similarly, if the applicator is used to try to hook material out of the canal it may actually push material down into the horizontal canal, which may in turn lead to an impaction of material in the canal or damage to the tympanum and a resulting otitis media.

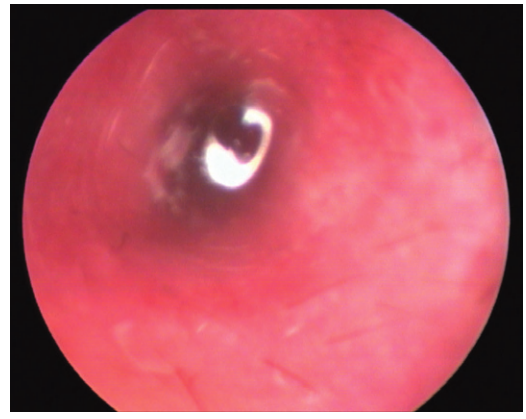


Figure 5.46 Water pooled in the ear canal.



Figure 5.47 Over-exuberant cleaning, particularly with dry cotton wool, can lead to ulceration of the sensitive ear canal.

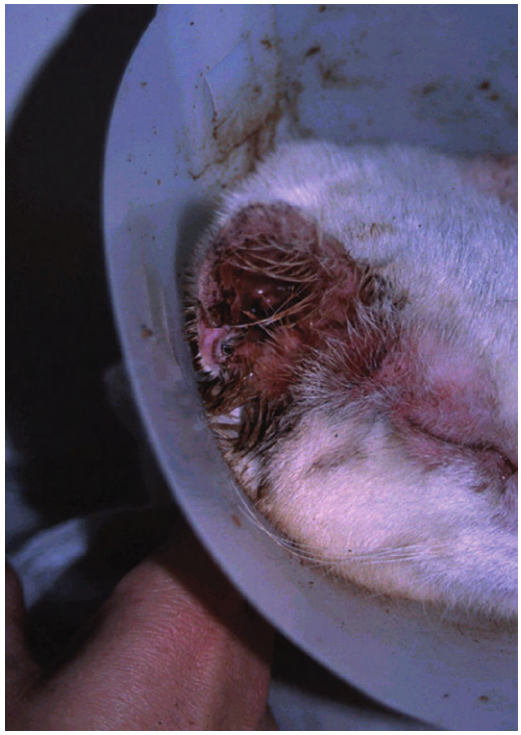


Figure 5.48 Severe bacterial infection in cat with feline leukaemia virus.

5.9.5 Systemic Disease

Factors that cause immunosuppression can predispose animals to infection. This is particularly important in cats with feline leukaemia virus (FeLV) or feline immunodeficiency virus (FIV) (Fig. 5.48) and is also recognized in dogs with renal, pancreatic, or hepatic disease.



Figure 5.49 Staphylococcal ear infection showing typical discharge.

5.10 PERPETUATING FACTORS

Perpetuating factors, like predisposing factors, do not cause disease but drive the process once it has been established and prevent resolution of the disease even after the primary trigger has been identified and treated successfully. The four main factors are infection with bacteria and yeast, otitis media, and chronic change in the canal.

5.10.1 Bacterial Infection

Gram-Positive Infection

Typical discharge where Gram-positive infection is identified is a waxy/yellow purulent discharge (Fig. 5.49). The wall of the canal is erythematous, oedematous, and hyperplastic (Fig 5.50a, b) and moderately pruritic. In chronic disease the eardrum may be ruptured (Fig. 5.50c).

Gram-Negative Infection

The typical discharge where Gram-negative infection is identified is a yellow/green malodorous mucoid discharge (Figs 5.51–5.53). The wall of the canal is erythematous, commonly ulcerated and painful (Figs 5.54, 5.55). In chronic disease it is unusual for the eardrum to be intact. In addition to the slime produced as a protective mechanism by some Gram-negative bacteria that is mixed with the pus, the mucoperiosteum of the tympanic bulla also

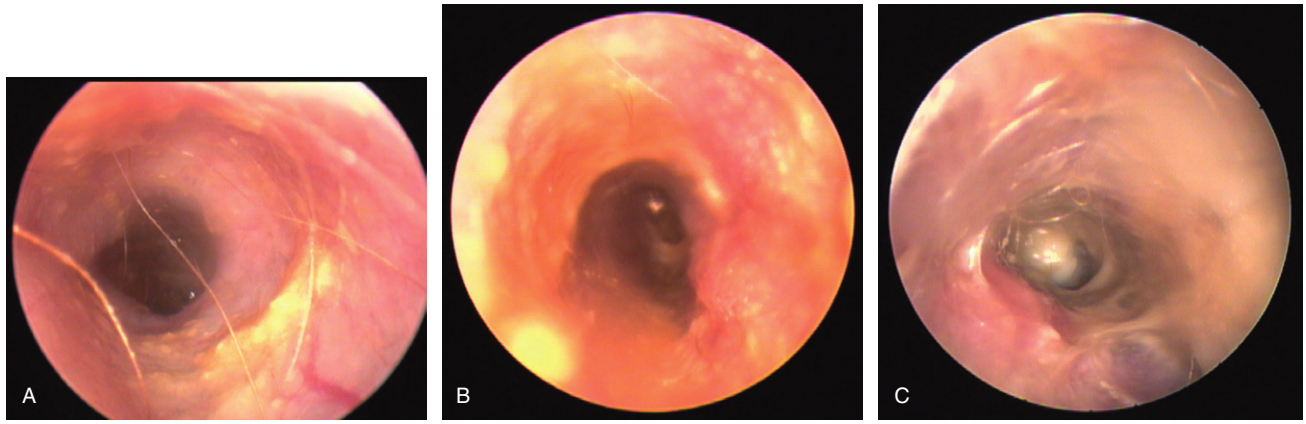


Figure 5.50 Ear canal of dog with staphylococcal infection (**A–C**).



Figure 5.51 Pseudomonas infection in dog showing typical discharge.

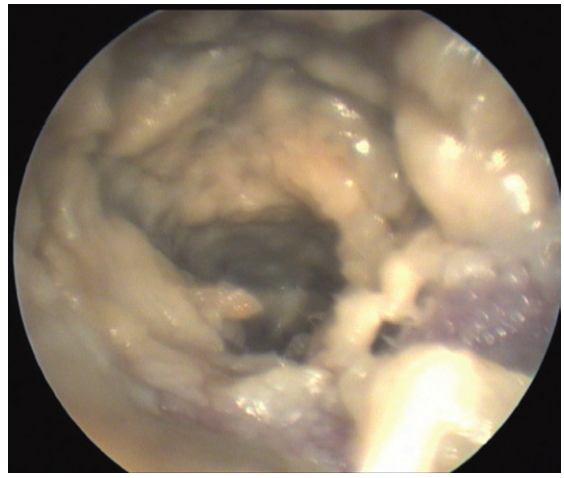


Figure 5.53 Typical ear canal in a case of pseudomonas otitis showing the thick caseous discharge.



Figure 5.52 Mucoid discharge typical of pseudomonas infection.

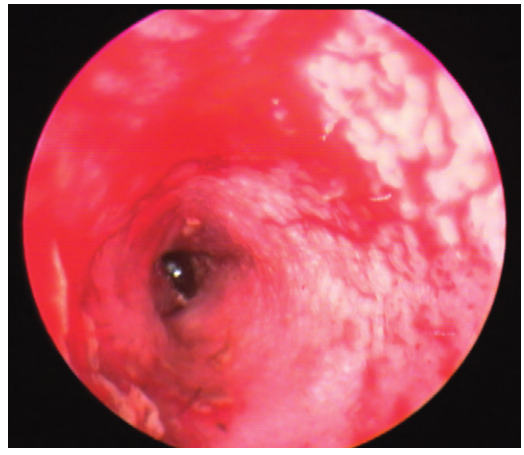


Figure 5.54 Ulcerated painful ear canal in pseudomonas otitis.

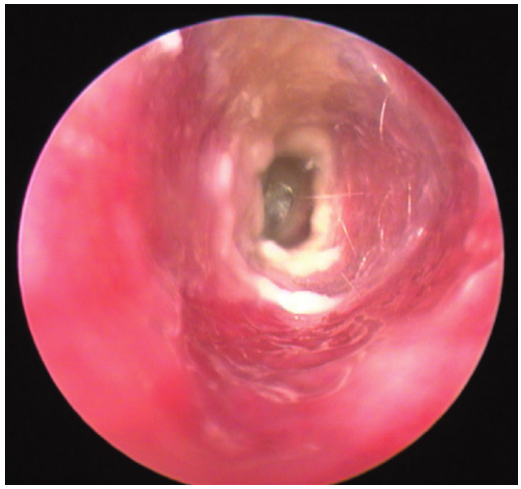


Figure 5.55 Severely damaged ear canal with loss of eardrum in a case of pseudomonas otitis.



Figure 5.56 Typical discharge in a case of malassezia otitis.

produces mucus that can become mixed with the aural discharge.

5.10.2 Yeast Infection

The typical discharge when yeast infection is identified is a thick ceruminous discharge (Fig. 5.61). The wall of the canal is oedematous and hyperplastic (Figs 5.57, 5.58), but ulceration is uncommon. The canal is usually highly pruritic. In chronic disease ceruminous gland hyperplasia can lead to narrowing of the lumen (Fig. 5.59).

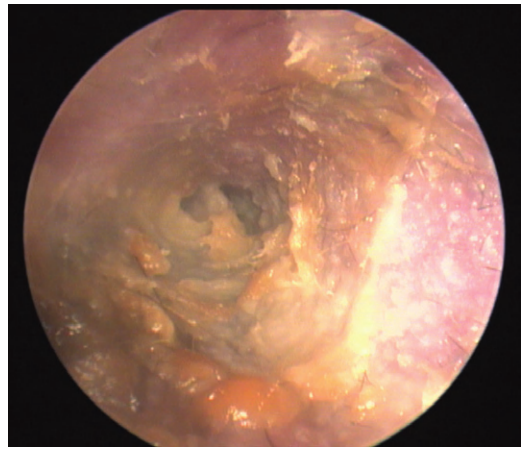


Figure 5.57 Typical ear canal in malassezia otitis.

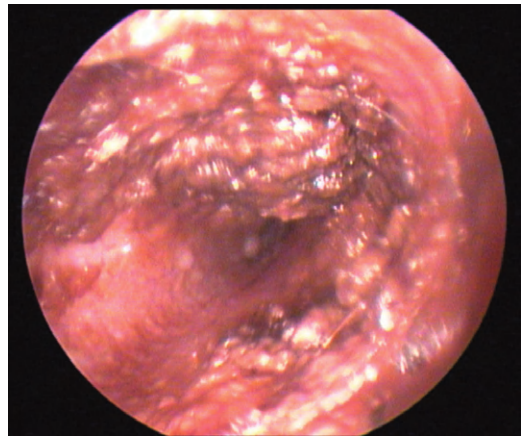


Figure 5.58 Malassezia otitis showing thick ceruminous discharge.

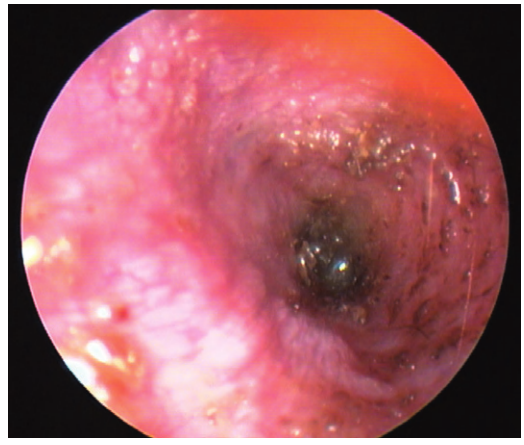


Figure 5.59 Ceruminous gland hyperplasia in a case of a malassezia otitis.

5.10.3 Otitis Media

See Chapter 6.

5.10.4 Chronic Change

Changes Due to Inflammation

These changes will usually lead to a reduction in the lumen of the canal. In acute disease this is caused by dermal oedema caused by dilation of blood vessels and increased vascular permeability. These changes are easily reversed by anti-inflammatory medication. However, as the disease process becomes more chronic, reversibility becomes less likely. Chronic inflammation leads to changes within the simple migratory epithelium which becomes a hyperplastic stratified squamous epithelium. As the disease process progresses, hyperplasia of the three main cell types in the dermis occurs: namely, the sebaceous glands, ceruminous glands, and fibroblasts. As glandular tissue increases the lumen of the canal is reduced further, causing more severe stenotic change. Different breeds appear to differ in their response to chronic inflammation. In some dogs hyperplasia is predominantly glandular due to changes in the sebaceous and ceruminous glands. Cocker Spaniels are recognized as demonstrating ceruminous gland hyperplasia. When the glandular tissue is affected the hyperplasia of the glandular tissue produces a 'cobblestone' appearance. The progression of the disease can be seen in Figs 5.60–5.63. In

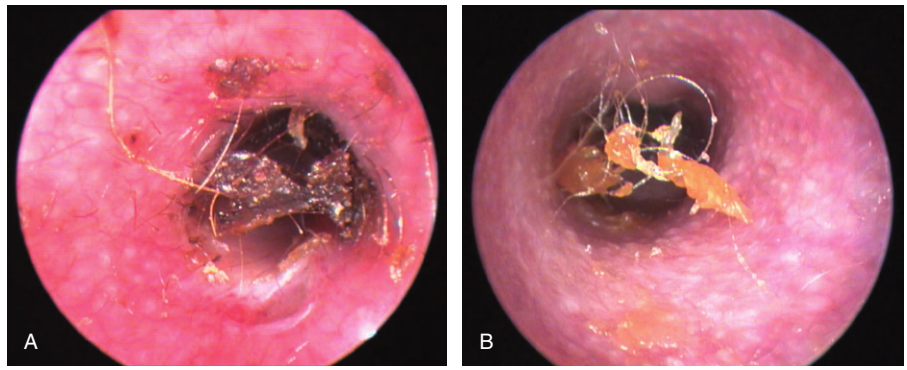


Figure 5.60 Early ceruminous gland hyperplasia (**A, B**).

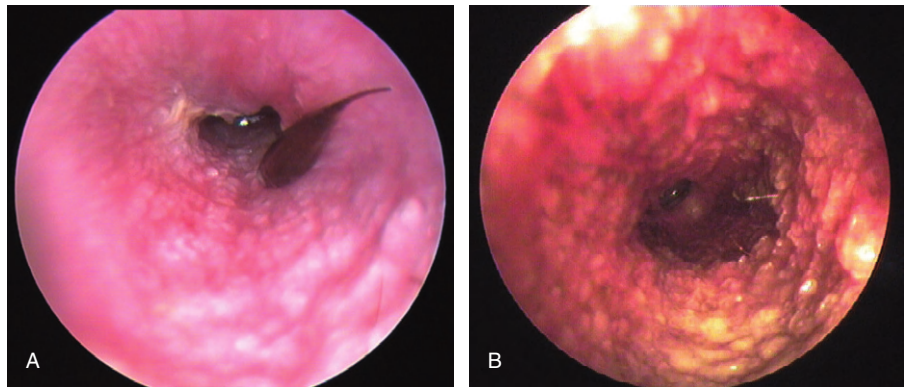


Figure 5.61 Moderately severe ceruminous gland hyperplasia (**A, B**).

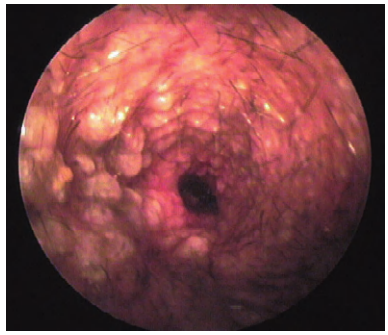


Figure 5.62 Severe ceruminous gland hyperplasia.

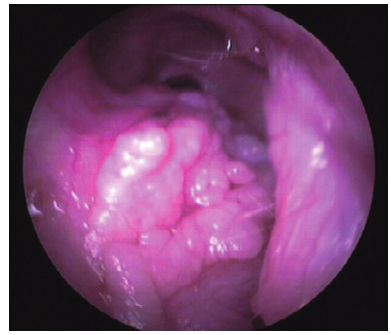


Figure 5.63 Very severe ceruminous gland hyperplasia showing the formation of polypoid growths.

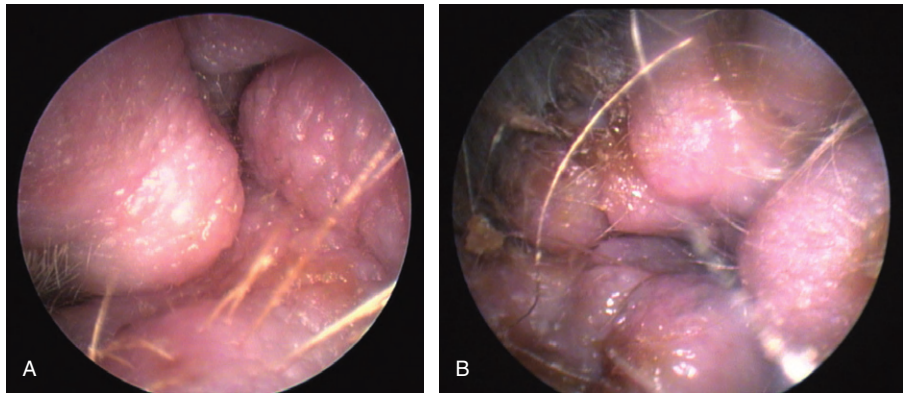


Figure 5.64 Chronic fibrotic change in the ear canal of a dog (**A, B**).

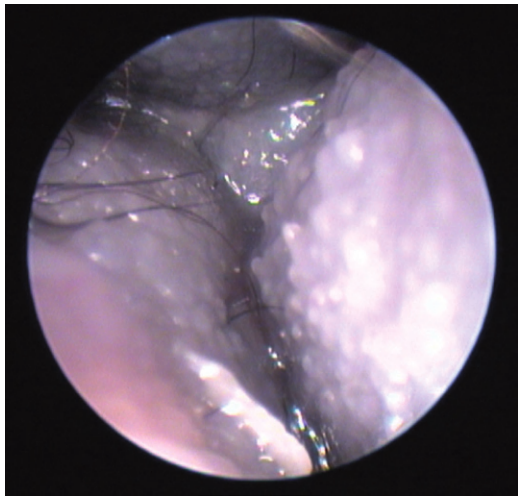


Figure 5.65 Severe irreversible fibrotic change in the ear canal of a dog.

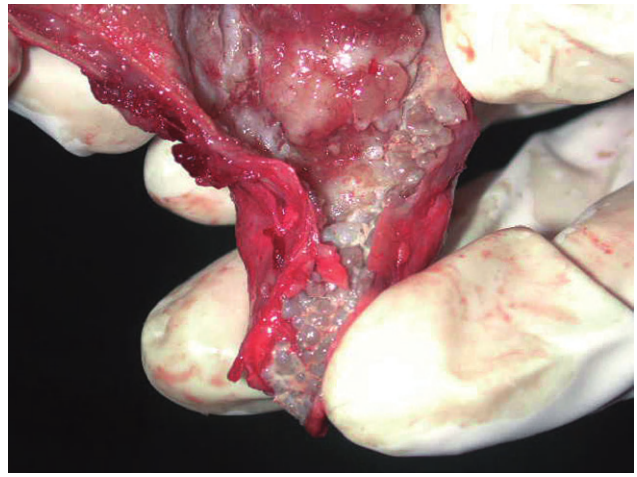


Figure 5.67 Irreversible glandular change in the ear canal of a dog.

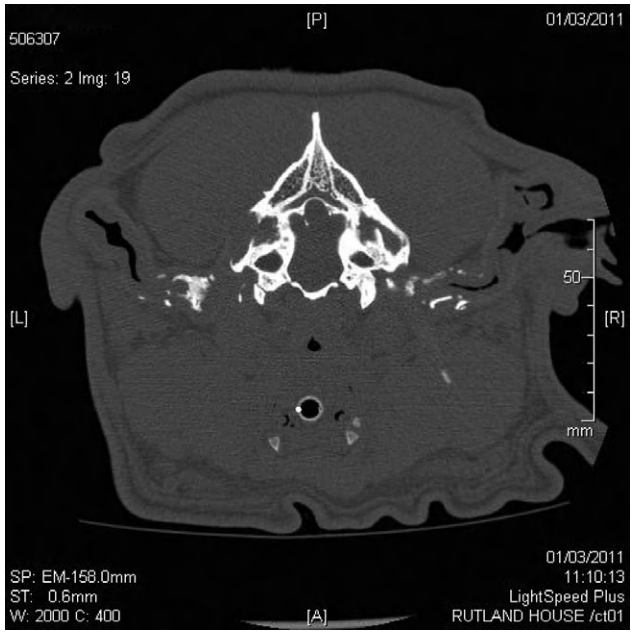


Figure 5.66 CT showing signs of severe otitis media with calcification of the canal.

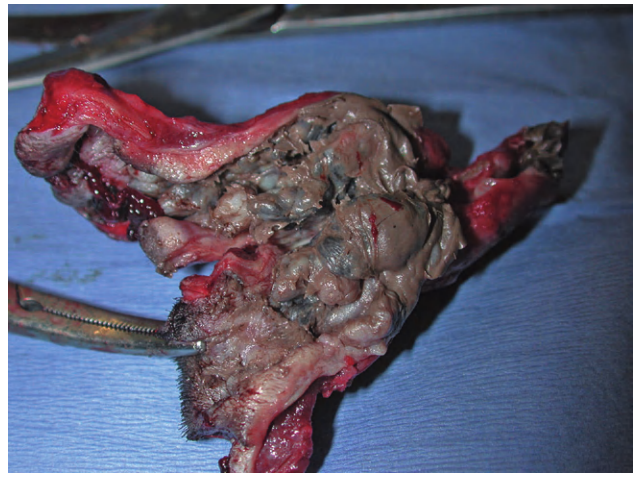


Figure 5.68 Severe irreversible glandular change in the ear canal of a dog.

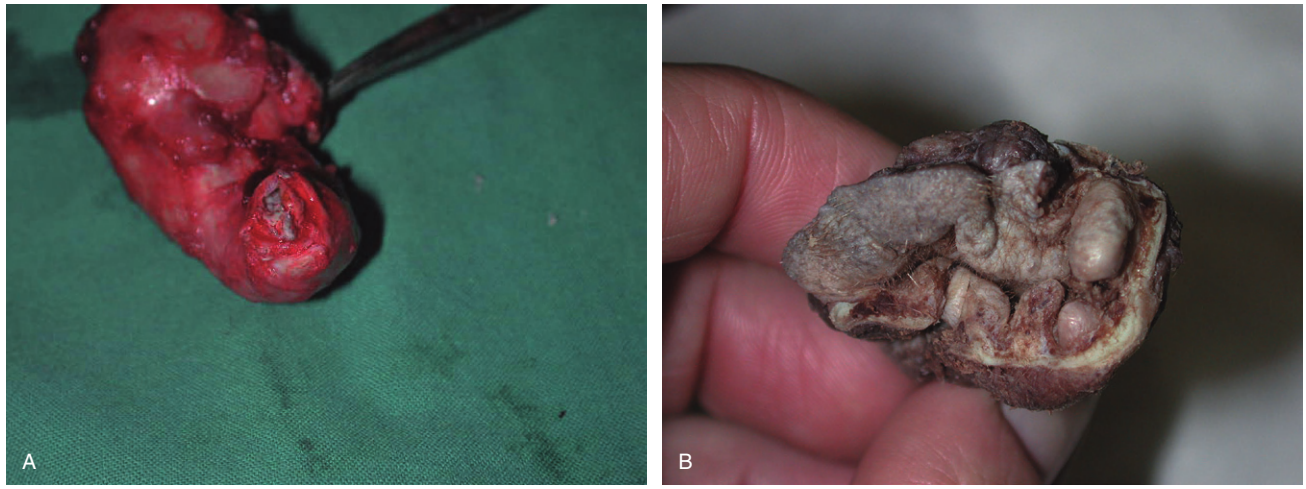


Figure 5.69 Irreversible fibrotic change in the ear canal of a dog (**A, B**).

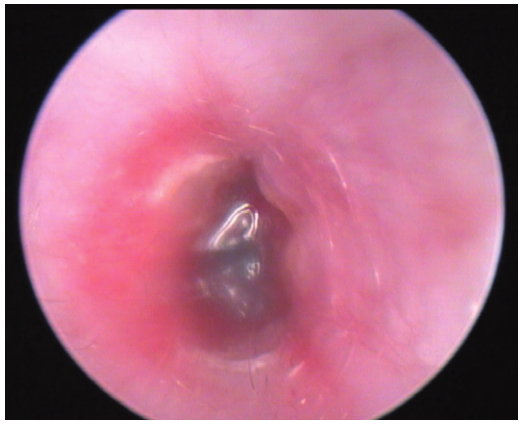


Figure 5.70 Partial stenosis of ear canal due to trauma.

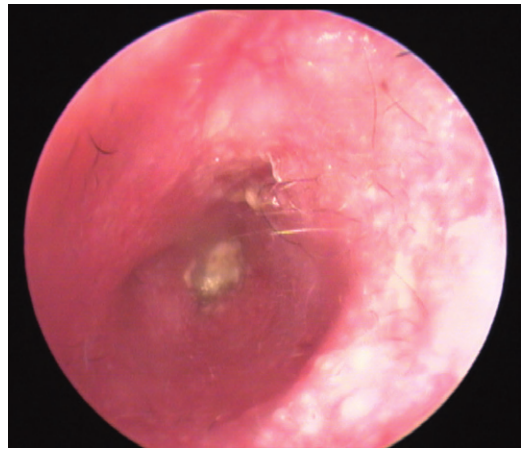


Figure 5.72 Stenosis of the ear canal due to abnormal healing after surgery.

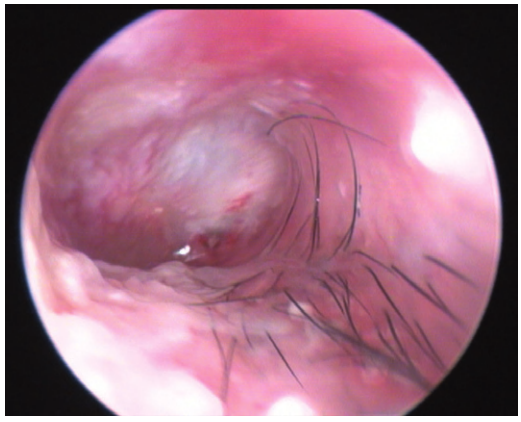


Figure 5.71 Complete stenosis of ear canal as a result of trauma.

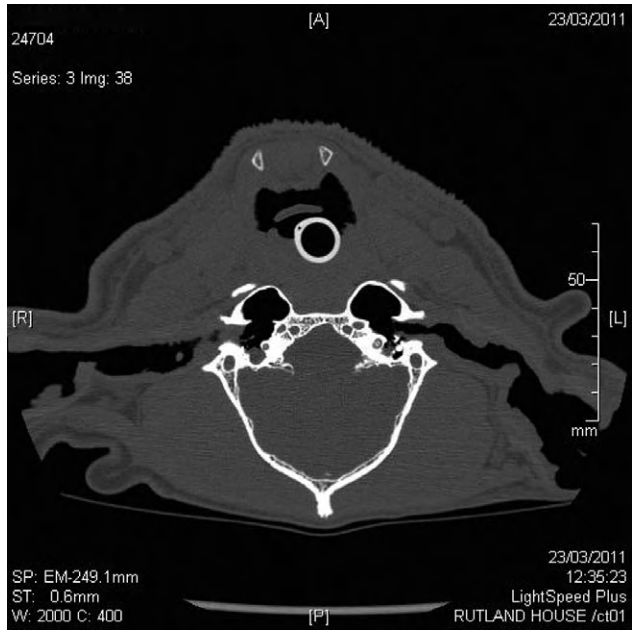


Figure 5.73 CT scan showing stenosis of the right ear canal in the horizontal portion of the canal.

VetBooks.ir

other dogs the changes are principally fibrotic (Figs 5.64, 5.65). Fibrosis can unfortunately progress to osseous metaplasia as the soft tissue within the canal becomes ossified (Fig. 5.66). Ears at this stage are often referred to as being chronically irreversibly damaged and commonly need radical surgery (Figs 5.67–5.69).

Changes Due to Trauma

These usually result in stenosis of the ear canal. Acute trauma to the ear pinna, where it is pulled sharply, can produce damage at the junction of the auricular and annular cartilages. This often causes separation of the canal at the point of weakness and subsequent stenosis (Figs 5.70–5.73). Other damage to the canal can be caused by road traffic accidents, bite wounds, and postoperative fibrosis after laser surgery.

VetBooks.ir

CHAPTER 6
CONDITIONS OF THE MIDDLE EAR

Sue Paterson

6.1	Aetiology and Pathogenesis of Acute Otitis Media	107	6.2	Chronic Otitis Media	115
			6.3	Investigation of Otitis Media	115

Otitis media (OM) is by definition disease within the middle ear, and can be acute or chronic.

6.1 AETIOLOGY AND PATHOGENESIS OF ACUTE OTITIS MEDIA

OM can result from descending disease as an extension from problems within the external ear canal; as ascending disease from the nasopharynx via the eustachian tube; by haematogenous spread; or from primary disease within the tympanic bulla. In the dog OM is usually recognized as a progression from chronic infectious otitis externa. This may be due to infection, particularly with Gram-negative bacteria, or from foreign bodies within the canal perforating the tympanum. These may be extraneous material such as grass seeds or ceruminoliths caused by failure of epithelial migration.

Ascending infection, possibly from respiratory infection, occurs in the cat; descending infection is far less common. When disease spreads from the external ear canal it must penetrate the tympanic membrane. This may be because the tympanic membrane has become porous or perforated due to infection, inflammation, or a foreign body. The tympanic membrane can heal in the face of OM trapping abnormal discharge and infection within the tympanic bulla. Careful visualization of the tympanum is therefore essential when assessing an animal for OM. Cholesteatoma occurs when there is invagination of the tympanic membrane into the epitympanic recess. If the tympanic membrane becomes adherent to the cochlear promontory a cholesteatoma can form. When disease starts in the middle ear it may be caused by primary secretory abnormalities or growths within the tympanic bulla. Primary secretory otitis media (PSOM) is most commonly recognized in the Cavalier King Charles Spaniel and can occur without signs of otitis externa. Dogs present with typical signs of OM (see Table 6.2). Nasopharyngeal polyps are rare in the dog; most growths in the middle ear tend to be more aggressive neoplastic processes. Feline PSOM has not been identified. OM is more commonly caused by nasopharyngeal polyps originating within the tympanic bulla. Growths within the feline ear often present with signs of otitis externa, media, and interna. Occlusion of the external ear canal due to an extension of the polyp through the tympanic membrane into the horizontal canal commonly causes infectious otitis externa, often with Gram-negative bacteria. Growth of the polyp within the middle ear frequently leads to damage to the sympathetic nerves that traverse the bulla septum, leading to signs of Horner's disease, and where pressure is exerted on the round window vestibular disease can also be seen.

The mucoperiosteum which lines the tympanic bulla responds to inflammation by producing mucus: this traps infection within the bulla and perpetuates the inflammatory process. Changes occur within the bulla as the disease becomes more chronic and the simple cuboidal epithelium changes to a pseudostratified columnar epithelium. Where long-standing oedema and an inflammatory infiltrate are present granulation tissue forms within the bulla, making the area less accessible to therapy (Fig. 6.1). As the disease progresses, further fibrosis and calcification of the soft tissue occurs and the bulla progresses to become irreversibly damaged.

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

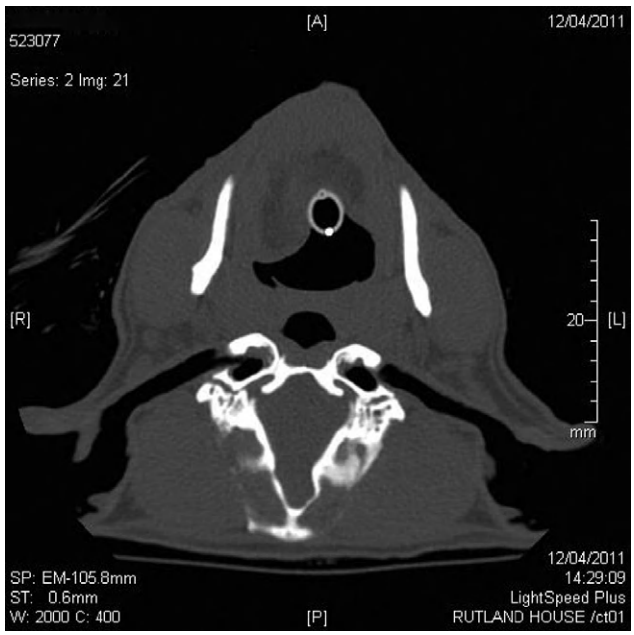


Figure 6.1 CT scan showing early formation of granulation tissue in middle ear.

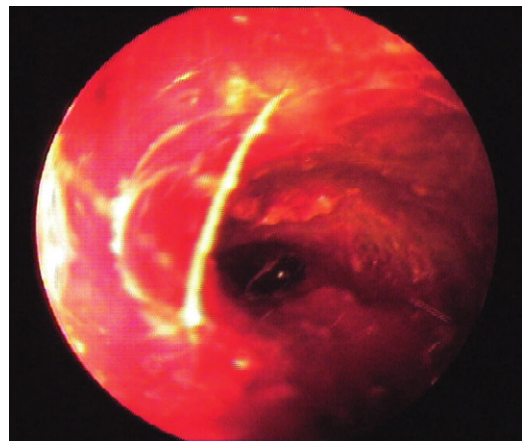


Figure 6.2 Severe pseudomonas infection.

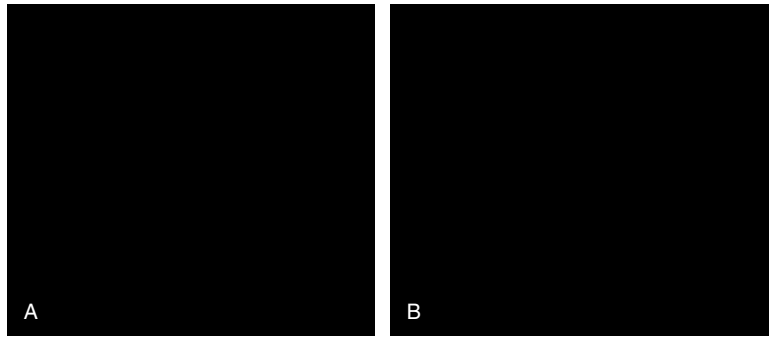


Figure 6.3 A grass seed perforating the tympanic membrane in the horizontal canal (**A, B**).

6.1.1 Otitis Media as a Result of Descending Disease

Infection

Chronic bacterial otitis externa is the most common descending infectious trigger for OM. Where infection has been present for more than 6 months it is uncommon for OM not to be present. Long-standing disease and chronic change predispose the ear to Gram-negative infection (Fig. 6.2) which tends therefore to be the most frequently identified pathogen. Malassezia rarely causes OM.

Foreign Bodies

Where foreign bodies penetrate the tympanic membrane they can lead to inflammation and secondary infection in the middle ear. Grass awns and other organic material can become embedded in wax in the horizontal canal, causing irritation and leading to head shaking and subsequent perforation of the tympanic membrane (Fig 6.3). Impacted medication, particularly some of the canker powders used by owners and groomers, can lead to damage to the eardrum. This can dry out within the canal causing subsequent damage. Ceruminoliths are accumulations of wax, skin cells, and hair that can form in the horizontal canal as a result of a reduction in epithelial migration (Fig. 6.4), the ear's natural cleaning mechanism. Where ceruminoliths become adherent to the fine hairs at the base of the horizontal canal they can cause acute discomfort leading to head shaking and tympanic membrane damage (Fig. 6.5).

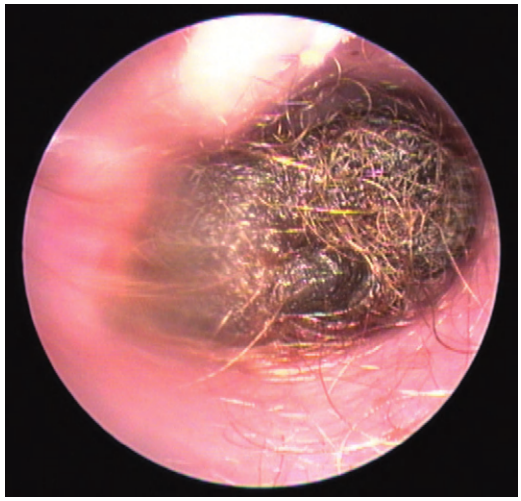


Figure 6.4 Ceruminolith formed from hair and wax impacted deep in the horizontal canal of a dog.

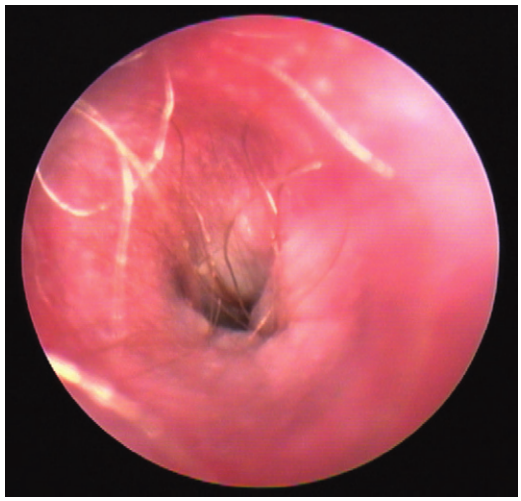


Figure 6.5 Hair penetrates the tympanic membrane and will enter into the middle ear.

Cholesteatoma

Aetiology and Pathogenesis

Cholesteatomas may be congenital or acquired and are created when there is a medial retraction of the tympanic membrane into the epitympanic recess due to negative pressure within the tympanic bulla; this may be as a result of chronic OM or abnormalities in eustachian tube function. This invagination of the keratinizing squamous epithelium of the tympanic membrane leads to the formation of a false middle ear within the epitympanic recess as the eardrum can adhere to the cochlear promontory (Fig. 6.6).

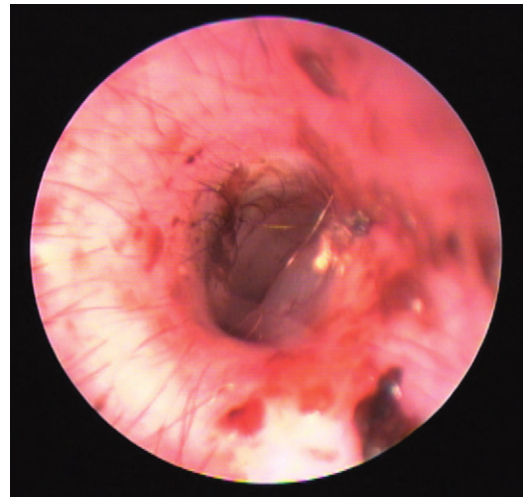


Figure 6.6 Cholesteatoma shown down the video otoscope.

Congenital lesions are most commonly recognized in young animals and are thought to occur due to abnormalities in the growth of the tympanic membrane. Acquired lesions in humans are thought to be associated with the long-term use of topical products based on propylene glycol. The same link has not been seen in dogs, but acquired lesions can occur as a result of chronic disease especially where there is marked stenosis or occlusion of the canal. Due to the slow progressive growth of the lesion it can causes severe bone changes at the contour of the tympanic bulla, including osteolysis, osteoproliferation, and osteosclerosis; expansion of the tympanic cavity; and sclerosis or osteoproliferation of the ipsilateral temporomandibular joint and paracondylar process. Cholesteatoma can cause lysis of the petrosal part of the temporal bone, leading to intracranial complications.

Clinical Signs

Dogs often have signs of chronic otitis externa; in addition there may be localized pain especially when opening the mouth to eat or bark, and a head tilt.

Key Diagnostic Tests

- Clinical signs may be suggestive.
- Radiography reveals an increased density within the middle ear.
- On CT the cholesteatoma appears as an expansile tympanic cavity mass (Fig. 6.7); this is in comparison to OM where expansion of the tympanic cavity is not seen. Other changes are as described earlier.



Figure 6.7 Cholesteotoma shown on a CT scan.

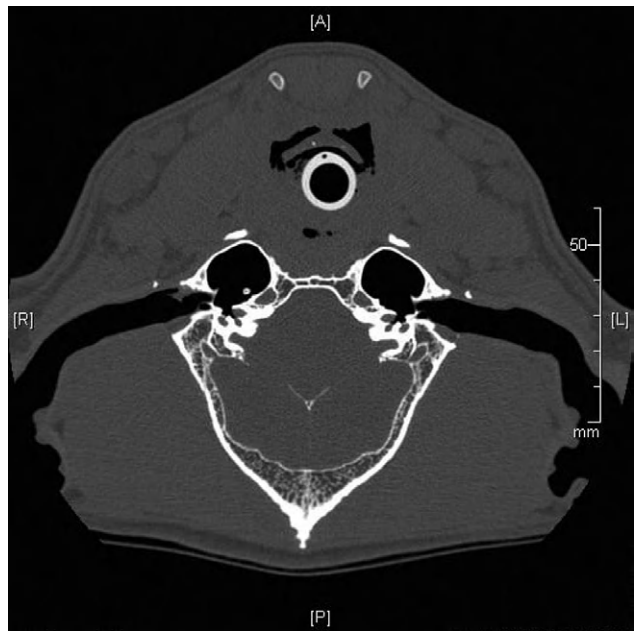


Figure 6.8 A small mineralized otolith can be seen in the right ear on this CT scan.

Otoliths

Aetiology and Pathogenesis

The origin of otoliths is unknown. They may be caused by the mineralization of necrotic debris from a previous episode of otitis externa where the disease has resolved and the eardrum has healed. They may occur de novo from disease within the middle ear. They have not been recorded in cats.

Clinical Signs

Although they have be associated with active signs of canine OM and in dogs with vestibular disease they are often present as an incidental finding in dogs, asymptomatic for ear disease, undergoing scanning for an unrelated problem. In humans otoliths are known to be a cause of labarynthitis.

Key Diagnostic Tests

- Otoliths are structures that can be identified most easily on CT scans of the middle ear in dogs; they appear as mineralized opacities (Figs 6.8, 6.9).

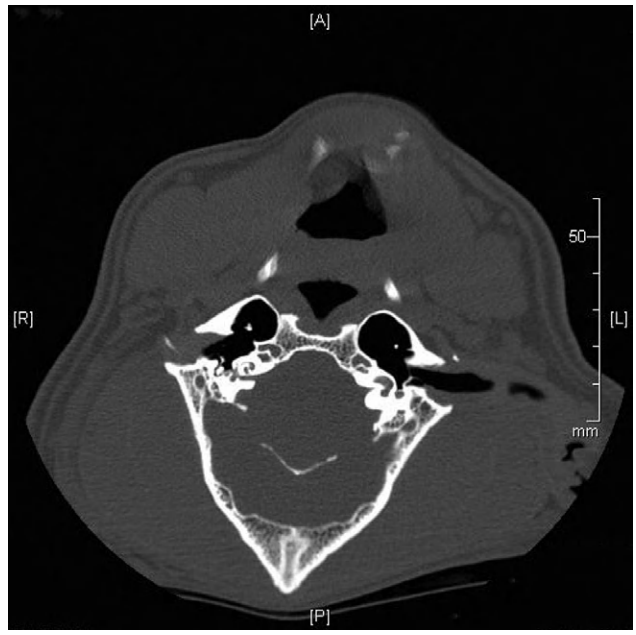


Figure 6.9 Otoliths can be seen as small mineralized deposits in both tympanic bullae. These are probably incidental findings.

6.1.2 Otitis Media Originating in the Middle Ear

Primary Secretory Otitis Media

Aetiology and Pathogenesis

Secretory abnormalities within the middle ear may be caused through overproduction of mucus within the middle ear or abnormalities in the elimination process, possibly due to failure of the mucociliary escalator and eustachian tube dysfunction. The Cavalier King Charles Spaniel is known to suffer from PSOM. Typically the middle ear contains thick tenacious mucus, 'glue ear'

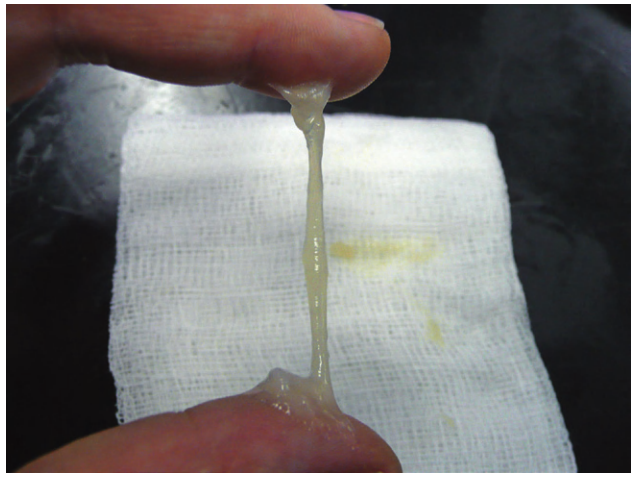


Figure 6.10 Tenacious mucus from a case of primary secretory otitis media (PSOM).



Figure 6.12 Head tilt due to PSOM in a Cavalier King Charles Spaniel.



Figure 6.11 Lack of menace response in a dog with facial nerve paralysis due to PSOM.

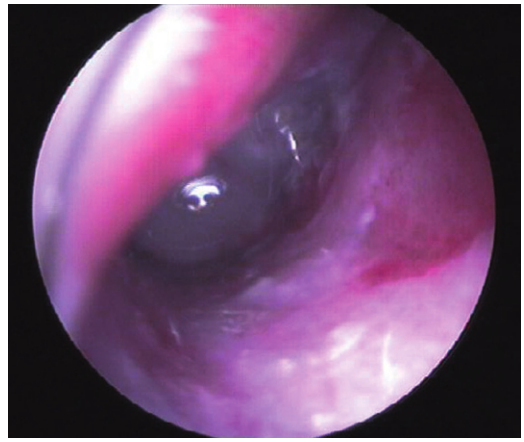


Figure 6.13 Bulging tympanic membrane in a case of PSOM.

(Fig. 6.10), which causes bulging of the intact tympanic membrane.

Clinical Signs

OM due to PSOM is unusual in that there is often no concomitant otitis externa. In contrast to other causes of OM these dogs often demonstrate neurological signs, in addition to the typical signs of OM (see Tables 6.2–6.4). These include ataxia, facial paralysis (Fig. 6.11), nystagmus, head tilt (Fig. 6.12), and deafness which is often acute in onset. Neurological disease is caused by the increase in pressure within the tympanic bulla due to mucus accumulation. Deafness is usually a conductive problem caused by mucus build-up.

Key Diagnostic Tests

- Visualization of the tympanic membrane reveals that is bulges outwards due to the presence of fluid within the middle ear (Fig. 6.13). This can be seen with either a hand-held otoscope or a video otoscope.
- When myringotomy is performed the pressure within the bulla leads to rapid loss of viscous grey–yellow mucus through the myrintogomy hole (Fig. 6.14). Diagnosis can be difficult due to the overlap of the clinical signs with other diseases, notably syringomyelia. Brainstem auditory evoked response (BAER) traces usually show a 50–60% reduction (Fig. 6.15) which usually improves once the mucus has been flushed from the middle ear.

- Radiography or CT of the tympanic bulla reveals changes consistent with a middle ear effusion with or without a concurrent bulla osteitis (Fig. 6.16).

Growths

Aetiology and Pathogenesis

Growths can be identified within the middle ear of the dog and cat. In the dog growths are a rare cause of OM, whereas

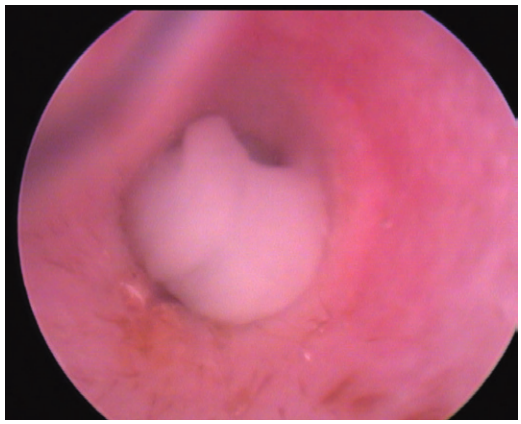


Figure 6.14 Thick tenacious mucus is released into the canal after myringotomy in a case of PSOM.

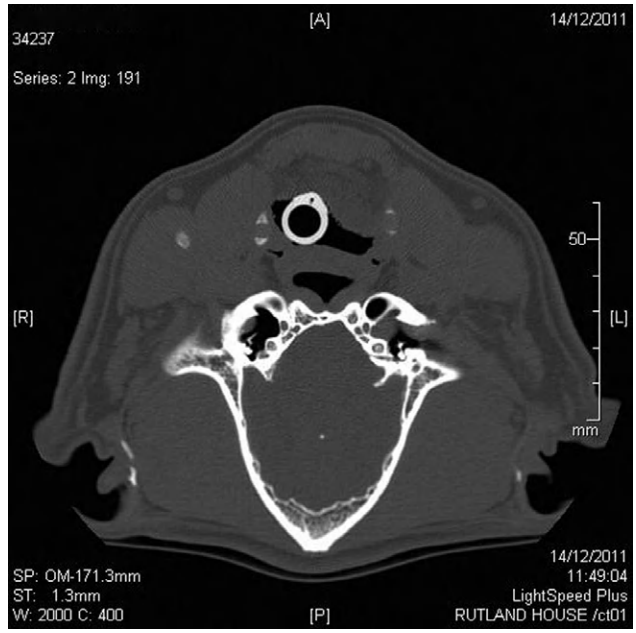


Figure 6.16 CT scan from a case PSOM showing fluid densities in the middle ear.

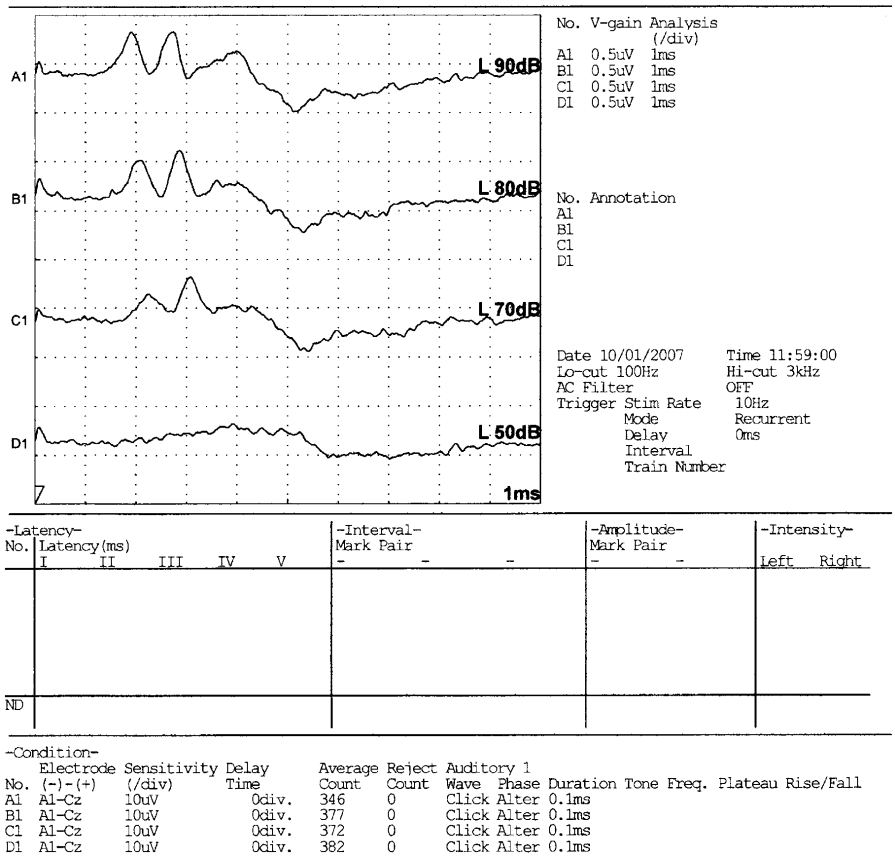


Figure 6.15 Marked hearing loss is seen due to lack of conductive hearing due to mucus retained within the middle ear.

they are very common causes of ear disease in the cat. Benign lesions in both the dog and cat are usually adenomas and fibromas. Malignant tumours of the middle ear are very rare. Adenocarcinomas and squamous cell carcinomas have been identified in both species; in addition T cell lymphoma has been recorded in the cat as a cause of OM. Nasopharyngeal polyps are the most common benign lesions to affect the middle ear of cats.

Clinical Signs

Clinical signs depend on the position and growth of the mass. OM is invariably seen; however, where the growth extends into the external ear canal it will also lead to signs of OE. Where an expansile growth puts pressure on the round and oval windows it may lead to vestibular disease.

Key Diagnostic Tests

Where the growth extends in to the external ear canal it may be visualized by otoscopy (Figs 6.17–6.20). Radiography reveals the presence of a dense soft tissue mass within the tympanic bulla in malignancy there may be signs of bulla osteitis. CT findings are predominantly signs are lysis of the contour of the tympanic bulla or the petrosal part of the temporal bone (Fig. 6.21), soft tissue swelling around the middle ear (Fig. 6.22) and marked contrast enhancement.

Nasopharyngeal Polyps

Aetiology and Pathogenesis

Nasopharyngeal polyps are benign lesions that originate within the middle ear and can extend either down the eustachian tube to the back of the throat or up through the tympanic membrane into the external ear canal. They are

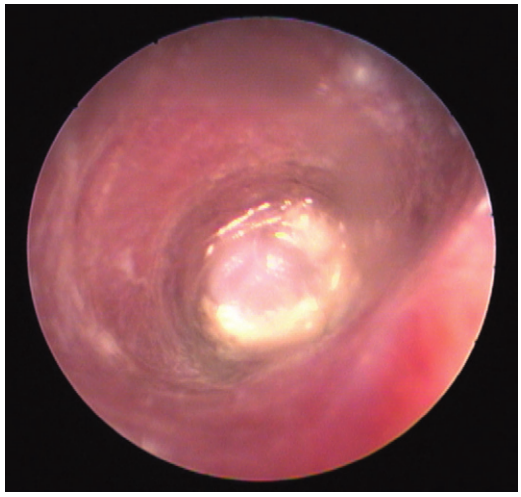


Figure 6.17 Benign growth originating from the middle ear extending into the external ear canal.

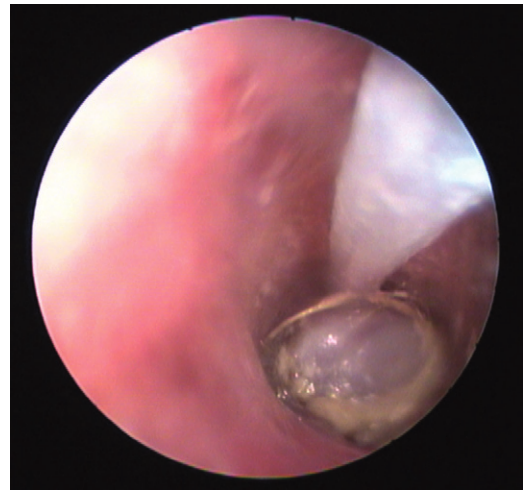


Figure 6.18 The catheter can be passed around the growth, suggesting it is not attached to the ear canal and probably originated in the middle ear.

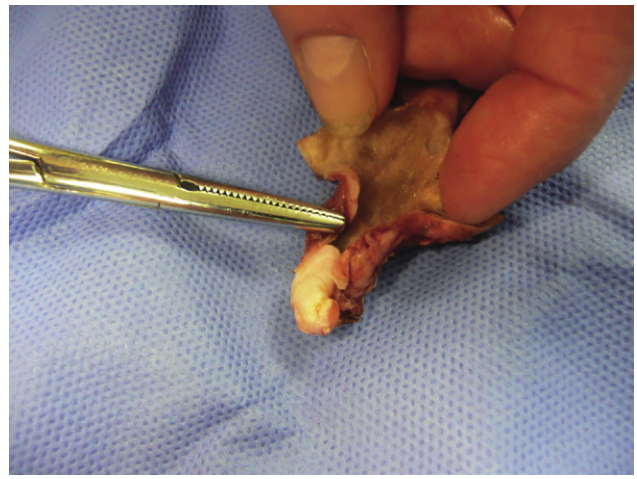


Figure 6.19 Ear canal after removal, showing the position of the growth sticking up into the canal.

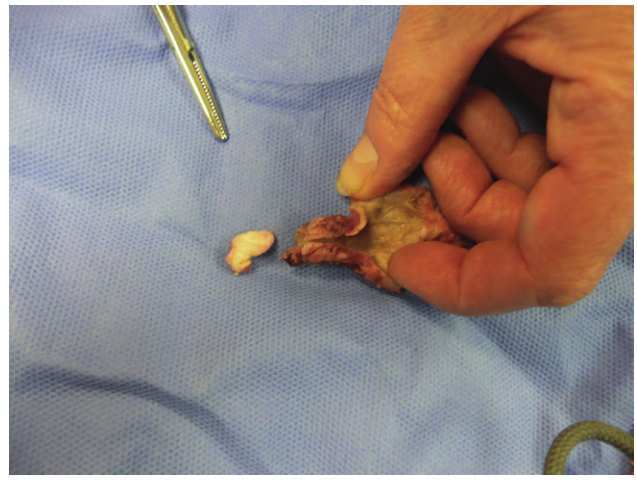


Figure 6.20 The canal showing the growth once it has been removed.

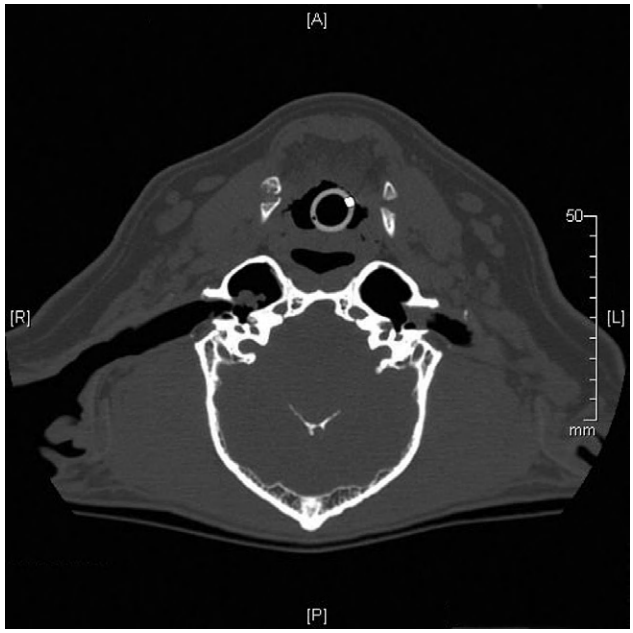


Figure 6.21 Polypoid growth in the middle ear of dog on CT scan.

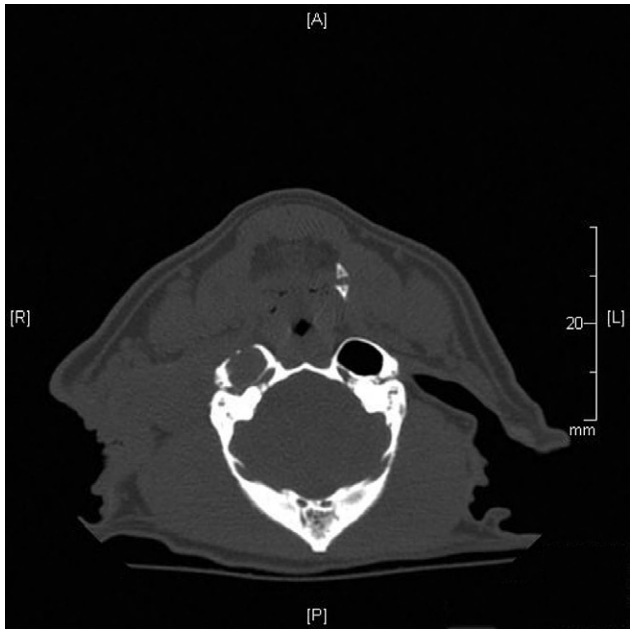


Figure 6.22 Destructive soft tissue tumour in middle ear showing loss of the tympanic bulla.

common lesions in cats and very rare in dogs. Polyps are thought to be inflammatory, although the exact cause is unknown a high proportion of cats with polyps have a previous history of respiratory disease. Polymerase chain reaction (PCR) studies have failed to identify viral particles within polyps.

Table 6.1 Clinical signs suspicious of a feline nasopharyngeal polyp

Clinical signs when the polyp extends down the eustachian tube	Clinical signs when the polyp extends up into the external ear
Nasal discharge	Head shaking
Change of voice tone	Otitis externa often with secondary infection
Coughing and sneezing	Otitis media with Horner's syndrome (Fig. 6.23)
Dysphagia	Otitis interna causing nystagmus, head tilt and incoordination (Fig. 6.24)
Respiratory stridor, dyspnoea	



Figure 6.23 Horner's syndrome in a cat (Picture courtesy of Sebastien Monclin).

Clinical Signs

See Table 6.1.

Key Diagnostic Tests

- Video otoscopy reveals a pink to red fleshy mobile mass deep in the ear canal (Figs 6.25–6.27).
- Manipulation of the polyp often leads to release of material from the middle ear.
- CT and radiography are only necessary if the mass does not protrude into the external ear canal.
- Histopathology reveals a well-vascularized fibrous tissue stroma covered with respiratory epithelium.



Figure 6.24 Head tilt and incoordination in a cat with a nasopharyngeal polyp.

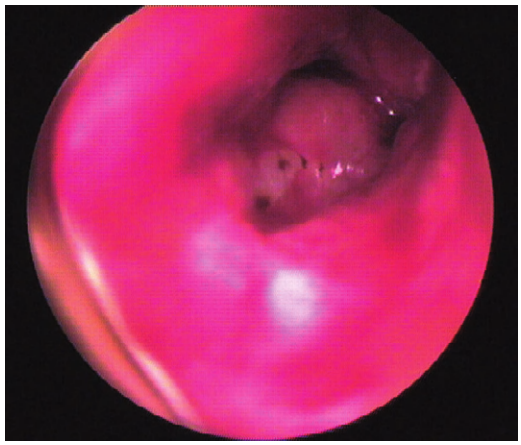


Figure 6.25 Single polyp deep within the horizontal canal.

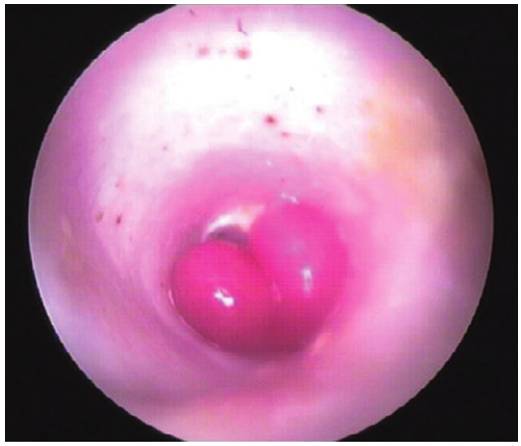


Figure 6.26 Bilobed nasopharyngeal polyp in a cat's ear.

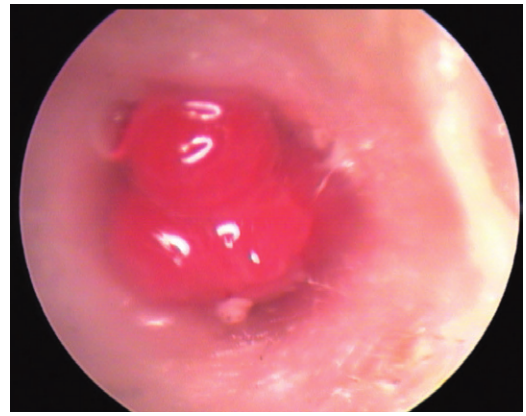


Figure 6.27 Trilobed nasopharyngeal polyp in a cat's ear.

6.1.3 Otitis Media as a Result of Ascending Infection

OM occurs in the cat as a result of ascending infection through the eustachian tube to the middle ear. The precise mechanism of infection is unknown but the presence of respiratory pathogens as part of the infectious flora in OM suggests it may occur secondary to respiratory infection in the cat. Organisms implicated in feline primary OM include streptococci and staphylococci. Mycoplasma and bordetella have also been isolated from cases but it is unclear what role these organisms play in OM.

6.2 CHRONIC OTITIS MEDIA

Chronic changes within the tympanic bulla are common sequelae of long-standing OM in the dog and cat. In acute disease fluid can be identified on CT scans. In chronic disease fluid may still be present but more often granulation tissue is found within the tympanic bulla (Fig. 6.28). Inflammation within the tympanic bulla causes the lining of the bulla called the mucoperiosteum to change from cuboidal to pseudostratified columnar ciliated epithelium. The lamina propria thickens in response to ongoing inflammation and oedema and granulation tissue forms (Fig. 6.29). Further change leads to the formation of dense connective tissue with bone spicules within it (Figs 6.30, 6.31).

6.3 INVESTIGATION OF OTITIS MEDIA

6.3.1 History and Physical Examination

A carefully taken history and physical examination can help establish a diagnosis of OM before the ear itself is examined (Tables 6.2–6.4).

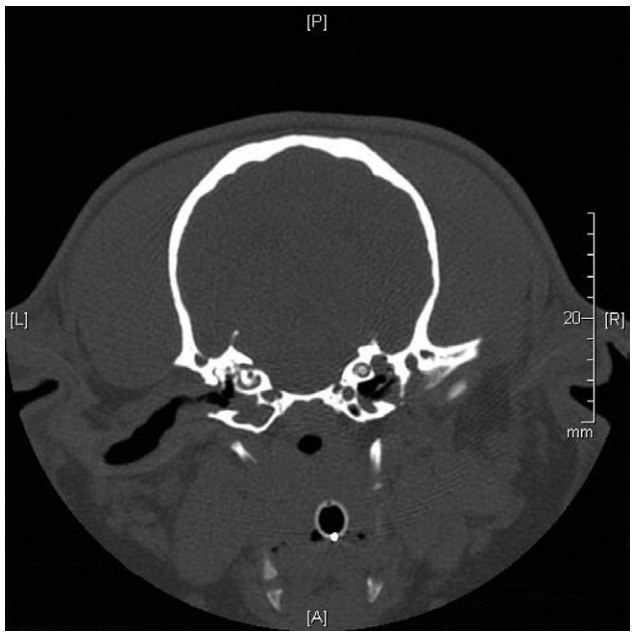


Figure 6.28 Mild granulation tissue formation in the middle ear of a case of otitis media.

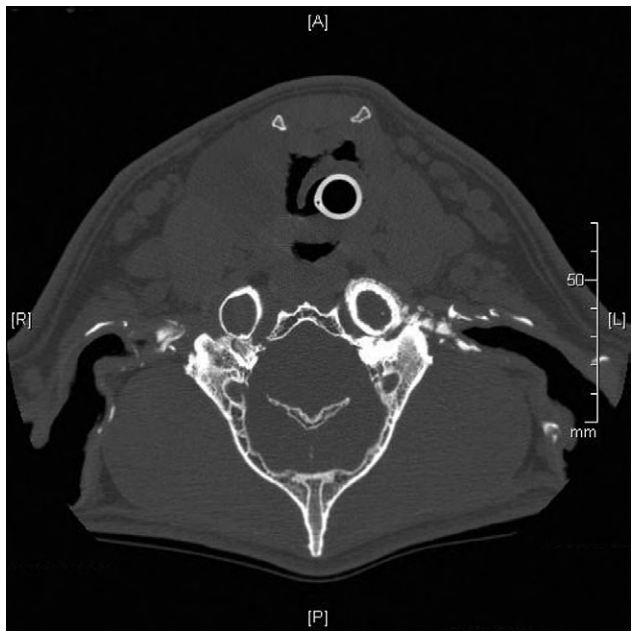


Figure 6.30 Advanced otitis media showing thickening of the tympanic bullae which is particularly severe on the left-hand side. This is accompanied by marked calcification of the ear canals bilaterally.

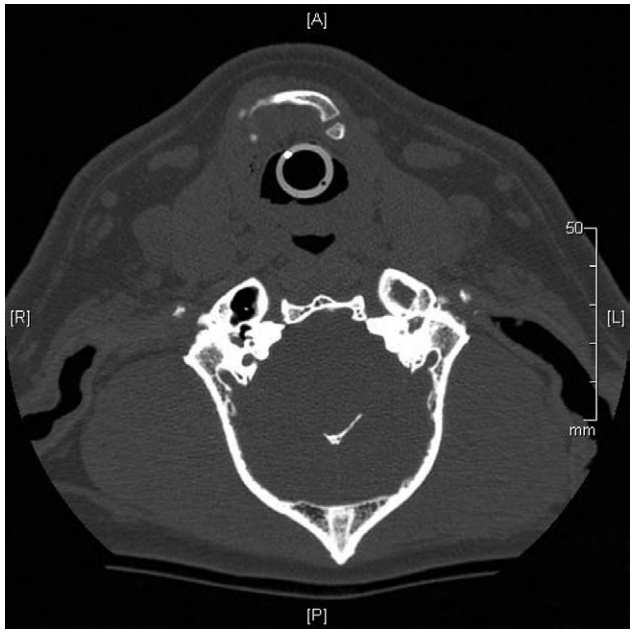


Figure 6.29 Marked granulation tissue formation in the left middle ear combined with formation of granulation tissue and loss of the lumen of the canal. Note otolith in tympanic bulla on right side.



Figure 6.31 Severe bony change in the tympanic bulla on the left-hand side in a chronic case of otitis media.

Table 6.2 Historical information and clinical signs suspicious of otitis media. In some cases signs of otitis interna may occur concurrently, e.g. in vestibular disease (see Chapter 7)

History	Clinical signs
Reluctance to open mouth, catch a ball, chew hard food, yawn, or bark	Pain on opening of jaw
Hearing problems	Pain when ear pinna is gently stretched
Animal losing food out of the side of its mouth when eating	Sympathetic nerve damage – Horner's syndrome (commonly cat) (see Table 6.3)
Dog manifests signs of pain by whining, holding head to one side or rubbing the face along the floor or on furniture or scratches at ears	Facial nerve damage (commonly dog) (see Table 6.4)

Table 6.3 Sympathetic nerves and evidence of damage within the middle ear

Sympathetic nerve components within the middle ear	Observed clinical signs associated with damage
Sympathetic postganglionic nerves to the eye and orbit	Horner's syndrome Anisocoria with ipsilateral miosis Ptosis upper eyelid Narrowing of the palpebral fissure Enophthalmos Protrusion of the third eyelid (Fig. 6.32)

Table 6.4 Facial nerve components and evidence of damage within the middle ear

Facial nerve components within the middle ear	Observed clinical signs associated with damage
Motor innervations to superficial muscles of head, neck, face, and external ear	Ear and lip droop (ipsilateral side) Asymmetrical commissure of mouth Wide palpebral fissure (ipsilateral side) Ipsilateral sialosis Reduced and eventual loss of corneal, palpebral, and menace response (Fig. 6.33)
Sensory neurons from taste buds and other visceral receptors from soft palate, nasopharynx, and nasal cavity	None
Preganglionic parasympathetic fibres that synapse and send ganglia to lacrimal gland, glands of nasal mucosa, salivary glands, buccal and lingual mucosa	Loss of innervation to lacrimal gland leads to keratoconjunctivitis sicca (ipsilateral side) Loss of innervation to glands of nasal mucosa leads to a dry, hyperkeratotic nasal mucosa (ipsilateral side; Fig. 6.34)



Figure 6.32 Bilateral Horner's syndrome in a cat (Picture courtesy of Sebastien Monclin).



Figure 6.33 Facial nerve paralysis in a dog showing loss of motor innervation.

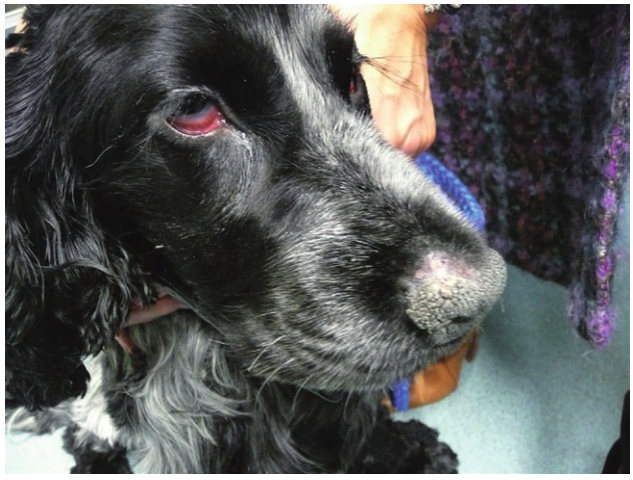


Figure 6.34 Facial nerve damage in a dog due to middle ear disease showing parasympathetic abnormalities in the form of a dry crusted nose and keratoconjunctivitis sicca.

6.3.2 Assessment of the Tympanic Membrane

Video Otoscopy

Careful assessment of the tympanic membrane is important as a diagnostic step in OM to decide if it is normal, ruptured, or has healed after a previous episode of infection. Where an intact but abnormal tympanic membrane is present myringotomy is useful to obtain samples from the middle ear; or advanced diagnostic imaging techniques such as CT or MRI may be necessary.

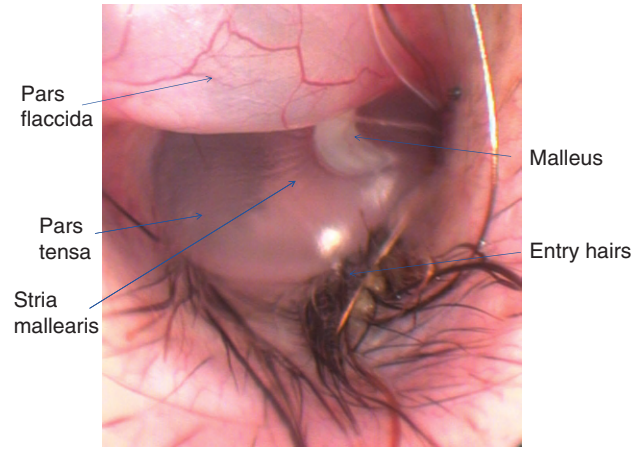


Figure 6.35 A normal tympanic membrane showing labels of appropriate structures.

Intact Normal Tympanic Membrane

When the tympanic membrane is intact and appears to be normal (Fig. 6.35), OM is an unlikely finding. When damage has occurred to the tympanic membrane much of the original structure of the pars tensa is lost, especially the fine stria mallaris.

Intact Abnormal Tympanic Membrane

When the tympanic membrane is intact but appears to be abnormal the clinician should be suspicious of a previous episode of OM. The tympanic membrane can heal in the face of infection. As many as 70% of cases of OM may have an intact tympanic membrane. Common abnormalities of the tympanic membrane include opacity of the pars tensa with loss of the normal striations (Fig 6.36) In some cases material such as discharge, debris, and hair can be visualized behind an intact membrane.

Where fluid is present within the bulla in such diseases as primary secretory OM the tympanic membrane will bulge outwards (Fig. 6.37). Where negative pressure exists within the bulla the tympanic membrane may be sucked into the middle ear and can become attached to the cochlear promontory creating a cholesteatoma (Fig. 6.38).

When the tympanic membrane is intact but is abnormal myringotomy should be performed. This is technically difficult to achieve safely without the use of a video otoscope.

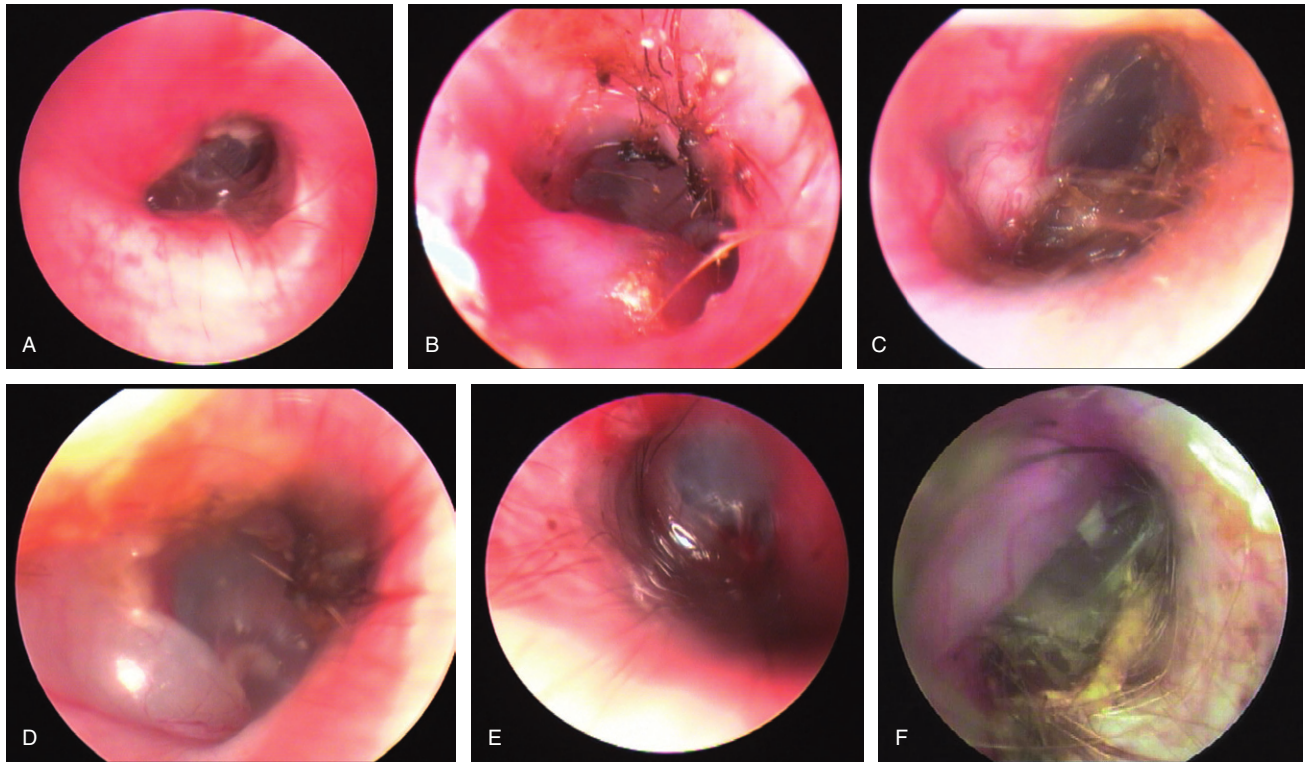


Figure 6.36 Abnormal tympanic membranes (**A–F**).

TECHNIQUE FOR MYRINGOTOMY

- The ear canal should be cleaned using warm water to remove debris.
- A 6 or 8 French gauge urinary catheter should be cut at the end to make a point and introduced down the ear canal (Fig. 6.39).
- The pointed end of the catheter can be gently pushed through the pars tensa at the five or seven o'clock positions. It should be angled downwards and advanced into the tympanic bulla (Figs 6.40–6.43).
- A clean 2 mL syringe can be attached to the catheter and gently aspirated. If fluid is obtained it may be submitted for cytology and culture.
- If dry tap is found then 1 mL of sterile water can be introduced down the catheter and then aspirated back and submitted.

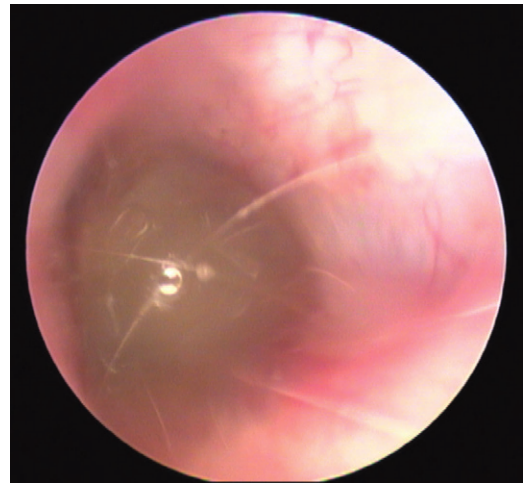


Figure 6.37 Tympanic membrane is intact but bulging due to the presence of fluid within the middle ear.

Ruptured Tympanic Membrane

Accurate visualization of the tympanic membrane requires good illumination and high magnification. Small tears of the tympanum can be missed when the area is assessed using a hand-held otoscope (Fig. 6.44), but more extensive damage can be seen using only simple equipment (Fig. 6.45). When the tympanic bulla is ruptured, the area beyond the end of the horizontal canal can be visualized. A small clump of short brown–black hairs is found in most dogs on the floor at the end of the horizontal canal just in front of the tympanic membrane. These small structures, often referred to as 'entry hairs' (Fig. 6.46), can help orientate the clinician within the canal when the tympanic membrane has been completely destroyed. When the

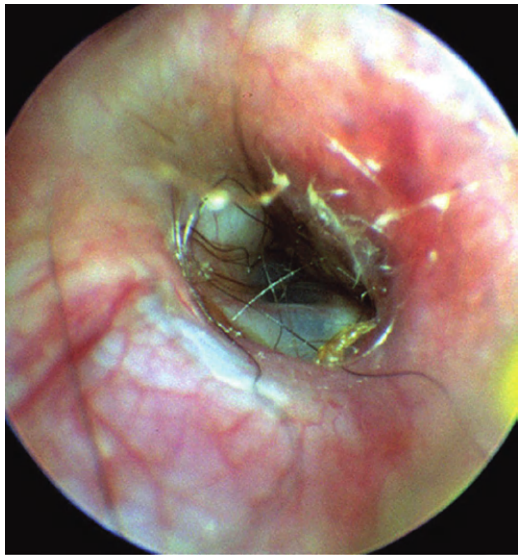


Figure 6.38 Tympanic membrane has been pulled into the tympanic bulla due to negative pressure.

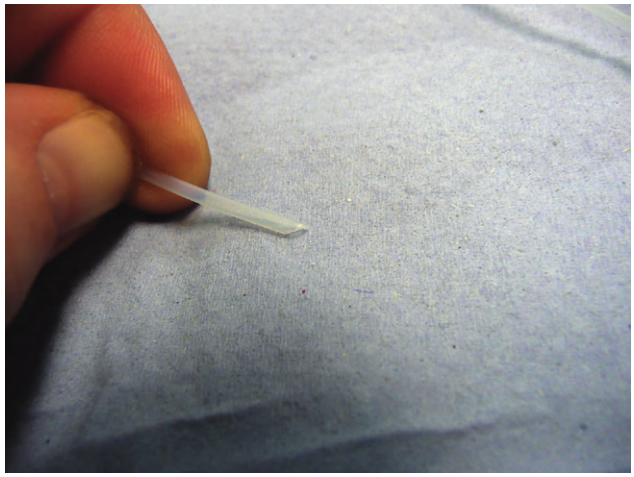


Figure 6.39 Catheter cut to a point to facilitate myringotomy.

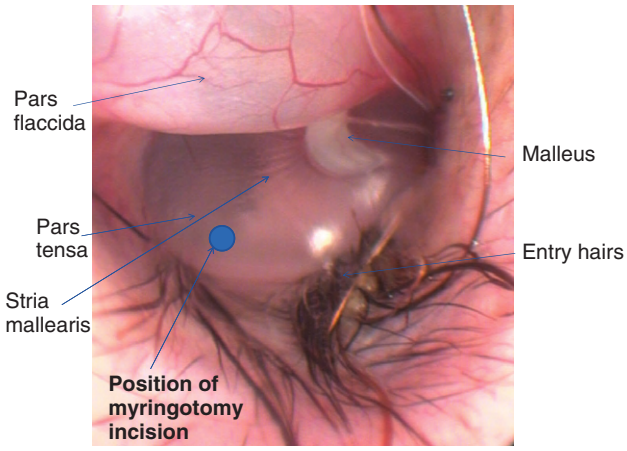


Figure 6.40 Normal tympanic membrane showing position of myringotomy.

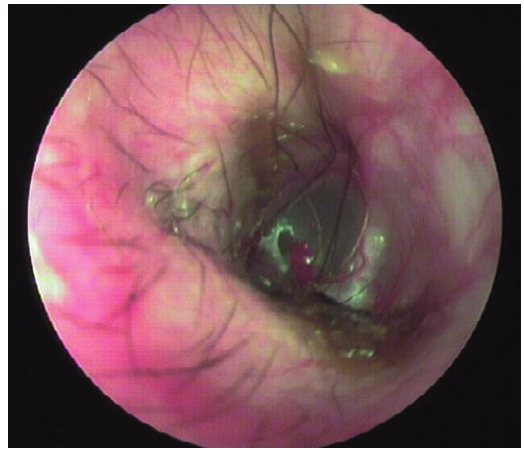


Figure 6.41 Myringotomy hole made in the pars tensa of the eardrum of a dog.

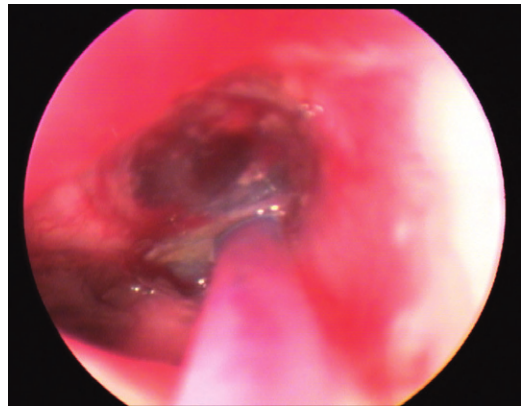


Figure 6.42 Catheter introduced into the ear in order to perforate the pars tensa.

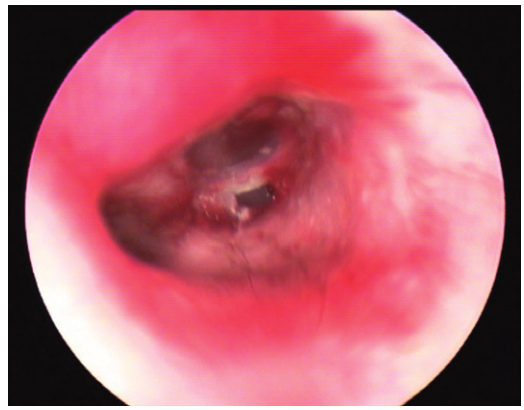


Figure 6.43 Myringotomy hole in pars tensa. Note the tympanic membrane is grossly abnormal.

tympanic membrane has been lost, structures such as the cochlear promontory can be viewed down a video otoscope: it normally appears as a glistening white structure with a jagged edge along its lower border (Fig. 6.47). When damaged the promontory can look irregular and covered with discharge (Fig. 6.48) or show signs of haemorrhage, which with poor lighting can be mistaken for an intact tympanic membrane (Figs 6.49, 6.50). On some occasions material can be observed within the middle ear.

Other Methods to Assess Patency of Tympanic Membrane

- Visual assessment with hand-held ototoscope can be undertaken by filling the canal with warm saline and looking for air bubbles rising as the animal breathes.
- Gentle probing of the tympanum can be performed using a small-diameter catheter introduced into the canal and used to probe the drum. Where the probe bounces off a spongy surface it is likely the eardrum is intact;

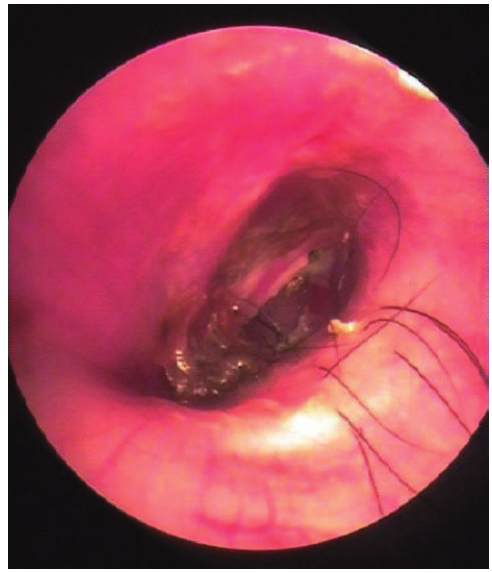


Figure 6.46 Small brown bristly entry hairs may be seen at the end of the horizontal canal and can act as a landmark as to the position of a normal tympanic membrane.

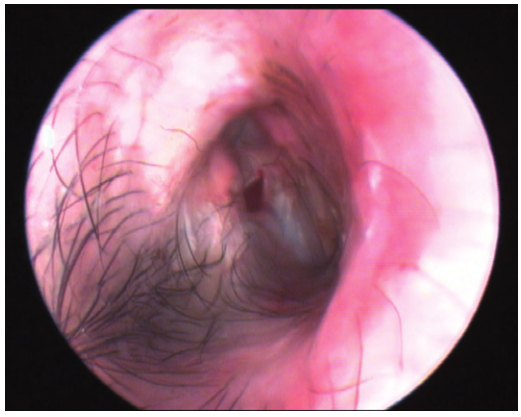


Figure 6.44 Small tear in tympanic membrane may not be visible down a hand-held otoscope.

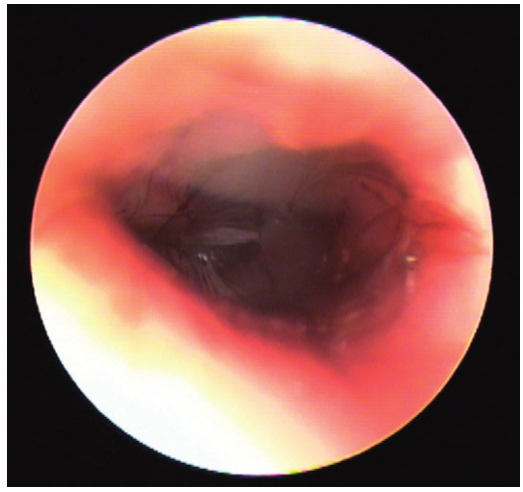


Figure 6.47 The white shiny cochlear promontory can be seen behind the normal tympanic membrane.

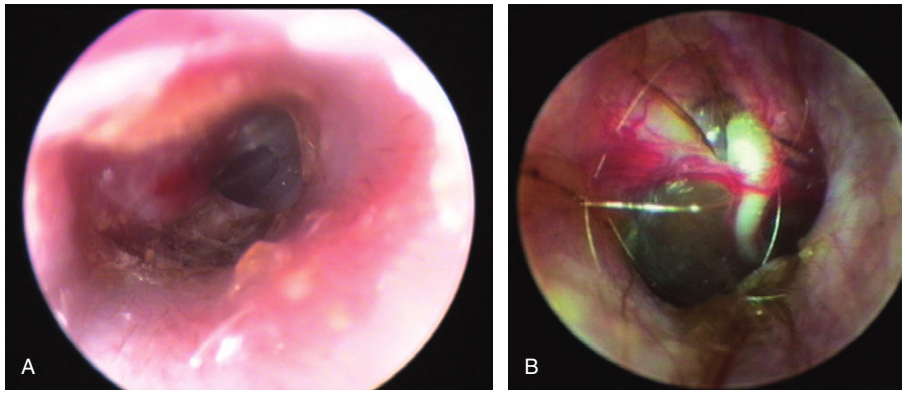


Figure 6.45 Large tears in tympanic membrane are visible with both a hand-held otoscope and a video otoscope (**A, B**).

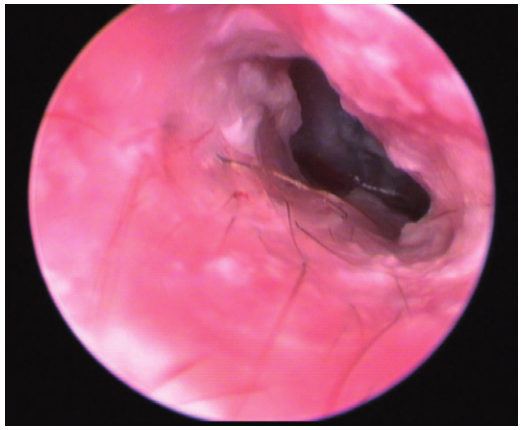


Figure 6.48 The cochlear promontory becomes irregular and can be covered with discharge when infection extends into the middle ear.

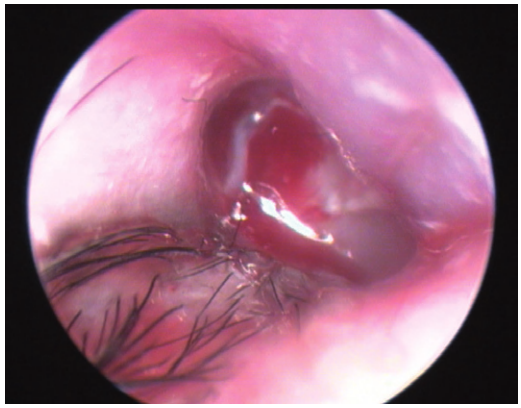


Figure 6.49 Haemorrhage on the cochlear promontory can give the impression of a normal tympanic membrane.

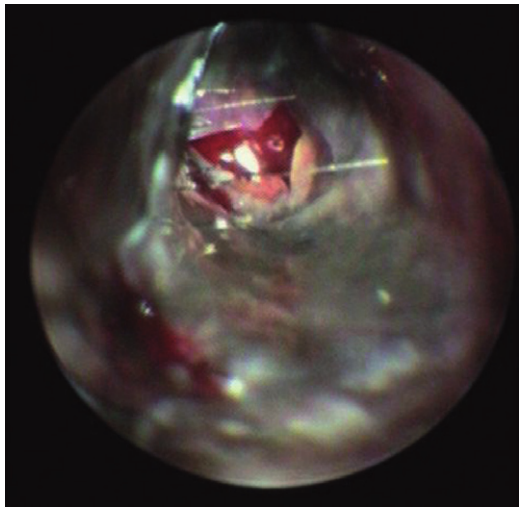


Figure 6.50 Haemorrhage on the cochlear promontory.

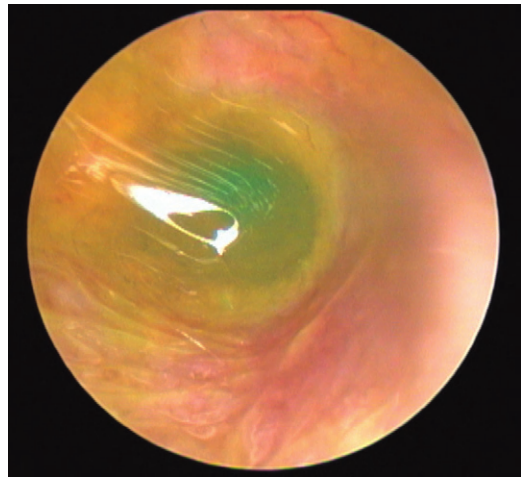


Figure 6.51 Dilute fluorescein stain in the ear can be used to assess the patency of the tympanic membrane.

where it bounces off a hard surface it is more likely to be hitting the cochlear promontory, suggesting the ear is ruptured.

- Instillation of dye, either warm dilute povidone iodine or dilute fluorescein (Fig. 6.51), into the ear canal. If fluid can be visualized at the back of the throat or down the nose it suggests that the tympanic membrane is ruptured.
- Positive contrast canalography, auditory tympanometry, or CT/MRI (see Chapter 3 for details).

6.3.3 Assessment of the Middle Ear

Palpation

Palpation of the middle ear can be undertaken using a urinary catheter. This may be passed into the tympanic bulla and bounced off the wall of the bulla. If the bulla is chronically damaged and contains granulation tissue the catheter bounces off a spongy surface, which feels different from the hard surface within the undamaged bulla that is lined by the mucoperiosteum.

Radiographic Assessment

See Chapter 3 for techniques for radiographic positioning. In dogs with minimal bony change the bullae appear as normal thin circular osseous structures. The cortical outline should be thin and the middle of the bullae should be radiolucent due to their air content. In chronic disease bone within the bulla may show proliferative or lytic change. Lytic changes are usually associated with malignant neoplasia or osteomyelitis. Where the bullae appear radio-

VetBooks.ir

paque it is usually an indication of exudate or a soft tissue mass (polyp, neoplasia) within the bulla.

CT Examination

A CT scan of the middle ear is useful in differentiating bony lesions within the bulla from soft tissue lesions (see Chapter 3).

MRI

Although MRI can be used to examine the middle ear, it is more useful in investigating inner ear problems (see Chapter 3).

VetBooks.ir

VetBooks.ir

CHAPTER 7
DISEASES OF THE INNER EAR

Karen Tobias

7.1 Vestibular Apparatus 125 7.2 Cochlea 136

The inner ear of the dog and cat is contained within the petrosal portion of the temporal bone and includes the cochlea and the vestibular apparatus. Diseases of the inner ear therefore may result in abnormalities in hearing or balance.

7.1 VESTIBULAR APPARATUS

The vestibular apparatus is critical for activating unconscious reactions to movement and gravity. In an effort to maintain balance, input on head position and linear, rotational, and angular acceleration is collected from receptors in the sacculus, utriculus, and semicircular ducts (Fig. 7.1). This input is transmitted by the vestibular portion of the vestibulocochlear nerve (CN VIII) to vestibular nuclei within the medulla oblongata and pons, directly adjacent to the fourth ventricle. A few fibres from the vestibular nerve bypass the vestibular nuclei, extending directly to the cerebellum.

Projections from the vestibular nuclei extend to the brainstem, spinal cord, and cerebellum. Within the brainstem, some axons terminate in the motor nuclei of the oculomotor (CN III), trochlear (CN IV), and abducent (CN VI) nerves, which control reflex eye movement. Axons also reach the emetic centre of the reticular formation. Axons passing through the spinal cord within the vestibulospinal tract extend to neck, trunk, and leg muscles. Stimulation of neuronal cell bodies of these axons increases tone in extensor muscles on the ipsilateral side and inhibits tone of flexor muscles on the ipsilateral side and extensor muscles of the contralateral side. Projections to the cerebellum allow coordination of eye, neck, trunk, and limb position with head position. Additionally, pathways from the vestibular nuclei to the cerebral cortex allow mediation in response to conscious perception of movement and gravity.

7.1.1 Vestibular Disorders

Vestibular dysfunction is classified by location of the disease. Peripheral vestibular disease is caused by abnormalities of inner ear components, specifically those of the membranous labyrinth. Central vestibular disease is caused by abnormalities primarily within the brainstem and cerebellum.

Clinical Signs

Common clinical signs of vestibular disease included nystagmus, strabismus, head tilt, and abnormalities in balance and locomotion such as ataxia, circling, falling, or rolling. Other clinical signs may include postural deficits, vomiting, behavioural changes, Horner's syndrome, facial nerve paralysis, and other cranial nerve abnormalities. Characterization of these signs may help to differentiate peripheral and central vestibular disorders (Table 7.1).

Nystagmus

Nystagmus is defined as an involuntary, rhythmic movement of the eyes. In healthy animals, *physiological nystagmus*, which is also known as the *vestibulo-ocular or oculocephalic reflex*, helps to stabilize visual images on the retina. As their heads move, normal cats and dogs will try to fix their gaze on an object, 'following' it with their eyes until they can no longer see it (Fig. 7.2). This results in a slow, gradual movement of the eyes toward the object as the head turns away, producing a 'slow phase' of normal

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

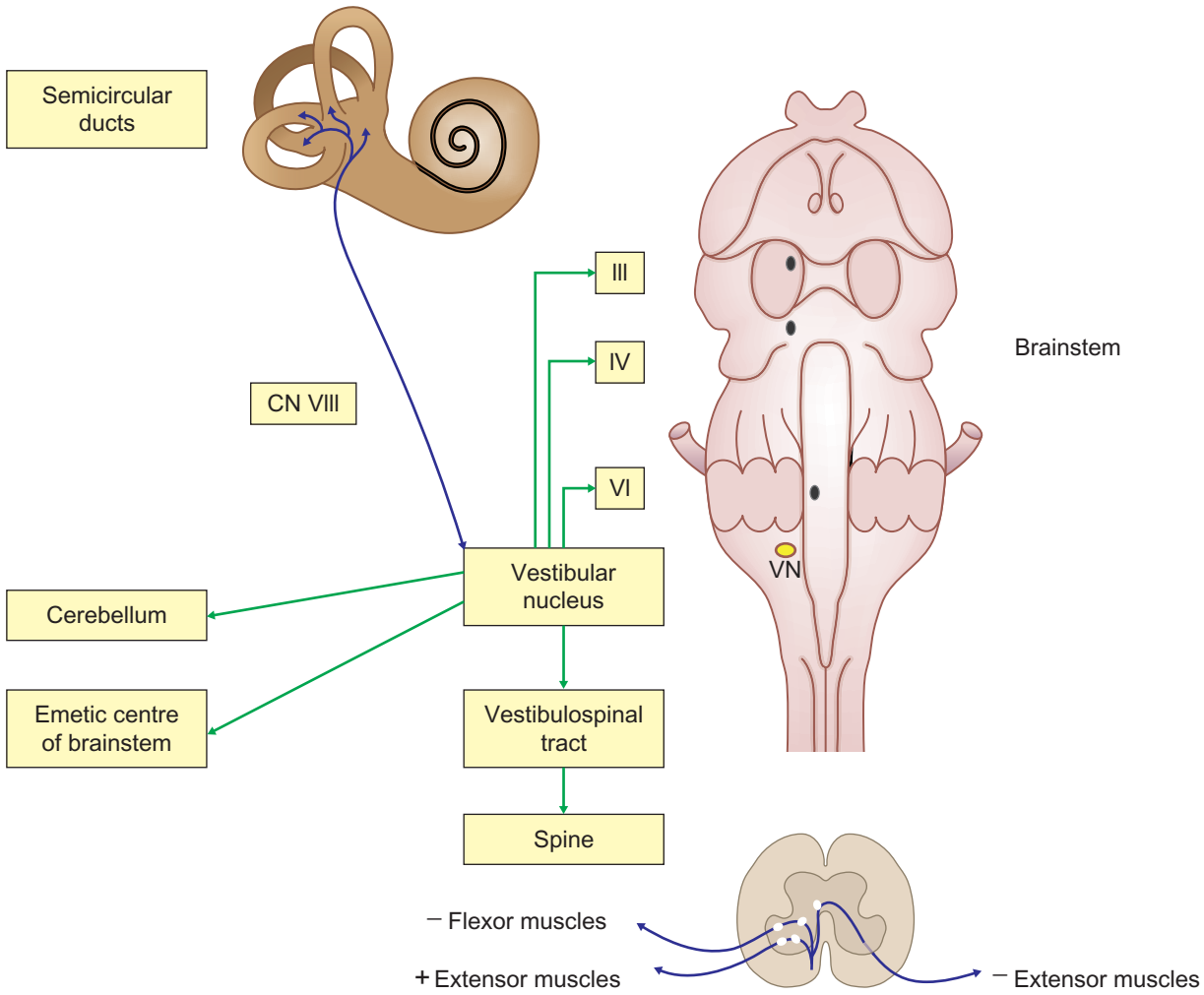


Figure 7.1 The vestibular pathway.

eye movement opposite the direction of head movement. Once extraocular muscles reach their stretch threshold they contract, and the eyes move rapidly toward the direction of the head movement, producing a 'fast phase'. Normal animals should only have nystagmus when the head is moved. If it is present when the head is in a normal, neutral position; a fully extended, resting position; or a flexed, lateral position, the nystagmus is considered pathological.

Pathological nystagmus, or abnormal movement of the eyes, is commonly seen in dogs and cats with central or peripheral vestibular disorders (Fig. 7.3). Pathological nystagmus associated with vestibular disease can be horizontal, vertical, rotary, or positional. *Positional nystagmus* occurs only when the animal's head is placed in an unusual position (e.g. flexed laterally or extended fully). Animals with vestibular disease can also have abnormal (delayed or absent) physiological nystagmus. Nystagmus is described by the direction of the fast phase; for example, an animal with a fast phase in a horizontal plane to the left would have a left horizontal nystagmus.

Nystagmus in animals with peripheral vestibular disease is usually rotary or horizontal, although rotary nystagmus may be difficult to distinguish from vertical in some animals. Nystagmus with peripheral vestibular disease does not change direction with the animal's head position, and its fast phase is always away from the side of the lesion. In animals with chronic peripheral vestibular disease, nystagmus may be initiated by a sudden change in position, such as with rolling the animal on its back (Fig. 7.4), but the direction of nystagmus does not vary. Animals with peripheral vestibular disease may have delayed or absent physiological nystagmus.

Animals with central vestibular disease can have vertical, rotary, or horizontal nystagmus, and the nystagmus may change in character or direction with change in head position. In animals with bilateral peripheral or central disease, nystagmus is absent.

Table 7.1 Vestibular disease: lesion location based on clinical signs

Clinical signs	Peripheral vestibular disease	Central vestibular disease
Nystagmus	+	+
Abnormal physiological	+	+
Horizontal	+	+
Rotary	+	+
Directional variation with head position	–	+
Strabismus	+	+
Head tilt	+	+
Ataxia	+	+
Circling	+	+
Abnormal postural reactions	–	+
Proprioceptive deficits	–	+
Loss of limb strength	–	+
Cranial nerve dysfunction (except VII and VIII)	–	+
Horner's syndrome	+	(rare)
Altered mentation	–	+
Disorientation	+	+
Deafness	+	(rare)

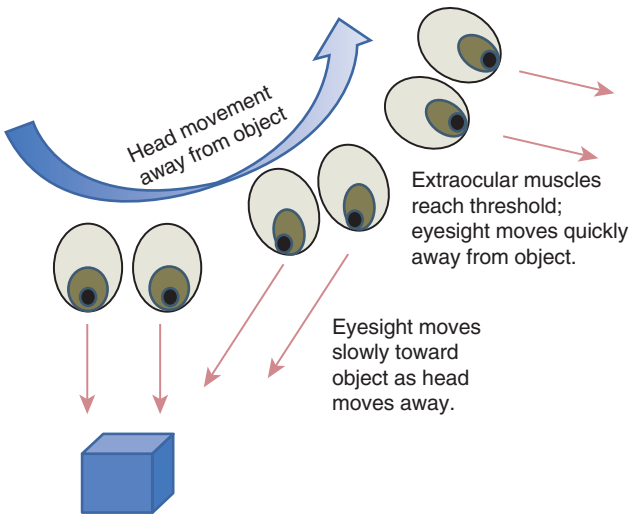


Figure 7.2 Physiological nystagmus.

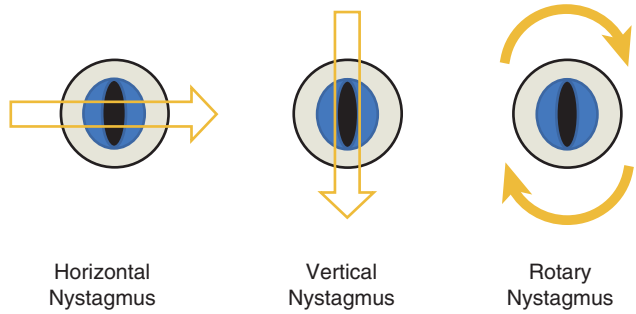


Figure 7.3 Eye movements seen with pathologic nystagmus.



Figure 7.4 In animals with chronic vestibular disease, pathologic nystagmus may be initiated by a sudden change in position. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

Strabismus

Strabismus is an abnormal position of the eye in relation to the palpebral fissure or orbit. Strabismus that is consistent with all positions of the head is indicative of abnormalities in the nerves or extraocular muscles that control eye movement (Fig. 7.5). *Vestibular strabismus* is an inconsistent abnormality in eye position. Affected animals have ventral or ventrolateral eye position that appears or worsens when the head and neck are extended but disappears with changes in head position, as long as their extraocular muscles are functional. The lesion is usually located on the same side as the strabismus.

Head Tilt

A head tilt is categorized by the side of the head on which the ear is directed more ventrally (Fig. 7.6). Head tilts in animals with peripheral vestibular disease are typically directed toward the side of the lesion. Head tilts associated with central disease are also usually ipsilateral to the lesion;



Figure 7.5 In this dog strabismus was consistent with all positions of the head, indicating a problem with extraocular muscles or nerves that control eye movement.



Figure 7.7 Head tilt to the left and ipsilateral facial nerve paralysis in a Pug with otitis interna secondary to otitis externa/media.



Figure 7.6 With peripheral vestibular syndrome, head tilt and ataxia are toward the side of the lesion. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

however, contralateral (paradoxical) head tilt has been reported.

Abnormalities in Balance and Locomotion

These abnormalities may manifest as ataxia, circling, rolling, or an inability to stand. *Ataxia* and *circling* (tight circles) are seen with central and peripheral vestibular disease. Animals with either type of vestibular disease can also have rolling; if it persists longer than 48 hours, however, it is more likely to be associated with a central lesion. As with nystagmus and head tilt, ataxia and circling are usually toward the side of the lesion, with the exception of a few cerebellar diseases. Animals with *bilateral vestibular dysfunction* have a wide-based, crouched stance; large, side-to-side head excursions; no head tilt; and no normal or abnormal nystagmus. These animals usually have peripheral vestibular lesions.

Other Clinical Signs of Vestibular Disease

Abnormal postural reactions are only seen in animal with central vestibular dysfunction. Postural reactions such as hopping and hemi-walking can be difficult to evaluate in animals with peripheral disease because of their loss in equilibrium; therefore, evaluation of proprioceptive positioning is important in determining the location of the lesion. *Proprioceptive deficits* are only seen with central vestibular disease and always occur on the side of the lesion. Opisthotonus, hemiparesis, and tetraparesis have been reported with central vestibular disease but are not seen with peripheral vestibular disease.

Dysfunction of cranial nerves other than the facial and vestibulocochlear nerves is only seen with central vestibular disease. Abnormalities associated with trigeminal (CN V) and abducent nerve dysfunction are most often reported. Facial nerve paralysis is more commonly associated with peripheral vestibular dysfunction secondary to otitis media interna (Fig. 7.7). The facial nerve passes through a cleft in the tympanic bulla and is therefore exposed to any middle ear disease. Clinical signs of facial nerve dysfunction include drooling, facial muscle paresis or paralysis, loss

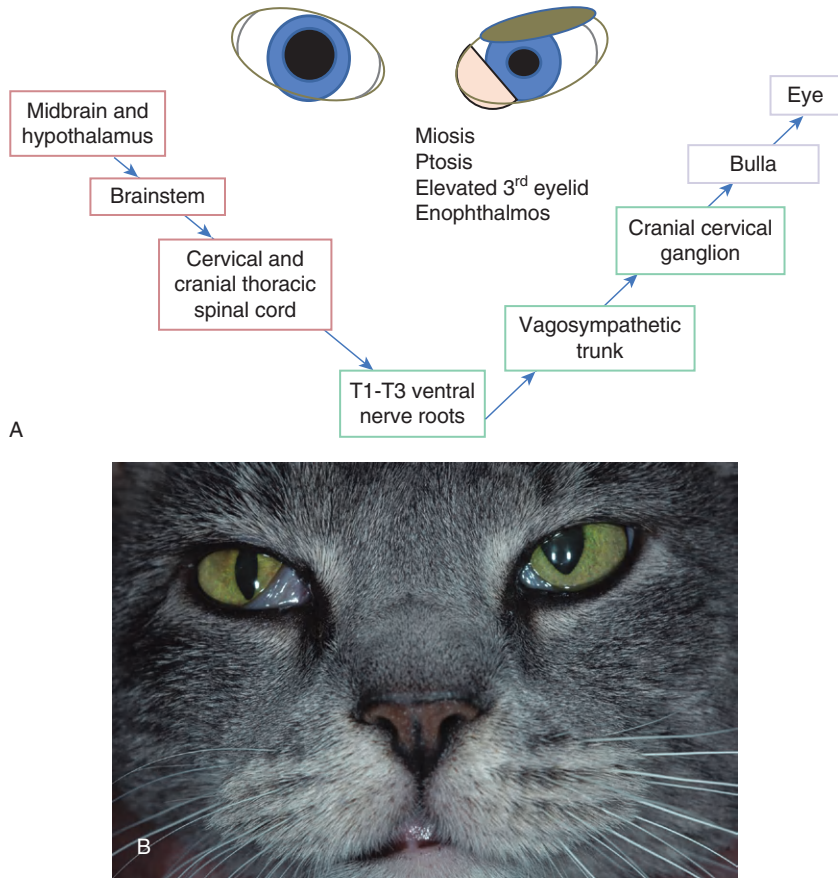


Figure 7.8 Horner's syndrome can be caused by lesions anywhere along the sympathetic pathway (**A**), including in the brain, brainstem, spinal cord, T1–T3 nerve roots, vagosympathetic trunk, base of the skull, middle ear, or retrobulbar region. Classic signs (**B**) include pupil constriction, drooping upper lid, elevated third eyelid, and retraction of the globe into the orbit.

of blink, and those associated with keratoconjunctivitis sicca (KCS).

Altered mentation is usually only seen in animals with central vestibular disease. This may be difficult to differentiate from disorientation, which is often seen with peripheral vestibular disease. Cats with otitis media may also appear obtunded because of pain.

Deafness is rare in animals with central disease. In animals with vestibular dysfunction, deafness is usually a result of abnormalities in sound wave conduction caused by conditions such as debris in the ear, ruptured tympanum, damage to auditory ossicles, accumulation of fluid, or neoplasia.

Other clinical signs of vestibular disease are associated with the primary aetiology and its location. For instance, animals with cerebellar disease may also have dysmetria and intention tremors, while animals with vestibular disease secondary to otitis media interna may have pain upon opening the mouth from inflammation around the temporomandibular joint, aural discharge secondary to otitis externa, or dyspnea or dysphagia secondary to a nasopharyngeal polyp.

Horner's syndrome includes miosis, ptosis, enophthalmos, and elevation of the third eyelid (Fig. 7.8). In animals with peripheral vestibular dysfunction, Horner's syndrome is often a result of otitis media from damage to sympathetic fibres as they course through the tympanic cavity. Because of their exposed position, these fibres are more easily damaged in cats, particularly during bulla osteotomy or traction polyp removal. Horner's syndrome is rarely associated with central vestibular disease.

7.1.2 Peripheral Vestibular Dysfunction

Aetiologies

Aetiologies of peripheral vestibular dysfunction include septic or aseptic otitis media interna (Fig. 7.9), inflammatory polyps, neoplasia, trauma, ototoxicity, generalized



Figure 7.9 Transverse CT images. Otitis interna in a 10 year old male castrated Pug with chronic otitis media and left-sided head tilt. Before the CT scan, the dog presented acutely for ataxia, falling to the left, and horizontal nystagmus with the fast phase to the right. The left bulla was enlarged with portions of the wall either thin or absent (**A**). Contrast enhancement was noted along the meninges and in the tissues around the bulla (**B**). A sterile cholesteatoma and meningitis were found on necropsy. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

polyneuropathy, hypothyroidism, and congenital dysfunction. If these aetiologies are ruled out, the condition is classified as idiopathic. Idiopathic vestibular syndrome and progression of otitis media to otitis interna, or labyrinthitis, are the most common causes of peripheral vestibular dysfunction. Besides infection, *otitis media* can have a variety of causes. These are discussed more thoroughly in Chapter 6.

In dogs, *bacterial otitis media interna* is usually secondary to ascending otitis externa. The most common bacteria are staphylococcus, proteus, pseudomonas, pasteurella, enterococcus, and *Escherichia coli*. The infection advances through an inflamed or ruptured tympanic membrane and crosses the round or oval window into the perilymph around the membranous labyrinth. Infection may also spread from haematogenous routes or from the pharynx, through the eustachian tube, and into the middle ear. As noted in Chapter 6, bacterial causes of otitis media interna are usually secondary to some other primary or predisposing condition. Cats are more likely than dogs to have extension of bacterial otitis interna to the meninges, which results in central vestibular dysfunction.

In cats, peripheral vestibular dysfunction may be secondary to an *aural or nasopharyngeal polyp* (Fig. 7.10). The associated otitis media interna can be aseptic. Preoperative vestibular signs may include head tilt, ataxia, and nystagmus; more commonly, nystagmus and ataxia are iatrogenic as a result of ventral bulla osteotomy for polyp removal. Postoperative nystagmus usually resolves within 24 hours after surgery; however, head tilt present before surgery is likely to persist postoperatively.

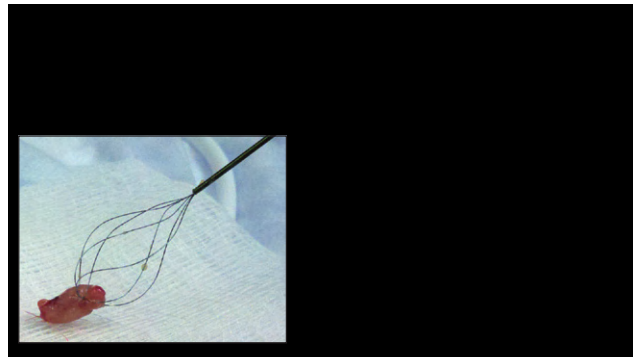


Figure 7.10 Under otoscopic guidance, this aural polyp was retrieved from a cat's horizontal ear canal and dorsolateral bulla by traction with a 3 mm nitinol tipless retrieval basket (inset) inserted through the port of the video otoscope. (Courtesy, Danielle Browning, LVT, UTCVM) © 2012 The University of Tennessee.

Neoplasia can cause peripheral vestibular dysfunction through direct invasion from the brain or middle ear

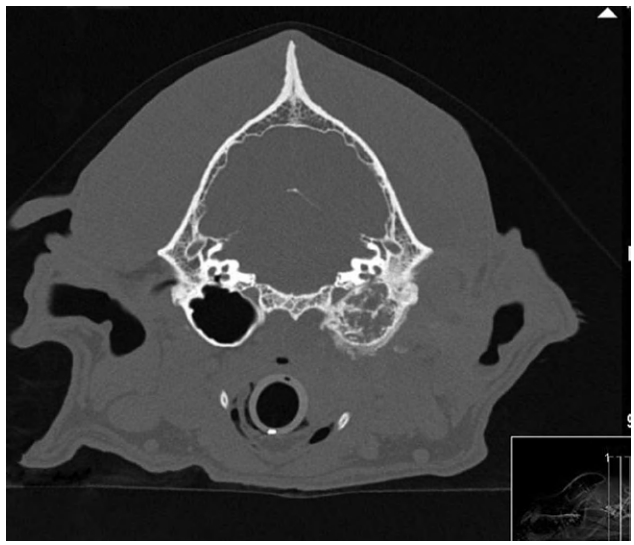


Figure 7.11 Transverse CT image. Osteosarcoma of the tympanic bulla in a 7 year old male castrated Chesapeake Bay Retriever with otitis externa/media, temporalis muscle atrophy, and Horner's syndrome. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

or secondarily (e.g. a tumour obstructing the external ear canal or eustachian tube) by inciting inflammation or fluid accumulation within the middle ear and secondary otitis interna (Fig. 7.11). Tumours arising from the ear canal are more common than those arising from the bulla or bony labyrinth. A variety of carcinomas and sarcomas of the middle or inner ear have been reported to cause peripheral vestibular disease, with squamous cell carcinoma reported most commonly in the cat. Lymphoma and vestibular neurofibroma have also been reported.

Ototoxicity has been reported with a variety of compounds (Table 7.2). Because many of the reports are based on experimental models, their clinical relevance is difficult to interpret. For instance, many studies on ototoxicity of topical substances used rodents as test subjects; compared with dogs and cats, these animals have much thinner tympanic, cochlear, and vestibular membranes, which may permit greater transit of noxious substances. Studies often use drugs at amounts and dosing intervals much greater than those used clinically and therapeutically, and the method of testing may involve unlikely clinical scenarios. For example, toxicity is instigated in some studies by direct application of the offending chemical to the cochlear window, a difficult feat to accomplish even in an animal with a ruptured tympanic membrane, assuming that the middle ear is replete with debris and its lining inflamed. On the other hand, most studies are performed on normal test subjects and therefore do not take into account effects of concurrent medications or illnesses that could potentiate toxicity.

Topical vestibulotoxicity may be dose or species related. For example, application of a 2% chlorhexidine solution directly into the middle ear of normal cats resulted in deafness and clinical signs of peripheral vestibular dysfunction, whereas administration of a 0.2% solution twice daily for 21 days in dogs caused no auditory damage or vestibular signs. In another study, application of 0.05% chlorhexidine to the middle ear in cats resulted in decreased numbers of cilia and edematous dilation of some nerve endings. Daily application of 0.4 mL of 10% gentamicin solution directly to the round window membrane and middle ear mucosa in normal cats was vestibulotoxic after 4 days. No effects were seen after 1 month, however, with daily application of solution concentrations less than 3%.

In normal dogs, twice daily topical application of 7 drops of a buffered aqueous solution containing gentamicin (3 mg/mL) produced no detectable vestibular or auditory dysfunction after 21 days, whether or not the tympanic membrane was intact. Similar results were found with application of a commercial flushing solution of Tris–EDTA and polyhexamethylene biguanide applied to the external or middle ear of normal dogs. In an older study, infusion of 1 mL of ceruminolytic agents containing propylene glycol and 2% dioctyl sodium sulfosuccinate or carbamide peroxide caused vestibular signs in some dogs.

Vestibulotoxicity of systemic drugs is usually associated with large doses, therapy for longer than 14 days, or concurrent impaired renal function. Aminoglycosides concentrate in perilymph and endolymph and damage hair cells in the cochlea and neuroepithelium of the maculae and crista. In one study, cats dosed with daily subcutaneous tobramycin (40–80 mg/kg) or gentamicin (20–40 mg/kg) demonstrated vestibular dysfunction after an average of 41 and 61 days of treatment, respectively. These doses are well above those recommended for clinical use. In another study, cats that received 20 mg/kg gentamicin or 22 mg/kg streptomycin twice daily for 14 days developed vestibular dysfunction. Partial improvement was noted 1 month after cessation of the drugs. Cats administered subcutaneous amikacin at 45 or 90 mg/kg/day developed no vestibular signs, while cats given subcutaneous gentamicin at 9 or 18 mg/kg/day developed vestibular function after an average of 68 or 42 days of treatment, respectively. Metronidazole may cause central vestibular dysfunction after 3 to 14 days, usually when administered at doses of 60 mg/kg/day or greater, although toxicity in dogs has been reported for doses as low as 39 mg/kg/day. Most affected dogs recover in 1–2 weeks.

Table 7.2 Effects of various medications on vestibular function in dogs and cats

Drug	Route	Dosage	Species	Effect
Tobramycin	Subcutaneous	40–80 mg/kg/day	Cats	Vestibular dysfunction an average of 41 days after initiating treatment
Gentamicin	Subcutaneous	9–18 mg/kg/day	Cats	Vestibular dysfunction an average of 42–68 days after initiating treatment
Amikacin	Subcutaneous	45–90 mg/kg/day	Cats	No evidence of vestibular function after >40 days of treatment
Metronidazole	Per os	≥60 mg/kg/day × 3–14 days	Dogs	Central vestibular dysfunction that usually resolves in 1–2 weeks
Chlorhexidine solution (2%)	Topical	0.6 mL applied directly into middle ear q48 h × 3 treatments	Cats	Peripheral vestibular dysfunction
Chlorhexidine solution (0.2%)	Topical	7 drops applied directly into middle ear twice daily × 21 days	Dogs	No vestibular dysfunction
Gentamicin sulfate solution (10%)	Topical	0.4 mL/day applied directly to round window and middle ear × 31 days	Cats	Vestibulotoxic after 4 days
Gentamicin sulfate solution (<3%)	Topical	0.4 mL/day applied directly to round window and middle ear × 31 days	Cats	No effect on vestibular function
Gentamicin sulfate, solution (3 mg/mL)	Topical	7 drops twice daily × 21 days	Dogs	No vestibular effects, irrespective of status of tympanic membrane
TrisEDTA + polyhexamethylene biguanide solution	Topical	2–5 mL twice daily × 14 days	Dogs	No vestibular effects, irrespective of status of tympanic membrane

Fractures or bony anomalies (Fig. 7.12) of the petrosal part of the temporal bone or tympanic bulla can result in signs of peripheral vestibular dysfunction and may be accompanied by facial nerve deficits. More commonly, vestibular signs are seen with iatrogenic trauma that occurs during curettage of the bulla or vigorous middle ear lavage.

Although uncommon, *congenital vestibular disorders* have been reported in litters of cats and dogs (Fig. 7.13). Affected breeds include Beagle, Dobermann Pinscher, Cocker Spaniel, Akita, Smooth Fox Terrier, and German Shepherd dogs and Siamese, Burmese, and Tonkinese cats. Clinical signs of peripheral vestibular dysfunction and unilateral or bilateral deafness may be detected by one month of age and may improve by 5 months of age, although some animals have persistent head tilt and deafness.

Other rare causes of peripheral vestibular disease include botfly (*Cuterebra*) migration through the ear canal into the inner ear, cryptococcosis otitis media interna in cats, and hypothyroidism in dogs. Hypothyroidism can also cause central vestibular disease; with either form, thyroid hormone supplementation results in rapid improvement.

When the underlying cause of peripheral vestibular dysfunction cannot be determined, vestibular disease is classified as idiopathic. *Idiopathic feline vestibular syndrome* is characterized by a sudden onset of non-progressive vestibular dysfunction, or an acute onset that progresses over 3 weeks. Idiopathic feline vestibular syndrome occurs most commonly in the summer months in the north-eastern United States and is often preceded by signs of upper respiratory tract irritation. Affected cats have no distinct



Figure 7.12 Transverse CT image. Petrous temporal and squamous bone malformation in a 1 year old female French Bulldog that acutely developed a left head tilt at 6 months of age. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.



Figure 7.13 Congenital vestibular disorders are rare in cats and dogs; in most young animals, the condition is acquired secondary to otitis media.



Figure 7.14 In this dog with idiopathic vestibular syndrome, ataxia resolved but the head tilt persisted.

signalment and can be of any age. Most cats have signs associated with unilateral peripheral vestibular disease – severe head tilt, disorientation, falling, rolling, nystagmus – with no other neurologic deficits. With bilateral disease, affected cats adopt a wide-based stance or crouch in response to severe disorientation, swing their heads widely from side to side, and lack normal physiological nystagmus. Some cats will have vomiting and anorexia. Results of otoscopy, radiographs, and advanced imaging are normal; most cats will improve without treatment within 3 days and are usually normal within 3 weeks. Head tilt may persist in some cats.

Idiopathic canine vestibular syndrome is also acute in onset and is noted primarily in older dogs (Fig. 7.14). Clinical signs are similar to those seen in cats; occasionally, dogs will have vomiting or nausea before the onset of vestibular signs. The disease is usually self-limiting, with most dogs spontaneously improving within 3 days and normal within 1–2 weeks. Occasionally, the head tilt persists.

Diagnosis

History and Physical Examination

Obtaining a thorough history and physical examination is critical for ruling in or out potential causes of vestibular dysfunction. Historical information should include any previous trauma, surgeries, ear or skin infections, allergies, pain, seizures, neurological or behavioural abnormalities, changes in eyesight or hearing, or respiratory or gastrointestinal signs. Onset, duration, and progression of any clinical signs should be determined. Clients should be questioned as to whether their pets undergo ear cleaning or receive any oral medications or topical products, including antimicrobials, ceruminolytics, and cleansing agents. A

full physical examination should include neurologic, ophthalmic, and otoscopic examinations and evaluation of hearing. Animals may need to be heavily sedated or anesthetized to permit evaluation of the external ear canal, tympanic membrane, and pharyngeal region (Fig. 7.15).

Baseline Diagnostics

Baseline diagnostics may include a complete blood count, chemistry panel, and urinalysis to evaluate the animal for underlying systemic disease. A thyroid panel is performed in dogs suspected to have hypothyroidism. Thoracic radiographs and abdominal ultrasound are examined for evidence of neoplasia or systemic disease. A Schirmer tear test can be used to detect damage to the major petrosal nerve, a branch of the facial nerve that runs through the middle ear. Damage to this nerve can potentiate development of KCS.

Masses detected on otoscopy can be aspirated or biopsied for cytology and histology. If disease is localized to the middle ear, fluid for cytology and culture can be retrieved by myringotomy (see Chapter 6).

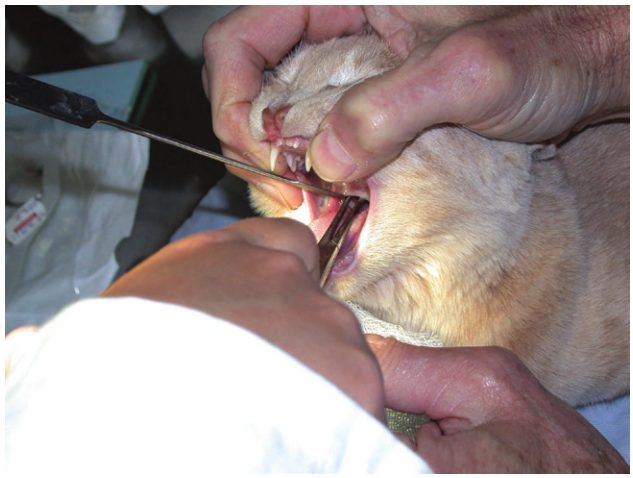


Figure 7.15 Nasopharyngeal polyps may be visualized in anesthetized cats by retracting the soft palate rostrally with a spay hook.

Imaging

Skull radiographs are most commonly used to detect otitis media but may also provide a diagnosis in animals with traumatic or destructive lesions. Abnormalities may include obstruction or calcification of the external ear canal, destruction of the tympanic bulla or bony labyrinth, fracture of the bulla or petrosal portion of the temporal bone, or soft tissue density within the middle ear (Fig. 7.16). About 25% of animals with otitis media may have normal skull radiographs, however, and therefore advanced imaging (CT or MRI) may be required to evaluate the middle ear and rule out other lesions. In many animals, skull radiographs are of limited use for diagnosis of otitis interna.

CT is usually considered the next step for evaluation of the peripheral vestibular system in cases where diagnosis is not evident or extent of disease cannot be determined based on skull radiographs. CT enhances bony changes associated with the bulla and petrosal portion of the tem-

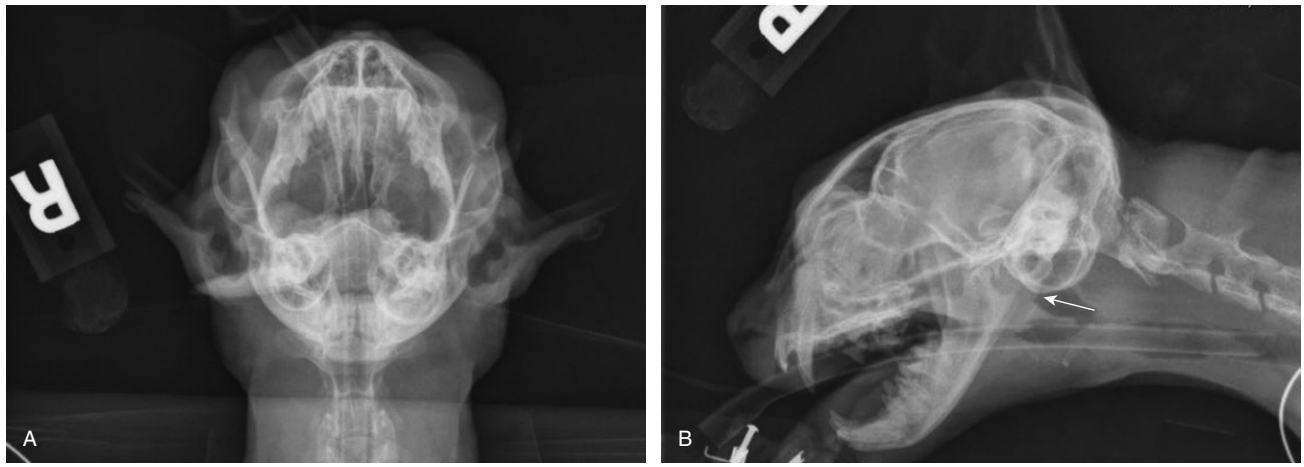


Figure 7.16 Skull radiographs in an 8 month old female spayed Ragdoll cat with a history of stertor and gasping. Enlargement and wall thickening of the left bulla are notable on the open mouth view (**A**), and a soft tissue density (arrow) is visible in the caudal nasopharynx on the lateral view (**B**). A nasopharyngeal polyp was removed through transoral traction and ventral bulla osteotomy. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

poral bone and is therefore helpful in patients with trauma or extension of middle ear neoplasia (Fig. 7.17). It is also more sensitive than skull radiographs for detecting moderate to severe otitis media in dogs and for detecting fluid or tissue in the bullae of cats (Fig. 7.18). Extension of otitis media interna into brain can also be detected.

MRI is not commonly used for diagnosis of otitis media but is the preferred imaging modality for neoplastic and inflammatory processes that affect soft tissues associated with the vestibular system (Fig. 7.19) or for ruling out hydrocephalus and cerebral, cerebellar, or brainstem lesions that cause central vestibular disease (Fig. 7.20). MRI may

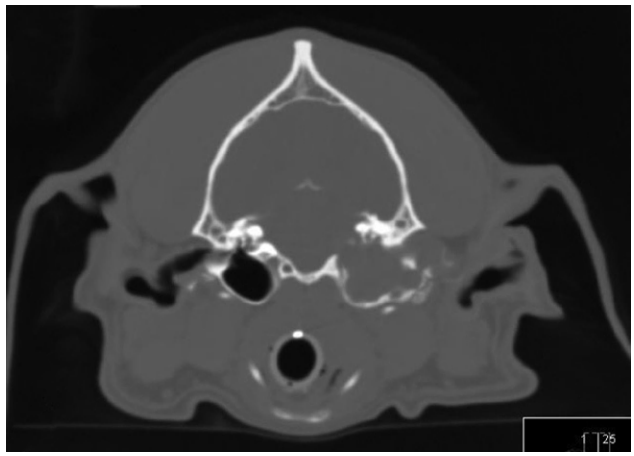


Figure 7.17 Invasive soft tissue mass in an 8 year old mixed breed male castrated dog presented with an acute onset of otitis externa and left head tilt. Expansion and lysis of the left bulla are visible, and the bulla is filled with a soft tissue density that extends into the external ear canal. Lysis is also seen along the calvarium, and extension into periauricular tissues and brainstem was noted on contrast studies. Ceruminous gland adenocarcinoma was suspected. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.



Figure 7.19 MRI of a 7 year old Swiss Mountain Dog with a 1 year history of left head tilt and more recent onset of left facial droop. Enhancement of the left facial and vestibulocochlear nerves (arrow), which are encased together in a sheath of dura as they travel through the internal acoustic meatus, was noted after injection of gadolinium. No improvement was seen with prednisone treatment. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

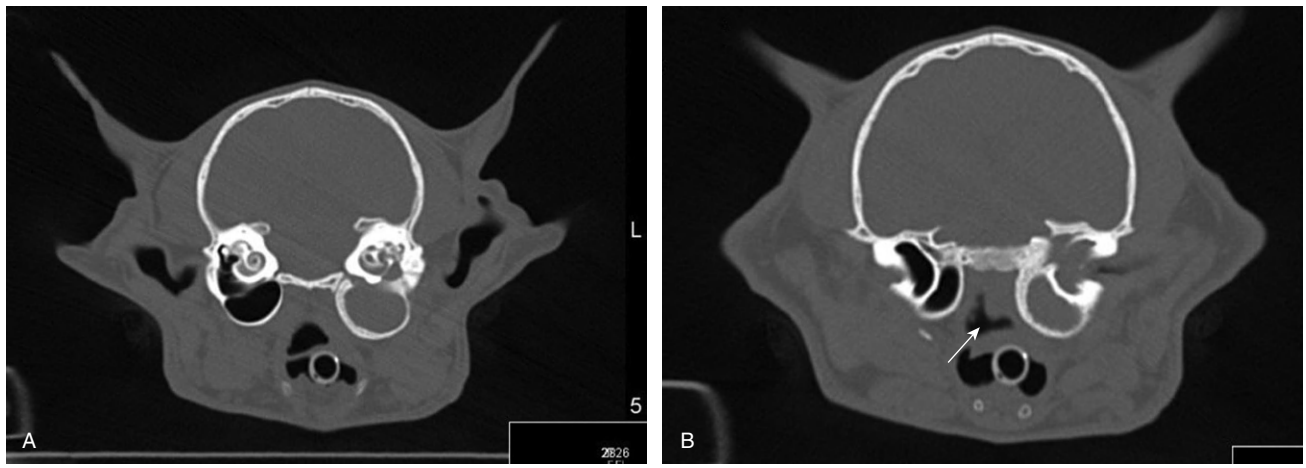


Figure 7.18 Transverse CT image. Otitis media in the cat from Figure 7.16. The left bulla is enlarged, thickened, and filled with a soft tissue density (**A**). Note the mass within the nasopharynx (arrow) (**B**). The septum dividing the normal right bulla is easily visible on both views. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

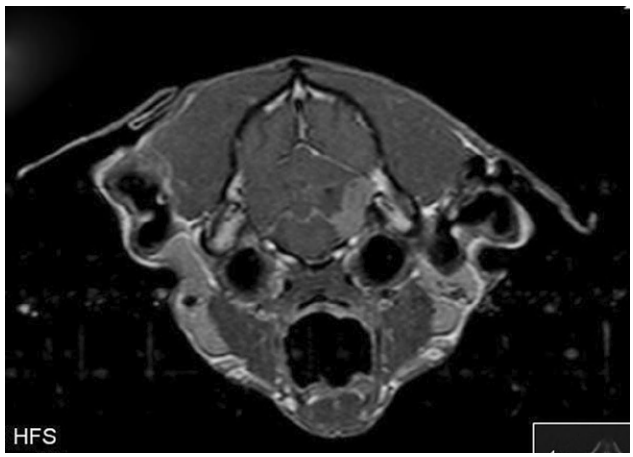


Figure 7.20 MRI of a 12 year old male castrated Greyhound with a 3 month history of weakness, ataxia, and head tilt to the left. A contrast-enhancing mass is present in the cerebellum and brainstem and is surrounded by oedema. Changes were consistent with meningioma. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

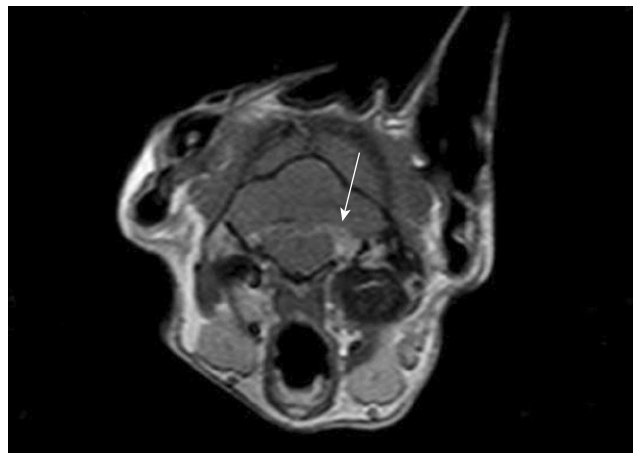


Figure 7.21 MRI of a 1 year old female spayed Siamese cat presented with left head tilt, circling, and falling to the left. On physical examination she had a horizontal nystagmus with the fast phase to the right, and poor physiological nystagmus to the left. On MRI the left bulla was thickened and destruction of the petrous temporal bone and cochlea were noted. Contrast enhancement (arrow) was seen in the bulla, meninges, choroid plexus, brainstem, and cerebellum. The final diagnosis was aseptic otitis media with secondary extension into the brain. (Courtesy, UTCVM Radiology) © 2012 The University of Tennessee.

also detect extension of infectious otitis media interna into the meninges or parenchyma of the brain (Fig. 7.21). Lesions are detectable on MRI in 85% of cats and 100% of dogs with central vestibular dysfunction and 57% of cats and 74% of dogs with peripheral vestibular dysfunction.

7.2 COCHLEA

Like the vestibular apparatus, the cochlea lies within the petrous temporal bone. Within the bony, spiral shell of the cochlea is the cochlear duct, a fluid-filled chamber that houses specialized sensory epithelium. Hair cells, or stereocilia, along this epithelium translate sound waves propagated by the fluid into neural impulses that are carried via the cochlear portion of the vestibulocochlear nerve to the brainstem and on to the cerebral cortex.

7.2.1 Hearing

Hearing initially relies on a series of physical events that conduct sound energy to the *organ of Corti*, the neural apparatus that includes the hair cells, tectorial membrane, and support cells (Fig. 7.22). Normally, sound waves are funnelled by the external ear canal to the tympanic membrane, resulting in its vibration. As the tympanic membrane vibrates, its movements are transmitted through the chain of ossicles (incus, malleus, and stapes) to the membrane of the oval window at the foot of the stapes (Fig. 7.23). Movement of the stapes and its associated membrane displaces perilymph within the adjacent scala vestibuli of the cochlear duct. The resulting fluid wave is transmitted up the scala vestibuli, through helicotrema, and down the scala tympani of the cochlear duct, causing the round window to bulge into the middle ear. Simultaneously, the movement vibrates the basilar membrane of the scala tympani and, subsequently, the adjacent endolymph of the scala media. Fluid waves passing through the endolymph within the scala media move the tectorial membrane, which contacts the sensory hair cells, stimulating their depolarization. Sound waves can also be detected by *direct bone conduction* (*osteotympanic conduction*) in animals with normal neural pathways but abnormal external or middle ear structures (Fig. 7.24).

The external acoustic meatus opens in puppies and kittens between 6 and 14 days of age (Fig. 7.25), followed by gradual widening of the canals over the next 5 weeks. Sound perception, however, is evident in kittens as young as 5 days of age and in puppies as young as 10 or 11 days of age, even if the external acoustic meatus is not completely open. Most puppies and kittens have normal hearing of environmental sounds by 1 month of age.

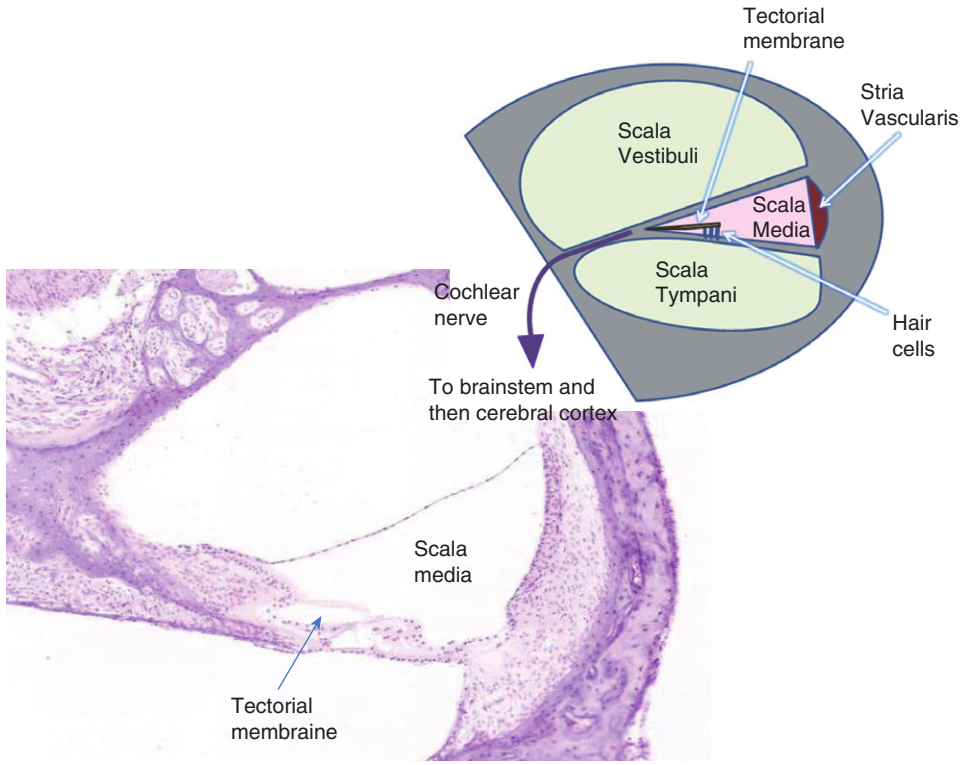


Figure 7.22 Histological cross-section (left) and diagrammatic representation (upper right) of a portion of the cochlea. Vibrations from the stapes are transmitted through the oval window to the perilymph within the scala vestibuli, which joins the scala tympani at the helicotrema. The fluid wave travels along the basilar membrane, the movement of which rocks the hair cells that project up into the gel coating of the tectorial membrane. The to and fro motion of the hair cells along the membrane stimulates them to fire, sending impulses down the cochlear nerve. (Histological section courtesy of UTCVM Virtual Microscope) © 2012 The University of Tennessee.

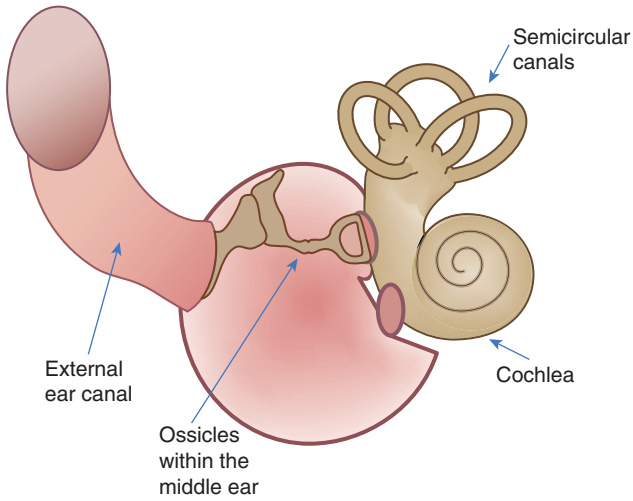


Figure 7.23 Schematic of sound wave conduction path. Sound waves enter the external canal and vibrate the tympanic membrane. The ossicles within the middle ear transmit the vibrations to the membrane of the oval window.



Figure 7.24 The mastoid process (arrow) lies just caudal to the stylomastoid foramen, where the facial nerve exits the skull. In people, a mechanical vibrator is placed against the skin over the mastoid process to test hearing via bone conduction to the cochlea. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

7.2.2 Deafness

Deafness is classified as sensorineural or conductive, inherited or acquired, and early (congenital) or late onset. Conductive deafness results when something interferes with conduction of sound waves from the external environment to the cochlear duct, whereas sensorineural deafness is usually caused by abnormalities of the organ of Corti or cochlear nerve.



Figure 7.25 The external acoustic meati are still closed in these 7 day old Yorkshire Terrier puppies.

Conductive Deafness

Conductive deafness may occur from external ear canal or middle ear obstruction with tissue or debris, tympanic membrane rupture, immobility of the tympanic membrane or auditory ossicles, or abnormalities of perilymph. The most common aetiologies are otitis media and severe proliferative otitis externa. Neoplasia of the middle or external ear canal may also prevent air transmission or movement of the tympanic membrane, ossicles, or oval window membrane (Fig. 7.26). Congenital conductive deafness from absence of the external acoustic meatus is rare; however, traumatic avulsion of the ear canal at a young age may result in stenosis or atresia of the canal (Fig. 7.27).

Sensorineural Deafness

Sensorineural deafness occurs primarily with damage to the tectorial membrane, stereocilia, or cochlear nerve. Although brainstem and cortical diseases could theoretically cause sensorineural deafness, the damage would need to be so extensive as to make these aetiologies unlikely. Types of sensorineural deafness include inherited deafness, ototoxicity, cochlear nerve degeneration, and age-related changes.

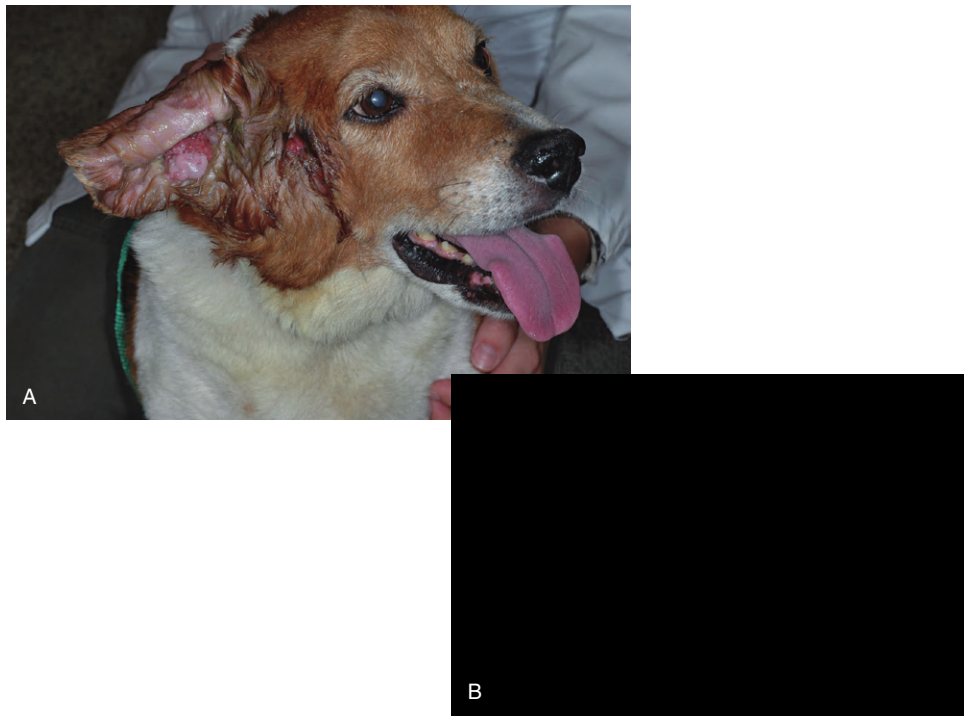


Figure 7.26 Unilateral canal obstruction and secondary deafness in a 13 year old Beagle with ceruminous gland adenocarcinoma. The mass obstructed the ear canal, resulting in canal rupture and development of sinus tracts (**A**). The proliferative mass extends out of the external ear canal (**B**). The mass was completely removed with pinnectomy, total ear canal ablation, and lateral bulla osteotomy; however, lymphatic invasion noted on histologic samples increased the risk for future metastatic disease.

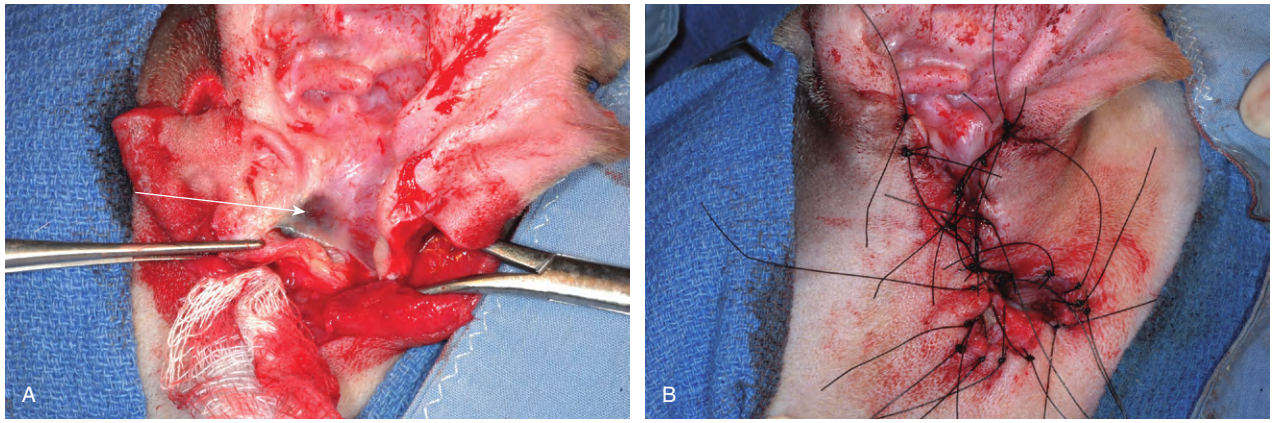


Figure 7.27 Canal stenosis or atresia in a 5 year old female spayed Beagle mix with a history of bilateral intermittent facial swelling and otitis since 7 weeks of age. **(A)** The lateral ear canal was opened to expose the obstruction (arrow). Both horizontal canals ended in blind sacs, and periauricular abscesses were present bilaterally. **(B)** The horizontal canals remnants were sutured to the skin in a technique similar to a vertical ear canal resection.

Inherited Sensorineural Deafness

Inherited (congenital) sensorineural deafness has been reported in cats and in over 60 breeds of dogs. In affected animals, hearing is usually present for the first few weeks of life but disappears by 2 months of age, raising the suspicion that this is a degenerative process. Two underlying aetiologies are suspected: dysfunction of the stria vascularis (pigment-associated deafness) or degeneration of the stereocillia (abiotrophic form).

Pigment-associated inherited deafness occurs most often in animals that are partially or completely white. In normal animals, melanocytes in the stria vascularis that lines the scala media are responsible for maintaining high potassium concentrations in the endolymph. In animals with pigment-associated deafness, melanocytes are absent. Potassium is no longer transferred into the endolymph, resulting in loss of endocochlear potential and subsequent degeneration of the stereocilia.

White cats are commonly affected with this condition; in fact, those with white coats and two blue irises have an incidence of almost 50% (Fig. 7.28). Incidence in cats with one blue eye is lower; however, these cats are at a greater risk than cats with irises that are not blue. Dalmatians are the most commonly affected dog breed, with an incidence of unilateral or bilateral deafness in nearly 30% of puppies. Dalmatians with blue eyes have a higher incidence of deafness. The condition is inherited as an autosomal dominant trait in this breed. In certain breeds of dogs, deafness has also been associated with merle, dapple, and piebald coat colours.

In animals with *abiotrophic inherited deafness*, the stereocilia degenerate despite a normal endocochlear potential.



Figure 7.28 Almost 50% of white cats with two blue eyes are diagnosed with pigment-associated inherited deafness. In this cat, the pinnae have an unusual contour because of laser resection for treatment of bilateral squamous cell carcinoma of the pinna margins.

This condition is usually inherited as an autosomal recessive trait and most commonly causes bilateral deafness. Stereovilli within the semicircular ducts may also be affected, resulting in signs of vestibular disease. In some breeds such as the Cavalier King Charles Spaniel, the deafness may not manifest until 3 or 4 years of age. Cavalier King Charles Spaniels are also predisposed to secretory otitis media, which can cause conductive hearing loss and neurologic signs.

Clinical signs of bilateral inherited sensorineural deafness are usually noted at an early age. Besides hearing loss,

affected animals may display behavioural changes such as aggression and increased vocalization, and they may be difficult to rouse from sleep.

Acquired Sensorineural Deafness

Causes of acquired sensorineural deafness include age-related changes, ototoxic substances, hypothyroidism, trauma, neoplasia, and chronic exposure to loud noises.

Presbycusis

Late onset sensorineural deafness, or *presbycusis*, is a decline in hearing associated with ageing. The underlying cause is not well understood. It may be a form of sensorineural deafness from degeneration of hair cells, spiral ganglion cells, the cochlear nerve, or cochlear nuclei; atrophy of the stria vascularis; or changes in the basilar membrane. Alternatively, it may be a form of conductive deafness secondary to arthrosis of the ear ossicle articulations. Affected animals are usually 8 years of age or older and therefore may also be prone to other diseases that could potentially affect hearing, such as chronic otitis externa or neoplasia.

Ototoxicity

Ototoxicity with subsequent auditory loss has been reported experimentally with systemic administration of a variety of drugs; however, as with vestibulotoxicity, the clinical relevance of these reports is hard to interpret. Likely, auditory loss is related to the species, drug dose, duration of treatment, and other factors such as renal function or concurrent drug administration (Table 7.3).

Systemic aminoglycosides are of greatest concern, particularly in cats. Effects of normal doses on auditory function, however, have not been reported. In cats, daily subcutaneous administration of amikacin at 90 mg/kg per day or 45 mg/kg per day resulted in loss of response to sound after an average of 39 and 78 days of treatment,

Table 7.3 Effects of various medications on hearing in dogs and cats

Drug	Route	Dosage	Species	Effect
Amikacin	Subcutaneous	90 or 35 mg/kg/day	Cats	Loss of response to sound after 39–78 treatments
Gentamicin	Subcutaneous	9–18 mg/kg/day	Cats	No effect on hearing after >40 treatments
Furosemide	Intravenous	50 mg/kg once	Dogs	Temporary deafness; however, diuretics may potentiate effects of other drugs, e.g. aminoglycosides
Cisplatin	Intravenous	100 mg/m^2	Dogs	Altered hearing threshold
Gentamicin sulfate solution	Topical	7 drops (3 mg/mL) twice daily × 21 days	Dogs	No effect on BAER tests, irrespective of status of tympanic membrane
TrisEDTA + polyhexamethylene biguanide solution	Topical	2–5 mL twice daily × 14 days	Dogs	No effect on BAER tests, irrespective of status of tympanic membrane
Chloramphenicol solution	Topical	400 mg/mL in gelfoam, one application directly to round window	Cats	Partial decrease in cochlear function
Chlorhexidine solution (2%)	Topical	0.6 mL applied directly into middle ear q48h × 3 treatments	Cats	Pronounced degeneration of cochlear and vestibular hair cells
Chlorhexidine solution (0.2%)	Topical	7 drops applied directly into middle ear twice daily × 21 days	Dogs	No changes in BAER

q48h, every 48 hours.

respectively. Daily subcutaneous administration of gentamicin at 18 and 9 mg/kg per day had no apparent effect on hearing, however. Intramuscular administration of neomycin in cats (50 mg/kg per day) resulted in progressive degeneration of spiral ganglion neurons. Intravenous administration of neomycin at 30 mg/kg per day for 22 days resulted in a complete loss of hearing based on brainstem auditory evoked response (BAER) tests. A single intravenous administration of 100 mg/kg of kanamycin sulfate resulted in reversible changes in BAER waveforms. In one Labrador, treatment with subcutaneous neomycin (500 mg total dose) for 5 days resulted in deafness.

Loop diuretics have a dose-related effect on auditory function, likely because of changes in potassium concentrations in endolymph. Large doses (e.g. 50 mg/kg IV in dogs), however, are required and effects are usually temporary. A combination of drugs may have more severe consequences. For instance, a single administration of kanamycin and the loop diuretic ethacrynic acid causes profound deafness in adult cats.

Cisplatin ototoxicity has been reported clinically in human patients. Experimentally, a single dose of cisplatin (100 mg/m^2) affected hearing threshold in normal dogs within 4 days of administration, and one dog developed acute deafness and blindness after four treatments.

Ototoxicity of *topical medications* is also dose and species dependent. Topical administration of gentamicin sulfate (3 mg/mL solution) twice daily for 21 days in the ears of normal dogs with intact or ruptured tympanic membranes had no effect on results of BAER tests. Similar results were found with external and middle ear application of TrisEDTA and polyhexamethylene biguanide ear flush. Direct application of a single dose of chloramphenicol (400 mg/mL) to the round window in cats decreases cochlear function; function returns within 2–4 days, although incompletely. In an older study, infusion of 1 mL of ceruminolytic agents containing propylene glycol and 2% dioctyl sodium sulfosuccinate, carbamide peroxide, or triethanolamine decreased auditory function in some dogs.

Diagnosis of Deafness

Initial diagnosis is based on owner observations. Dogs with complete bilateral deafness cannot be roused from sleep by loud sounds. Owners may also perceive behavioural changes in their dogs if unilateral or partial deafness is present. In predisposed breeds such as Dalmatians, however, auditory dysfunction may be diagnosed during routine hearing tests. Physical examination should include otoscopic and ophthalmic examinations for any signs of otitis externa or media. Advanced imaging may be necessary in patients suspected to have otitis media interna.



Figure 7.29 Preyer's reflex. The dog's ears are elevated and rotated forward from a resting position (inset) in response to sound. (Photographs by Phil Snow, UTCVM) © 2012 The University of Tennessee.

Hearing is difficult to assess by physical examination in a clinical setting. Response to sounds must be tested without visual cues or tactile signals from air movement or floor vibrations. Preyer's reflex – a pricking of the ears in response to sound – is a behavioural indication of hearing in dogs (Fig. 7.29). Unfortunately, this reflex is inconsistent at sound frequencies below 8000 Hz, which is well above the normal speaking pitch of humans, which averages 120 Hz in men and 250 Hz in women. This reflex may best be stimulated with a silent dog whistle that emits sound frequencies of 12 000–22 000 Hz. More objective measurement of hearing is obtained by evaluating BAER or impedance audiometry.

Impedance audiometry or *tympanometry* measures compliance of the tympanic membrane. The external ear is occluded, and pressure within the canal is measured during sound wave generation. If the results are normal, deafness is sensorineural. Impedance audiometry is not commonly performed in animals because the required equipment is constructed to fit the human ear canal.

Brainstem Auditory Evoked Response

BAER, also known as brainstem auditory evoked potential, detects unilateral or bilateral hearing deficits (Fig. 7.30). It has also been used to monitor progressive changes in hearing over time. The BAER test is usually performed under sedation or light anaesthesia in animals that are at least 6 weeks of age (Fig. 7.31). Electrodes positioned on the animal's head are used to detect neural response to auditory stimulus (Fig. 7.32). Earphones or ear plugs are

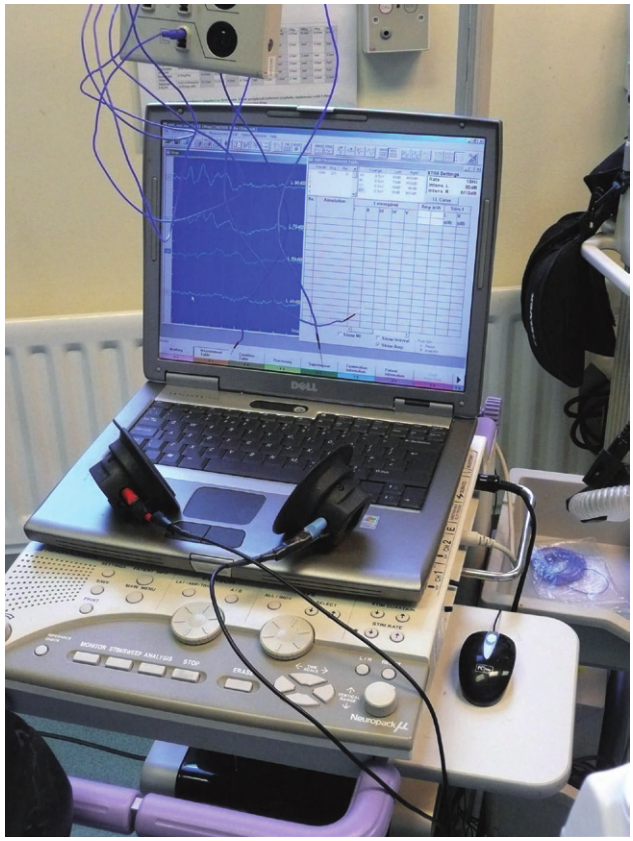


Figure 7.30 Hearing deficits are evaluated with a computerized system that measures response to sound transmitted through earphones.

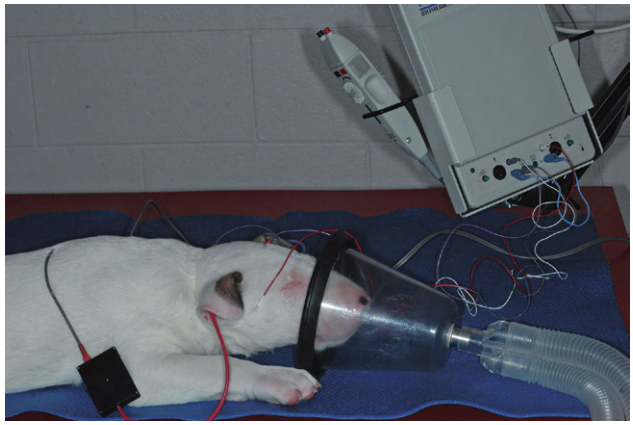


Figure 7.31 The animals are lightly anesthetized with gaseous anaesthetic such as sevoflurane to permit adequate sedation and rapid recovery.

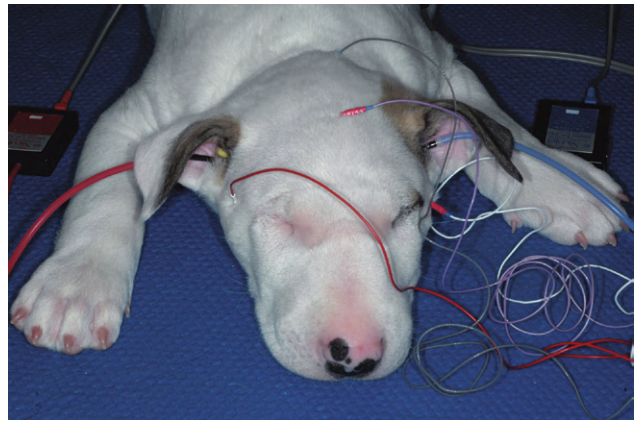


Figure 7.32 Electrodes are placed under the skin, and sound is delivered through earplugs or earphones.

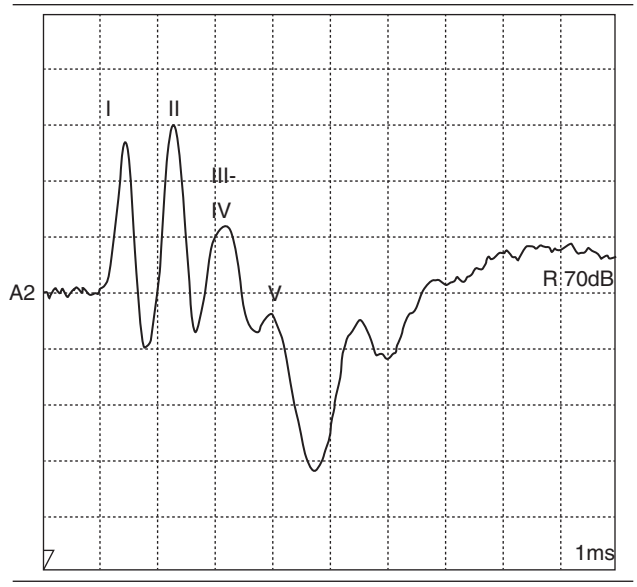


Figure 7.33 Normal BAER test. (Courtesy, UTCVM Neurology Service) © 2012 The University of Tennessee.

placed on the animal, and a series of clicks is delivered into one ear while hearing in the other ear is 'blocked' by white noise.

Clicks are delivered over a range of decibels, and response to the clicks is recorded as a series of waves that indicate electrical activity within the inner ear and successive levels of the brainstem. Wave I corresponds to the cochlear portion of the ipsilateral vestibulocochlear nerve as it leaves the internal auditory canal (Fig. 7.33). Wave II includes reactions of the vestibulocochlear nerve as it enters the brainstem. Waves III, IV, and V represent reactions within the auditory portions of the brainstem.

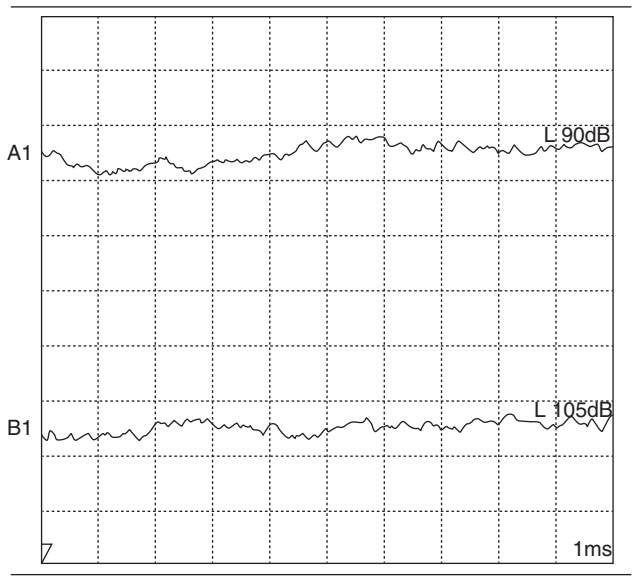


Figure 7.34 All waves are absent in animals with inherited sensorineural deafness or with severe lesions that interfere with travel of sound waves through the external or middle ear. (Courtesy, UTCVM Neurology Service) © 2012 The University of Tennessee.

The BAER test can be abnormal with conduction or sensorineural deafness (Fig. 7.34). In animals with inherited sensorineural deafness, all waves are usually absent. In animals with conductive deafness, the time, or latency, from the stimulus emission to the appearance of waves is prolonged, and wave amplitude is decreased. With severe lesions, animals with conductive deafness may have complete absence of waveforms. In those dogs, response to bone stimulation can be measured. Animals with conductive deafness will produce waveforms when vibrations are sent through the mastoid process, as long as the cochlea is normal.

In dogs with deafness associated with ageing, wave amplitudes are decreased. Normal waveforms may be produced by high-intensity sounds (80 dB) but not those in the normal canine hearing range (0–5 dB). When otoxicity is suspected as the underlying cause of deafness, BAER tests can be used to monitor patients for return of function after withdrawal of the offending substance. The BAER test has also been used for detection of brainstem lesions such as neoplasia, encephalitis, infarction, or brain death.

Falsely positive results for deafness from air-conductive BAER tests may occur in patients with ear canals filled with exudate, debris, or tissue; puppies or kittens less than 5 weeks of age; or hypothermic patients. Other causes include technical problems with the earphones, microphones, or electrodes. False-negative results are possible in deaf animals if lesions are located in regions not assessed by the test, such as the medial geniculate nuclei and the cerebral temporal lobes.

VetBooks.ir

VetBooks.ir

CHAPTER 8
SURGERY OF THE EAR

Karen Tobias

8.1	Pinna Lacerations	145	8.7	Subtotal Ear Canal Ablation with Lateral Bulla Osteotomy	157
8.2	Pinnectomy	146	8.8	End-To-End Anastomosis	158
8.3	Auricular Haematoma Drainage	146	8.9	Ventral Bulla Osteotomy	160
8.4	Lateral Wall Resection	148	8.10	Laser Ear Surgery	162
8.5	Vertical Ear Canal Resection	151			
8.6	Total Ear Canal Ablation with Lateral Bulla Osteotomy	154			

8.1 PINNA LACERATIONS

Lacerations may require surgical closure if they are full thickness or if they are partial thickness with the epithelium elevated from the cartilage as a two- or three-sided flap. Lacerations can be closed with absorbable or non-absorbable suture.

1. For partial thickness lacerations, close any dead space with a continuous suction drain or with partial thickness mattress sutures that engage epithelium and cartilage. Appose the skin along the laceration with simple interrupted sutures (Fig. 8.1). If an L-shaped tear is present, place the first suture at the corner of the L to realign the skin.
2. For full thickness lacerations, appose cartilage and skin by realigning the skin with simple interrupted sutures on each side of the pinna, starting at the margin of the ear (Fig. 8.2), or with simple interrupted sutures on one side and a vertical mattress pattern on the other. Trim excess cartilage if the skin cannot be apposed over the cartilage edge.
3. For lacerations or trauma that results in marginal defects or necrosis, perform a partial pinnectomy (see section 8.2).



Figure 8.1 Closure of partial thickness laceration. Superficial suture bites include only skin. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.



Figure 8.2 Closure of full thickness laceration. The pinna margin is apposed with the first suture, with bites excluding cartilage, if possible. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

4. Large partial thickness wounds near the base of the outer surface of the pinna are debrided and managed open until a healthy granulation bed has formed, then covered with single pedicle advancement flap from the lateral surface of the neck or muzzle or the dorsum of the head.
5. If a large, full thickness section of one pinna margin has been damaged or removed, the pinna can be reconstructed with distant flaps. The margins of the remaining pinna are debrided and a single pedicle flap is elevated from head or neck skin near the wound. The flap is sutured to epithelium along the convex (outer) surface of the pinna, and the ear is bandaged. Bandages are changed as needed. After 2 weeks, the flap is transected from the donor site to approximate the new margin of the pinna. A new flap is elevated along the dorsum of the head and sutured to the wound's epithelial margin along the concave surface of the ear, putting it in apposition (subcutis to subcutis) with the first flap. The ear is rebandaged. After 2 weeks, the second flap is transected and its margin is sutured to that of the first flap to form the final pinna margin.

8.2 PINNECTOMY

Pinnectomy is commonly performed in cats for resection of squamous cell carcinoma and in dogs for resection of mast cell tumours or removal of damaged pinna margins.

1. Clip and prep the surgical site.
2. Excise the affected tissue with laser, a scalpel blade, or cautery; take 1–2 cm surgical margins for neoplastic tissue. The laser will seal vessels up to 1 mm in diameter.
   a. For blade excision of large areas, place a non-crushing (e.g. Doyen) forceps along the proposed margin. Transect along the distal edge of the forceps with the scalpel. Leave the forceps in place for several minutes to provide haemostasis before suturing.
   b. For laser excision, use a 0.4 mm tip and set the laser on continuous mode at 8–10 W of power. With the laser, make a partial thickness cut to draw a line along the proposed margin to be resected. Then, starting at one pinna margin, begin the full thickness cut. Have an assistant apply lateral traction, perpendicular to the incision, to the pinna apex to retract the tissues and widen the surgical wound as the pinna is incised. Wipe char from the tissue as it forms; presence of char limits cutting and encourages collateral thermal damage.
3. Suture the medial and lateral skin edges together over the cartilage margin with 3-0 or 4-0 monofilament absorbable or non-absorbable suture in a simple continuous or interrupted pattern (Fig. 8.3). If bleeding vessels are encountered, seal them with bipolar cautery or ligate them by encircling them and the surrounding tissue with a partial or full thickness mattress suture.

8.3 AURICULAR HAEMATOMA DRAINAGE

Auricular haematomas are best treated early before granulation tissue formation and eventual contraction and fibrosis deform the pinna. Some authors recommend needle drainage (after clipping and aseptic preparation), flushing, and administration of oral glucocorticoids or instillation of a corticosteroid into the hematoma; others prefer more long-term drainage with cannulas, close suction systems, or incision. Incisional drainage may be necessary for chronic hematomas. In any case, the underlying cause of the head shaking or scratching must be identified and treated to prevent recurrence.



Figure 8.3 Pinnectomy closure. Skin from the concave and convex surfaces of the pinna are apposed to cover cartilage. (Photograph by Phil Snow, UTCVM) © 2012 The University of Tennessee.

8.3.1 Teat Cannula

1. Clip and aseptically prepare the ear.
2. With a number 11 blade, make a small stab incision in the apex of the ear at the dependent portion of the haematoma.
3. Insert a teat cannula through the stab incision and secure with suture.
4. Milk any fluid from the haematoma.
5. Bandage the ear flat against the dorsum of the head or ventrally along the neck or cheek to eliminate dead space. Change bandages as needed.
6. Remove the cannula after 1–2 weeks; leave the ear bandaged for 2 weeks.

8.3.2 Closed-Suction Drain

1. Clip and aseptically prepare the ear.
2. Remove the syringe adaptor end of a large butterfly catheter, and add 2–4 fenestrations to the distal 1.5–2 cm of the tubing (Fig. 8.4).
3. Insert a gauze sponge into the external ear canal. With a number 11 blade, make a small stab incision in the ventral (proximal) portion of the haematoma. Fluid will immediately drain from the haematoma cavity.
4. Insert the fenestrated end of the butterfly catheter into the hematoma cavity (Fig. 8.5). Place a purse string suture around the entry site to seal the wound, making it airtight around the tube. Secure the tube in place with a finger trap pattern.
5. Insert the needle end of the butterfly catheter into a vacuum blood tube (Fig. 8.6). Secure the tube to the animal's collar.

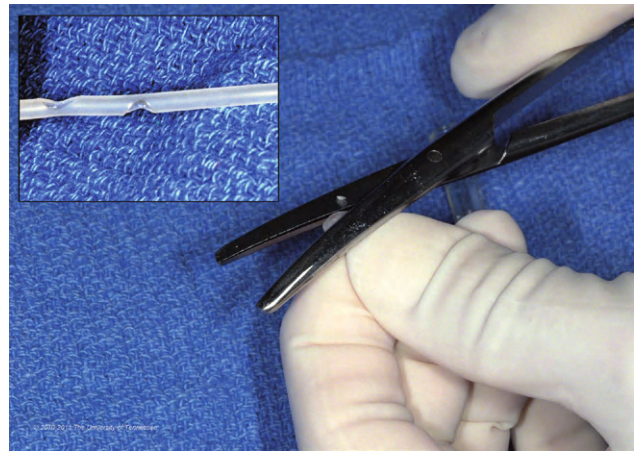


Figure 8.4 Continuous suction drain. After removal of the catheter adaptor end, extra fenestrations (inset) are made by folding the tube over and snipping off the corner of the fold. © 2012 The University of Tennessee.



Figure 8.5 The drain is inserted into the hematoma cavity through a puncture wound on the concave surface of the ear so that all the fenestrations lie within the hematoma cavity. © 2012 The University of Tennessee.



Figure 8.6 Final appearance of continuous suction drain for auricular hematoma treatment. The skin at the drain exit site is apposed with a mattress suture, and the drain is secured to the ear with a finger trap pattern, being careful to avoid crushing the tube. The needle end is inserted in a vacuum blood tube. © 2012 The University of Tennessee.

6. Bandage the ear as needed to eliminate dead space and prevent head shaking.
7. Replace the vacuum tube once it contains 2–3 mL or as needed to restore negative pressure on the drain.
8. Remove the drain after 1 week, then rebandage the ear for another week.

8.3.3 Incisional Drainage

1. Clip and aseptically prepare the ear.
2. Make a skin incision through the medial wall of the pinna into the haematoma cavity. Extend the incision so that it encompasses the entire length of the cavity. Flush the cavity with sterile saline.
3. To obliterate dead space in the haematoma cavity, place full thickness mattress sutures parallel to the vessels along the convex surface of the pinna; avoid penetrating the vessels. Tie sutures to appose skin and cartilage without necrosing underlying tissue (Fig. 8.7).
4. Place a sterile sponge over the incision site, and bandage the ear over the dorsum of the head or along the face or neck. Keep the ear bandaged for 2 weeks, changing the bandage as needed.
5. An alternative to incisional drainage with a scalpel is fenestration of the skin over the hematoma along concave surface of the pinna by use of a 4 mm skin biopsy punch or with a CO_2 laser. Fenestrations are placed about 1 cm apart, and the ear is bandaged for 1–2 weeks until the sites are healed.



Figure 8.7 The haematoma cavity is compressed with vertically oriented sutures that include tissue on the convex surface of the pinna. Sutures should be tightened to appose but not crush the tissue layers. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

8.4 LATERAL WALL RESECTION

Lateral wall resection is primarily used to correct congenital canal stenosis, resect laterally located masses within the vertical ear canal, or improve surgical exposure of deeper masses when video otoscopic equipment is unavailable. It is ineffective for treatment of chronic otitis externa, particularly in canals that are proliferative or calcified, but may improve local environmental factors and ease of topical medication administration. Failure rates are high in Cocker Spaniels, which tend to have progressive otitis externa.

1. Clip lateral portion of face, both sides of pinna, and around the base of the ear for complete clip, particularly if making the drain board by cutting the lateral wall from dorsal to ventral (with this technique, the pinna is usually held in one hand while the incisions are made with the other). Flush the canal with sterile saline and use chlorhexidine for the remainder of the prep.
2. Alternative clip: include only the lateral portion of face and concave surface of pinna, if the lateral canal wall



Figure 8.8 Lateral ear canal resection. A U-shaped incision, with end points at the tragohelicine notch and intertragic incisures, is made over the lateral wall of the vertical ear canal. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

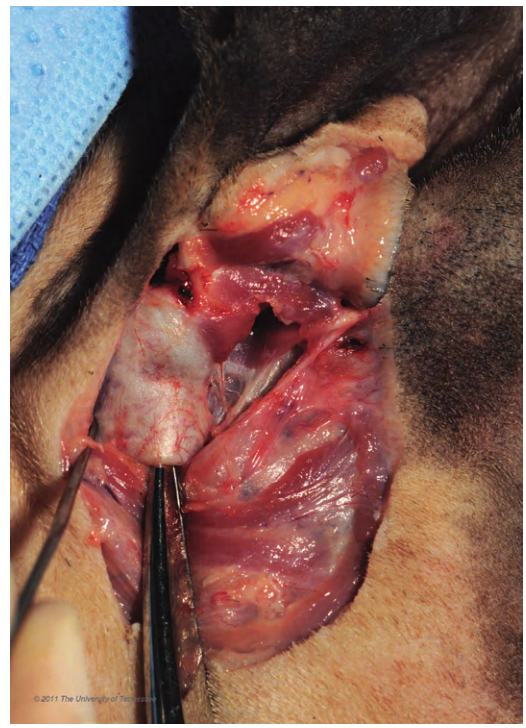


Figure 8.9 Lateral ear canal resection. Subcutaneous tissues and parotid gland have been dissected off the vertical canal. The tip of the scissors indicates the ventral (proximal) edge of the vertical canal. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

is to be opened with proximal (ventral) to distal (dorsal) cuts (this technique may not require manipulation of the pinna during surgery).

3. Position the animal in lateral recumbency. If the pinna is included within the surgical field, perform a hanging ear preparation of pendulous ears.
4. Make a U-shaped incision down the lateral surface of the face, over the canal, ending 1–2 cm ventral to the level of the horizontal canal (Fig. 8.8).
   a. Start the incision at the level of the tragohelicine (pretragic) notch (see Fig. 1.2).
   b. Continue ventrally to a level 1–2 cm ventral to the horizontal canal.
   c. Curve the incision back up and finish at the intertragic incisure.
5. Dissect the subcutaneous tissues under the skin flap with scissors, so that the skin flap can be reflected dorsally towards the pinna.
6. Dissect the subcutaneous tissues to expose the lateral wall of the vertical canal (Fig. 8.9). Retract the parotid gland ventrally as needed.
7. To make a drain board, develop a flap in the lateral wall of the vertical ear canal with two parallel cuts:

   Dorsal to ventral cut (full ear preparation):

   a. Position yourself facing the dorsum of the dog's head.
   b. Determine the canal direction by inserting a straight blunt probe or long straight haemostats into the external ear canal.
   c. Starting at the rostral (tragohelicine) notch, cut the cartilage ventrally 1–1.5 cm with sharp scissors (Fig. 8.10).
   d. Make a similar cut starting at the second notch (intertragic incisure).
   e. Continue 1 cm cuts on either side to the level of the horizontal canal, reorienting the scissors to follow the inward spiral of the vertical canal so that the final cuts end at the rostral and caudal midpoints of the horizontal canal.

   Ventral to dorsal cut (full or lateral ear prep):

   a. Identify the ventral most portion of the lateral wall of the vertical canal.



Figure 8.10 Lateral ear canal resection. The cartilage incisions are made from dorsal (distal) to ventral in 1–1.5 cm cuts, alternating between the rostral and caudal notches, and following the gentle inward spiral of the vertical ear canal. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.11 Lateral ear canal resection. The cartilage can be cut from ventral (proximal) to dorsal by making a stab incision at the site of the final flap position and incising dorsally to the ipsilateral notch. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

b. With a number 11 or number 15 blade, make a stab incision in the lateral canal wall at the proposed rostral margin of the flap.
c. Make a stab incision in the lateral canal wall at the proposed caudal margin of the flap.
d. Insert one blade of a Mayo or cartilage scissors into the stab incision and cut the lateral canal wall from ventral to dorsal, toward the intertragic incisure, along the proposed caudal margin of the flap (Fig. 8.11).
e. Insert one blade of a sharp-sharp scissors into the rostral stab incision and cut the lateral canal wall from ventral to dorsal, toward the tragohelicine notch, along the proposed rostral margin of the flap.

8. Reflect the flap ventrally and evaluate the new ear canal opening to the horizontal canal: the opening should be round, not oval or flat, and the flap should lie flat without obstructing the canal. Make additional cuts so that the flap hinges at the level of the midpoint or ventral 1/3 of the sides of the horizontal canals and the horizontal canal opening is round (Fig. 8.12).
9. Transect the dorsal (distal) half of the lateral wall flap and remove it and the attached skin.
10. Test the flap length to determine where the skin sutures will be placed.
11. Remove additional skin as needed at the base of the incision so that the flap has mild downward tension when extended ventrally.
12. If the flap of cartilage is thick and will not lie flat, make a hinge.
    a. At the point where you want the flap to bend, dissect between the cartilage and epithelial lining with a haemostat.
    b. Transect the cartilage, leaving the epithelial lining intact.
    c. Lay the flap down; it is acceptable for the transected cartilage ends to overlap.
13. Place a skin suture at each corner of the flap (Fig. 8.13). For interrupted sutures:
    a. Take a bite of skin along the skin margin.
    b. Take a bite of epithelial lining. If epithelium is thin, the cartilage can be included in the bite.
    c. Tie the suture so that it lies off to the side, with the knot away from the incision.

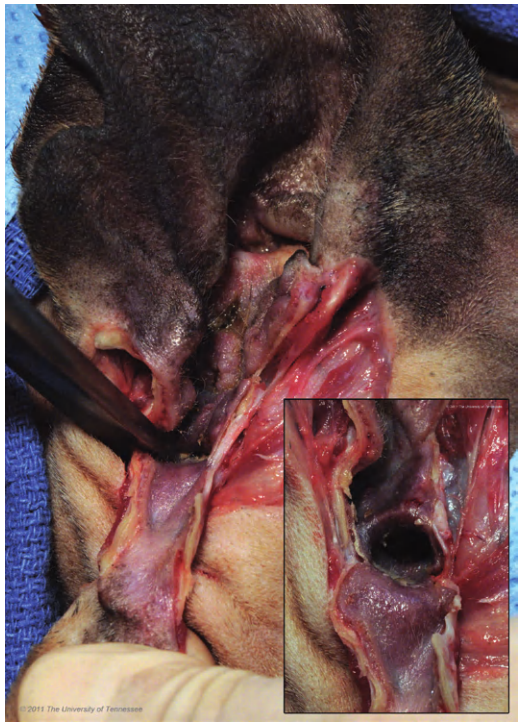


Figure 8.12 Lateral ear canal resection. Extra cuts are made cranially and caudally in the vertical canal so that the final opening is round (inset). (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

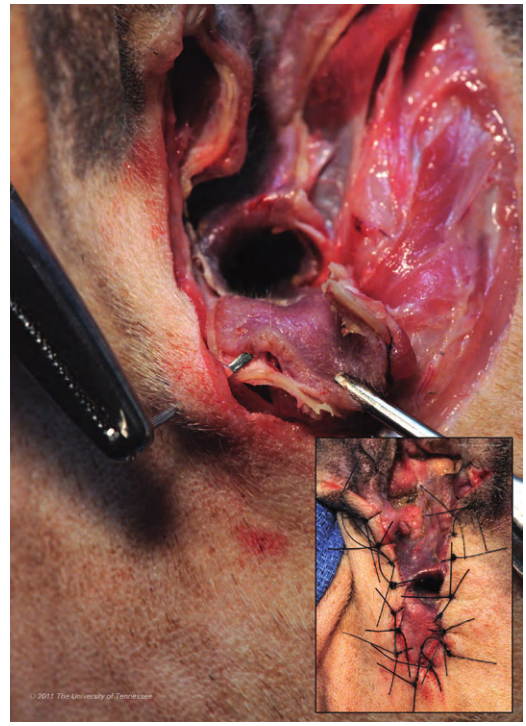


Figure 8.13 Lateral ear canal resection. The epithelial lining of the ear canal is sutured to the skin, placing corner and 'crotch' sutures first, then filling in the remaining sutures (inset). (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

Alternatively, place a vertical mattress skin suture at each corner of the flap using a 'near–near–far–far' bite pattern:

a. Take a bite of skin close to the skin margin opposite the flap, with the needle pointing toward the flap.
b. Grasp the flap with thumb forceps and pass the needle under and out the epithelial lining of the flap.
c. Pass the needle back through the lining and the cartilage of the flap, beyond and under the first bite.
d. Pass the needle under the skin margin opposite the flap and come out farther away from the first skin entrance site, passing directly under the initial suture bite.
e. Tie without excessive tension to appose skin and epithelial lining over the cartilage edge.

14. Next, place a skin suture on each side at the notch where the flap hinges, suturing to the surrounding skin margin.
15. Place the remaining skin sutures along the flap.
16. Close any remaining skin defects along the pinna margins.

8.5 VERTICAL EAR CANAL RESECTION

Vertical ear canal resection is recommended when the horizontal ear canal is normal and disease is limited to the vertical ear canal. When closing the vertical ear canal resection, it is important to make sure that there is minimal tension on the skin-to-pinna portion of the closure. It is helpful to temporarily approximate this portion of the incision to determine whether there is sufficient skin to close. More skin can be recruited by extending the vertical incision ventrally; rarely, advancement flaps are required.

1. Position the animal in lateral recumbency with the head on a towel. Perform a hanging ear preparation of pendulous ears. Flush the canal thoroughly with sterile saline and prepare the remainder of the ear with chlorhexidine.
2. Make a skin incision around the external canal opening at or below the level of the tragus, depending on the location and extent of the ear canal disease.
3. Extend the skin incision over the vertical ear canal. If needed, put a closed haemostat in the ear canal as a guide for the direction and length of the skin incision.



Figure 8.14 Vertical ear canal resection. The cartilage around the canal opening is perforated with a blade and then transected with scissors. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.15 Vertical ear canal resection. Initial dissection can be performed with a dry gauze sponge to remove fat and loose fascia. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

4. Make a stab incision through the cartilage on the dorsomedial aspect of the pinna (the concave surface of the pinna).
5. Insert one scissor blade through the stab incision (Fig. 8.14), and cut the cartilage around the external ear canal opening, following the skin incision.
6. Grasp the canal cartilage with Allis tissue forceps. Dissect through subcutaneous fat to expose the outer surface of the vertical ear canal.
7. With a dry gauze sponge, wipe off any fat or fascial attachments between the skin and vertical ear canal (Fig. 8.15).
8. Identify any muscle attachments between the vertical ear canal (auricular cartilage) and the head, and transect these at their insertion sites on the ear canal (Fig. 8.16). Transection can be performed with scissors, cautery, or laser.
9. Dissect any remaining soft tissue attachments between the vertical ear canal and head until the entire vertical ear canal is exposed and freely moveable to the level of the annular–auricular cartilage junction.
10. Transect the vertical ear canal:
    a. If the proximal vertical canal is healthy, transect it 1 cm distal to its junction with the horizontal canal.



Figure 8.16 Vertical ear canal resection. Muscles can be transected sharply at their attachments or severed with cautery or a laser. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.17 Vertical ear canal resection. If sufficient vertical canal is available, it can be cut at its rostral and caudal midpoints to make two flaps. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.18 Vertical ear canal resection. The skin is temporarily advanced from its original position (inset) to its final position. In most animals, the ventral skin incision will need to be extended or a new incision made to expose the vertical canal ends before suture apposition. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

b. If the entire vertical canal is diseased, transect it at its junction with the horizontal canal.

11. If a 1 cm vertical ear canal remnant is available, transect the remaining vertical ear canal circumference at its rostral and caudal midpoints to make dorsally and ventrally based flaps (Fig. 8.17).

    When the flaps are retracted apart, the resulting ostium should be round. If it is not round, the rostral and caudal midpoints may need to be incised deeper to reach the junction of the vertical and horizontal ear canals (see Fig. 8.12).

12. To determine the final location of the ear canal remnant, temporarily advance the skin at the rostral and caudal margins of the vertical skin incision upward toward the pinna incisional margin (Fig. 8.18) to see where the skin will lie when the surgical wound is closed.

13. With the skin temporarily advanced to its final position, position the ear canal remnant so that its opening will be level with the horizontal canal.
    a. A separate skin opening can be made ventral to the vertical skin incision and the horizontal ear canal pulled through the new incision.
    b. Alternatively, the original vertical skin incision can be extended ventrally to allow positioning of the ear canal remnant within the ventral extent of that skin incision (see Fig. 7.30B).

14. Once the ear canal remnant is in its final resting position, place two skin sutures to hold the skin in its final position along the medial (concave) pinna margin. The final skin closure will usually be T-shaped.

15. Suture the ventral flap or rim of the ear canal remnant to the skin with simple interrupted or vertical mattress sutures. If necessary, remove additional skin ventrally so that the ear canal remnant is oriented in a horizontal plane and the ventral ear canal flap, if present, is pulled in a downward direction.

    If excessive tension is present on the horizontal canal or flaps, the cartilage can be tacked to underlying subcutis with interrupted sutures of absorbable monofilament before skin closure.

16. Suture the midpoints of the rostral and caudal circumference of the ear canal rim to the skin. If flaps are present, these sutures will be placed at the rostral and caudal hinges of the flaps.

17. Suture the skin to the incision along the medial pinna margin before completing the flap or rim sutures.



Figure 8.19 Vertical ear canal resection. Final appearance after skin closure. In this case, a separate opening was made for the vertical ear canal flaps so that the final orientation of the remaining canal was maintained in a horizontal position. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

18. Suture the skin along the dorsal ear canal rim or flap and then along the vertical portion of the skin incision (Fig. 8.19).

8.6 TOTAL EAR CANAL ABLATION WITH LATERAL BULLA OSTEOTOMY

1. Follow steps 1–9 of vertical ear canal resection (section 8.5). Extend the vertical portion of the skin incision ventrally as needed so that it extends 1–3 cm ventral to the level of the horizontal ear canal.
   a. Position the animal in lateral recumbency with the head and neck on a towel and the head tilted in a slightly dorsal oblique position. Perform a hanging ear preparation of pendulous ears. Flush the canal thoroughly with sterile saline and prepare the remainder of the ear with chlorhexidine.
   b. Make an elliptical skin incision around the external canal opening at or below the level of the tragus, including all proliferative tissue, if possible.
   c. Extend the skin incision over the vertical ear canal. If needed, put a closed haemostat in the ear canal as a guide for the direction and length of the skin incision.

      In cats, an alternative is to make two diverging incisions that start at the rostral and caudal borders of the circular incision and extend ventrally (Fig. 8.20). The flap is retracted ventrally to expose the vertical ear canal. The final result, once the ear canal has been removed, is a ventrally based single pedicle flap that can be advanced dorsally during closure. Because tension is reduced, upright ear carriage is maintained after surgery.
   d. Make a stab incision through the cartilage on the dorsomedial aspect of the pinna (the concave surface of the pinna).
   e. Insert one scissor blade of a curved Mayo or cartilage scissors through the stab incision, and cut the cartilage around the external ear canal opening, following the skin incision (see Fig. 8.14).
   f. Grasp the canal cartilage with Allis tissue forceps. Using a combination of blunt and sharp technique, dissect through subcutaneous fat to expose the lateral aspect of the vertical ear canal.
   g. With a dry gauze sponge, wipe off any fat or fascial attachments between the skin and vertical ear canal (see Fig. 8.15). If possible, have an assistant retract the ear opposite to the dissection site along the canal.
   h. Identify any muscle attachments between the ear canal (auricular cartilage) and the head, and transect these at their insertion sites on the ear canal (see Fig. 8.16). Transection can be performed with scissors, cautery, or laser. To prevent pinna necrosis, avoid damage to branches of the greater auricular vasculature, especially those in the caudal half of the surgical site.

      Dissect any remaining soft tissue attachments between the vertical ear canal and head until the entire vertical ear canal is exposed and freely moveable to the level of the annular-auricular cartilage junction.
2. With Metzenbaum scissors, dissect the soft tissues immediately adjacent to the horizontal ear canal to the junction of the annular cartilage and external acoustic meatus.
   a. Retract the surrounding soft tissues with a Gelpi, Weitlaner, or circular ring retractor (Fig. 8.21).
   b. The facial nerve loops around the ventral half of the horizontal canal and may be adherent to it caudally or, occasionally, rostrally. The facial nerve is usually palpable before it is visible. Keep scissor tips immediately adjacent to cartilage and gently spread tips parallel to the likely path of the nerve.



Figure 8.20 Right total ear canal ablation and lateral bulla osteotomy in a cat. **(A)** To approach the canal, a flap is made with two divergent skin incisions. **(B)** Final appearance. Closure with an advancement flap helps maintain upright ear carriage. (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.

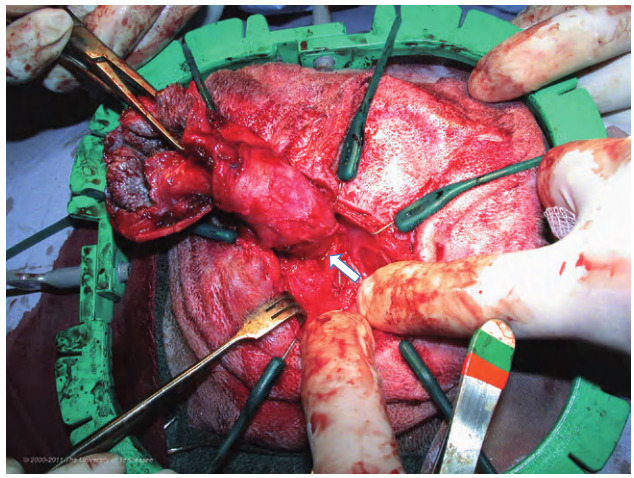


Figure 8.21 Right total ear canal ablation and lateral bulla osteotomy in a dog. Once the vertical canal is exposed, dissection should proceed cautiously along the horizontal canal to avoid damage to the facial nerve (arrow). In this dog, the facial nerve was adherent to the rostral margin of the horizontal canal. Ring retractors were used to maintain exposure. © 2012 The University of Tennessee.

   c. If the facial nerve has been incorporated in scar tissue around the canal, it may be necessary to transect the portion of the canal opposite to the facial nerve adhesions (see below) before being able to free up the nerve.

3. Once the skull is palpable dorsally and the external acoustic meatus is palpated ventrally, transect the horizontal canal attachments from the skull with curved Mayo or cartilage scissors.
   a. Start in an area away from the facial nerve or any soft tissues adhesions. Often the dorsal portion of the canal is the easiest to transect first.
   b. Make a stab incision at the cartilage–bone interface. Insert one scissor blade through the canal. Slowly advance the cut circumferentially; take small bites and only cut if the external scissor blade is visible. If possible, gently retract the facial nerve away from the scissors during canal transection (Fig. 8.22). If the nerve is difficult to avoid or visibility is limited, leave a small rim of cartilage; this rim can be removed with rongeurs or cartilage scissors once the ear canal has been removed.
   c. If the facial nerve is incorporated in soft tissue adhesions, transect the canal on the side opposite the adhesions. Then, tilt the partially transected canal

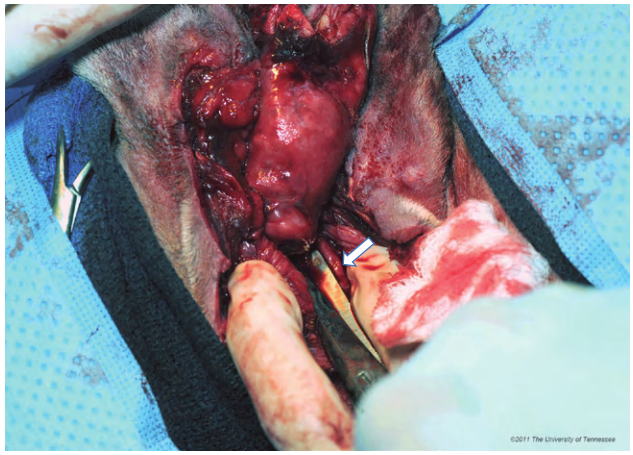


Figure 8.22 Total ear canal ablation and lateral bulla osteotomy in a dog. When transecting the horizontal ear canal, small bites should be taken with the tips of the scissors so that damage to the facial nerve (arrow) can be avoided. © 2012 The University of Tennessee.

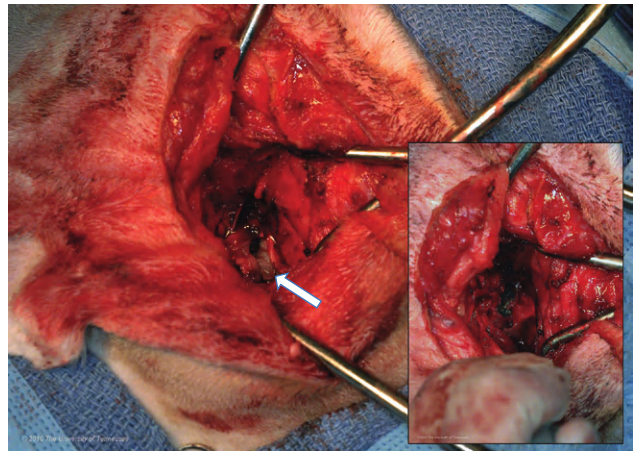


Figure 8.23 Total ear canal ablation and lateral bulla osteotomy in a dog. Cartilage remnants and epithelial lining (arrow) along the external osseous canal are removed with rongeurs to expose the rim of the external osseous canal and the lateral bulla wall (inset). © 2012 The University of Tennessee.

toward the nerve and incise the internal layers of the remaining canal through the cartilage layer. The canal can then be further retracted toward the adhesions and the remaining soft tissues transected from 'inside to outside' or gently peeled free from adhesions to avoid transecting the nerve.

4. Identify the facial nerve and branches of the external carotid artery visually or by palpation before proceeding. The facial nerve exits the stylomastoid foramen caudodorsal to the external acoustic meatus (see Fig. 1.13) and is separated from the canal by a ridge of bone.
5. Remove any cartilage remnants along the external osseous canal with scissors or Lempert or Beyer rongeurs (Fig. 8.23).
6. Remove the ventral rim of the external osseous canal with Cleveland, Zufal, or double action Ruskin rongeurs or a burr.
7. Remove the lateral wall of the bulla (Fig. 8.24) with a rongeur or a burr.
   a. Gently elevate any soft tissues from the lateral wall of the bulla with a Freer periosteal elevator. Avoid damage to the retroglenoid vein ventromedial to the bulla. The retroarticular vein is rostral to the external acoustic meatus.
   b. Insert a haemostat through the external acoustic meatus into the bulla to determine the location of the bulla and the site and direction in which the rongeur or burr must be positioned (Fig. 8.25).
   c. With Cleveland or Lempert rongeurs, remove small bites of bone from the ventral rim of the external

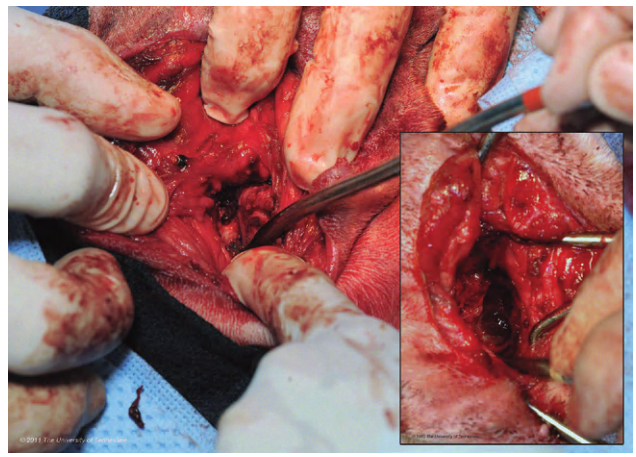


Figure 8.24 Total ear canal ablation and lateral bulla osteotomy in a dog. While the facial nerve is protected, the soft tissues covering the lateral wall of the bulla are gently elevated. The lateral wall of the bulla can then be removed with rongeurs or a burr to expose the contents (inset). © 2012 The University of Tennessee.

osseous canal; visualize jaw contents before engaging the rongeurs to remove tissue. The caudal ridge of bone can be removed with Kerrison or Lempert rongeurs.

   d. Continue ventrally directed bone removal with heavy rongeurs or a burr until the lateral surface of the bulla has been removed. Do not rongeur rostrally.



Figure 8.25 Left ventrolateral view of the canine skull. To determine the angle for removal of the lateral wall of the bulla, a curved haemostat can be inserted through the external acoustic meatus and into the bulla. Note the proximity of the stylomastoid foramen (blue arrow) caudal to the bulla, where the facial nerve exits the skull. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.26 Total ear canal ablation and lateral bulla osteotomy in a dog: final appearance. The first two skin sutures were preplaced to form the T-shaped closure before additional subcutaneous and skin sutures were added. In this dog, a continuous suction drain was placed in the subcutis because of infection in the area.

8. Gently remove any debris from the bulla with haemostats, alligator forceps, suction, or curette and culture the bulla contents. Avoid the medial portion of the dorsolateral extension (epitympanic recess) of the middle ear (see Fig. 1.14) where the round and oval windows are located.
9. Gently flush the bulla and surgical wound with warm saline and suction any residual fluid.
10. Close deeper tissues with one or two interrupted sutures of rapidly absorbable material. Avoid inclusion of the facial nerve.
11. Closure for uninfected wounds:
    a. Place two skin sutures to appose the skin along the proximal edges of the vertical incision to that of the concave surface of the pinna. Use simple interrupted or vertical mattress sutures and avoid inclusion of pinna cartilage, if possible.
    b. Once the initial skin sutures are in place, the subcutis will be in its final position. Place additional subcutaneous sutures to appose the remaining subcutis.
    c. Complete the skin closure. The final closure often appears as a T or inverted L shape (Fig. 8.26).
    d. For patients with upright ear carriage, the skin flaps can be extended, elevated, and advanced as needed to reduce tension on the pinna.
12. Closure of infected wounds:
    a. Place a continuous suction drain through healthy skin caudal to the surgical site and advance into the deeper subcutis region. Cut the drain to the appropriate length.
    b. Proceed with steps 11a–c.
    c. Secure the drain to the skin with a finger trap pattern.
13. Open wound management for infected wounds (Fig. 8.27):
    a. If desired, use a tie-over bandage to secure dressings: place suture loops along the skin incision at least 1 cm from the edge of the wound.
    b. Pack the wound with an antiseptic dressing (e.g. a honey-soaked gauze or sponge, a silver-coated alginate sponge containing dextrans, or antimicrobial impregnated gauze).
    c. Cover the dressing with an absorptive material (e.g. laparotomy sponge or gauze pads). If held in place with a tie-over bandage, use the suture loops and umbilical tape to lace the bandage in place.
    d. Cover the secondary layer with an adhesive drape, or wrap the entire head and proximal neck with cast padding and an outer wrap, being careful to avoid respiratory compromise from a tight bandage.

8.7 SUBTOTAL EAR CANAL ABLATION WITH LATERAL BULLA OSTEOTOMY

This technique is used to preserve upright ear carriage in dogs and cats with minimal to no disease of the distal



Figure 8.27 In this dog, the external ear canal was obstructed with proliferative tissue, and a periauricular abscess developed after rupture of the fascia connecting the vertical and horizontal ear canals. Total ear canal ablation and lateral bulla osteotomy were performed, and the wound was managed with daily bandage changes until a healthy granulation bed formed.

(dorsal) vertical ear canal. The remaining vertical canal exists as a blind-ended pouch; therefore, the technique is not suitable for animals with gross disease of the distal canal because of the risk of recurrence of clinical signs.

1. Make a vertically oriented skin incision over the vertical ear canal from the midpoint of the external orifice to a point ventral to the horizontal ear canal.
2. With a combination of blunt and sharp dissection, free the ventral two thirds of the vertical ear canal from its soft tissue attachments (Fig. 8.28).
3. Transect the vertical ear canal near its midpoint (Fig. 8.29).
4. Appose the cartilage of the medial (axial) and lateral (abaxial) walls of the distal (dorsal) vertical ear canal remnant with an interrupted or continuous pattern of absorbable monofilament suture material, taking care to avoid penetrating canal epithelium with the suture (Fig. 8.30).
5. Follow steps 2–9 of total ear canal ablation and lateral bulla osteotomy as described above (section 8.6).
6. Close subcutis and skin routinely (Fig. 8.31).

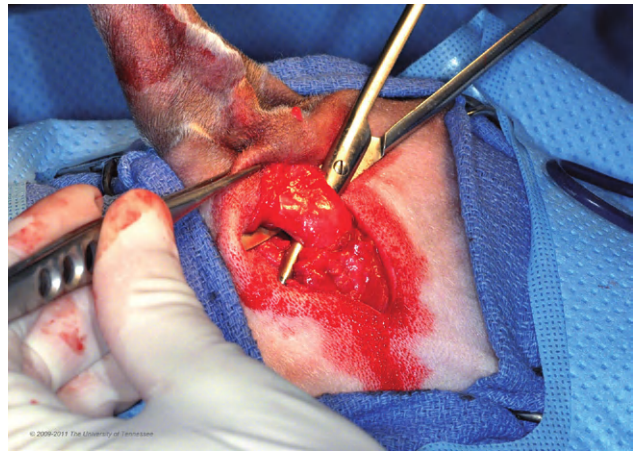


Figure 8.28 Subtotal ear canal ablation and lateral bulla osteotomy. The vertical ear canal is isolated with a combination of blunt and sharp dissection. © 2012 The University of Tennessee.

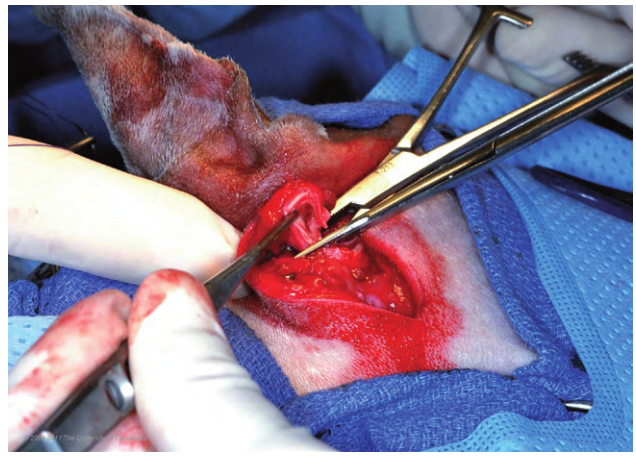


Figure 8.29 Subtotal ear canal ablation and lateral bulla osteotomy. The vertical ear canal is transected near its midpoint. © 2012 The University of Tennessee.

8.8 END-TO-END ANASTOMOSIS

8.8.1 Lateral Approach

This technique has been used for patients with congenital stenosis or stenosis secondary to trauma or other causes. It is used when the stenotic region is within the central portion of the external ear canal. Stenosis of the distal portion of the ear canal (near the normal auricular opening) can be treated with a pull through technique similar to a vertical ear canal ablation. Stenosis associated with periaural abscessation will likely require total ear canal ablation and bulla osteotomy.

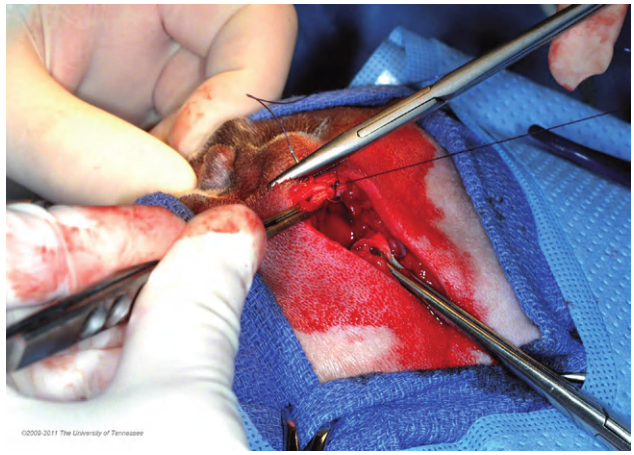


Figure 8.30 Subtotal ear canal ablation and lateral bulla osteotomy. The distal (dorsal) end of the transected vertical canal is sutured closed with a partial thickness, continuous pattern of absorbable monofilament. © 2012 The University of Tennessee.



Figure 8.31 Subtotal ear canal ablation and lateral bulla osteotomy. Final appearance after closure. © 2012 The University of Tennessee.

1. Perform a hanging ear preparation.
2. Incise the skin over the vertical ear canal from the tragus to a point ventral to the level of the horizontal canal.
3. At the level of the stenosis, dissect the ear canal free from its soft tissue attachments with a combination of blunt and sharp dissection.
4. Stabilize the canal proximal and distal to the stenosis with stay sutures. Transect the stenotic region and remove any tissue sealing the blind ends of the canals to produce a patent lumen for each segment. Flush and suction the area as needed to remove debris.



Figure 8.32 Traumatic canal transection in a dog. (Courtesy Dr Mark Bohling, UTCVM) © 2012 The University of Tennessee.

5. Appose the proximal and distal segments with interrupted sutures of 3-0 or 4-0 absorbable monofilament, starting at the medial wall. Sutures should engage cartilage but can exclude epithelium.
   If the transected ends are difficult to expose and visualize, preplace at least four sutures around the circumference of the canal before tying them. Add additional interrupted sutures as needed to complete the anastomosis.
6. Flush and culture the region as needed, then close the subcutis and skin routinely.

8.8.2 Caudal Approach

This technique can be used for acute separation of the ear canal between the annular and auricular cartilages (Fig. 8.32) in animals that do not have periauricular abscessation or otitis media.

1. Perform a hanging ear preparation.
2. Make a vertically oriented curvilinear incision through the skin caudal to the pinna and ear canal.
3. Sharply and bluntly separate the subcutaneous tissues to expose the separated canal ends. Transect the cervicoauricularis and parotidoauricularis muscles as needed. To facilitate identification of the distal canal segment, insert a probe through the external acoustic meatus.
4. If needed, place stay sutures on the proximal and distal segments.
5. Debride the canal ends to remove any fibrous tissue and to create a wide open lumen.
6. Appose the proximal and distal ends of the separated canal with full thickness interrupted sutures of monofilament absorbable material, placing the knots on the outside of the canals (Fig. 8.33).

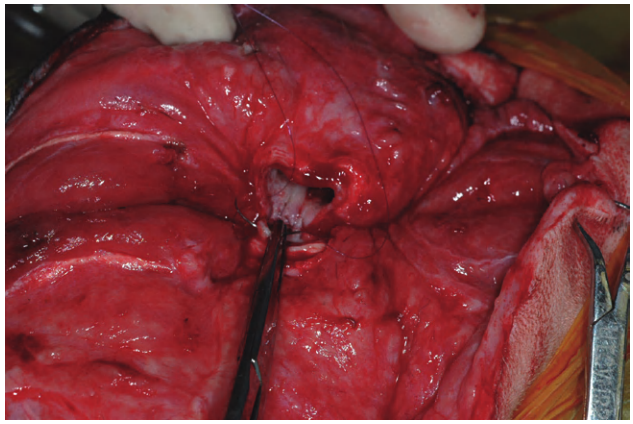


Figure 8.33 End-to-end anastomosis. After the wound is debrided and cleansed, the ends of the transected canal are reapposed medially. (Courtesy Dr Mark Bohling, UTCVM) © 2012 The University of Tennessee.

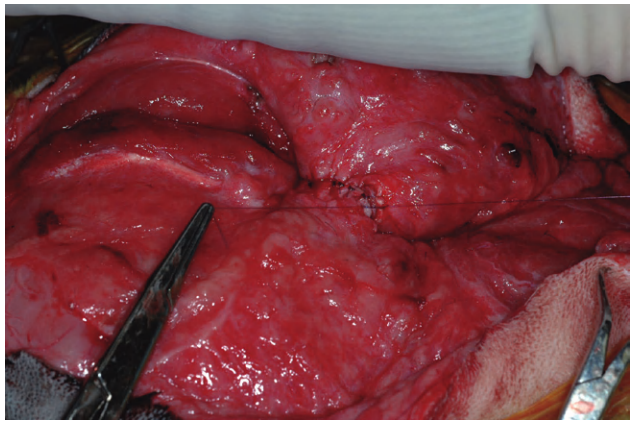


Figure 8.34 End-to-end anastomosis. Additional sutures are added to fill in the gaps. (Courtesy Dr Mark Bohling, UTCVM) © 2012 The University of Tennessee.

If apposition of the medial canal edges interferes with visualization, preplace sutures circumferentially. Once all sutures are in place, tie them to appose the canal ends.

7. Add additional sutures as needed (Fig. 8.34), then lavage and culture the wound.
8. If necessary, place a drain before closing the muscle, subcutaneous tissues, and skin routinely.

8.9 VENTRAL BULLA OSTEOTOMY

8.9.1 In the Dog

1. Clip and prepare the ventral cervical and intermandibular regions. Before the final preparation, position the dog in dorsal recumbency with the head and neck perfectly straight and the front limbs pulled caudally.
2. Make a paramedian incision over the region of the bulla, starting medial to the angle of the mandible and extending 1 cm caudal to the level of the external ear canal. The incision is centred midway between the ramus of the mandible and the wing of the atlas.
3. With a combination of sharp and blunt dissection, separate or transect the subcutaneous tissues, vertically oriented fibres of the platysma, and horizontally oriented fibres of the sphincter colli muscles. During dissection, open the instrument tips in a direction parallel to the incision line to reduce the risk of damage to tributaries of the linguofacial vein.
4. Retract the soft tissues with Gelpi or Weitlaner retractors.
5. Palpate the jugular process, a prominent bony spike caudal to the bulla that serves as the attachment for the digastricus muscle (see Figure 1.13). The bulla will be approximately 5 mm rostral and 5 mm medial to this boney prominence and dorsal to the digastricus muscle.
6. Bluntly dissect between the styloglossus and hyoglossus muscles medially and the digastricus muscle laterally.
   a. If the salivary glands are in the surgical field, retract them laterally.
   b. The myohyoideus muscle lies superficial to the styloglossus and hyoglossus muscles and medial to the digastricus. Fibres of this muscle may need to be retracted cranially, separated, or transected along its caudal edge (Fig. 8.35) to expose the underlying muscles. The lingual vein runs deep to the mylohyoideus muscle.
   c. During dissection, open the scissors parallel to the long axis of the head to avoid damage to the hypoglossal nerve, which lies on the lateral surface of the hyoglossal muscle and the medial surface of the styloglossus muscle, and the lingual artery, which lies dorsal to the hypoglossal nerve.
7. Retract the digastricus muscle laterally and the styloglossus and hyoglossus muscles and hypoglossal nerve medially with Gelpi or Weitlaner retractors to expose the jugulohyoideus muscle over the ventral surface of the bulla.

Important structures in the area include the facial nerve over the lateral surface, the small glossopharyngeal nerve over the ventral surface, and the vagus nerve over the ventromedial surface of the bulla. In some animals the external carotid artery and maxillary vein, or their branches or tributaries, are visible deep within the surgical field.

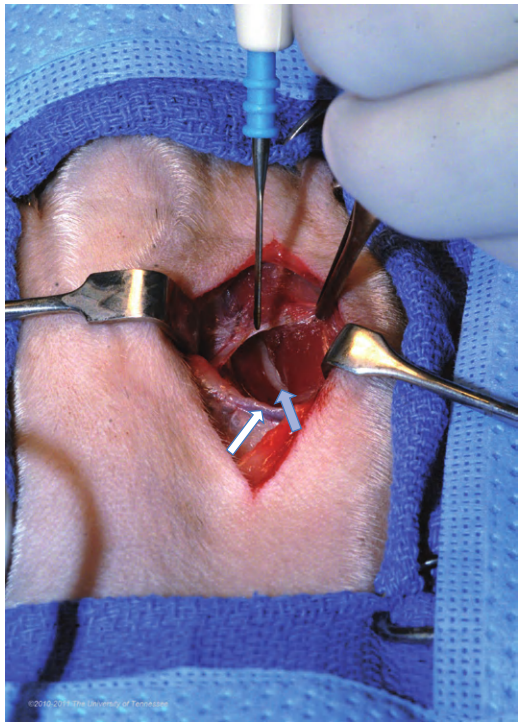


Figure 8.35 Left ventral bulla osteotomy in a dog. The mylohyoideus muscle is separated from the digastricus muscle with sharp dissection or cautery to expose the underlying styloglossus and hypoglassus muscles and the hypoglossal nerve (blue arrow), which lies between them. The hyoid venous arch (white arrow) is visible at the caudal extent of the incision. © 2012 The University of Tennessee.

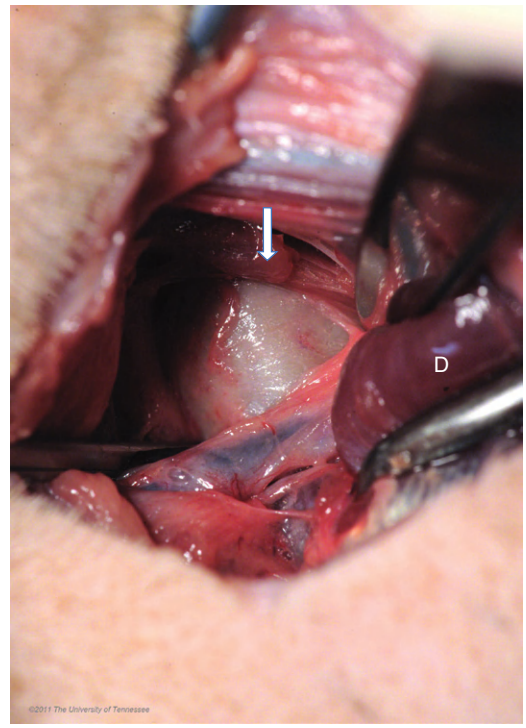


Figure 8.36 Left ventral bulla osteotomy in a dog. The jugulohyoideus muscle (arrow) has been incised and elevated with a periosteal elevator to expose the bulla. The digastricus (D) and stylogossus muscles have been retracted laterally and medially, respectively, with Gelpi retractors. © 2012 The University of Tennessee.

8. Separate the jugulohyoideus muscle fibres with scissors, haemostats, or periosteal elevator and elevate the muscle off of the bulla (Fig. 8.36).
9. With a burr or 1/8th or 3/16th intramedullary pin, make a hole in the ventral surface of the bulla. If hearing is to be preserved, use burrs sparingly since their vibrations may disturb the cochlear apparatus.
10. With rongeurs, extend the ventral bulla opening so that it is 7–10 mm in length (Fig. 8.37).
11. Curette the ventral, medial, and lateral portions of the bulla. Avoid the dorsal lateral aspect of the chamber (the epitympanic recess), which contains the entrances to the inner ear.
12. Culture the bulla contents, then gently lavage the bulla with warm saline and suction clean.
13. If there is extensive infection, place a drain. Otherwise, close the subcutaneous tissues and skin routinely.

8.9.2 In the Cat

1. Clip and prep the intermandibular and proximal ventral cervical regions and over the ventrolateral surface of the vertical ear canal. Position the cat in dorsal recumbency with the head and neck extended and perfectly straight before the final preparation.
2. Palpate the region of the bulla, which is midway between the angle of the mandible and the wing of the atlas. Make a skin incision over the bulla in a rostral–caudal direction.
3. With a combination of blunt and sharp dissection, separate the subcutaneous tissues over the bulla. Extend the incision through the platysma and sphincter colli muscles.
   Identify and retract tributaries of the linguofacial vein. In some cats, the hyoid venous arch may need to be ligated and transected
4. Palpate the bulla again to identify its location; the typanohyoid cartilage, which extends off the stylohoid bone of the hyoid apparatus, rests on the ventral surface of the bulla.

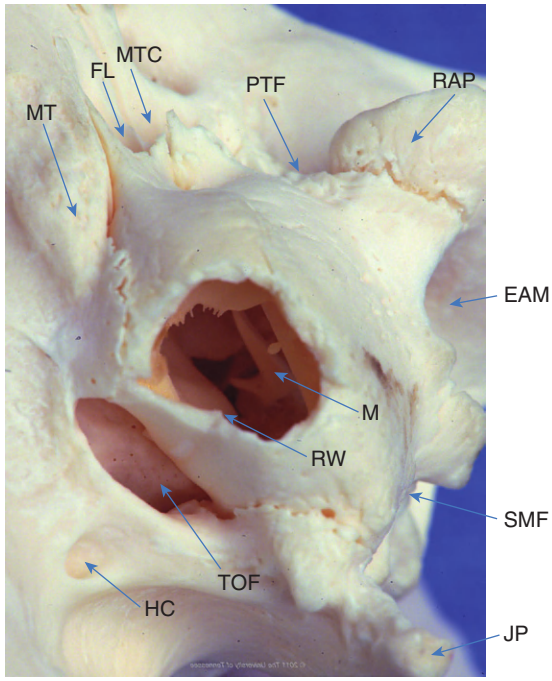


Figure 8.37 Left ventral bulla osteotomy in a dog. The ventral bulla wall has been opened with a Steinmann pin and rongeurs. Local structures include EAM, external acoustic meatus; FL, foramen lacerum; HC, hypoglossal canal; JP, jugular process; M, malleus; MT, muscular tubercle; MTC, musculotubal canal, RAP, retroarticular process; RW, round window; SMF, stylomastoid foramen; TOF, tympano-occipital fissure. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.38 Left ventral bulla osteotomy in a cat. Rostral is to the top of the photo. The platysma and sphincter coli have been transected and retracted to expose the hypoglossal nerve (arrow), which lies medial to the hyoglossal muscle. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

5. Separate and retract the digastricus muscle laterally and the styloglossus and hyoglossus muscles medially. The hypoglossal nerve is medial to the hyoglossal muscle (Fig. 8.38).
6. Retract the hypoglossal nerve and lingual artery medially and the digastricus laterally with self-retaining or Senn retractors to expose the jugulohyoideus muscle on the ventral surface of the bulla.
7. With a 1/8 or 3/16 inch (3 or 5 mm) Steinmann pin or burr, make a hole in the ventral surface of the bulla (Fig. 8.39).
8. Extend the ventral bulla opening with Kerrison or Lempert rongeurs or a burr until it is 7–10 mm in diameter. Vibrations from a burr may damage the cochlear apparatus.
9. Perforate the lateral most aspect of the septum between the large ventromedial compartment and the smaller dorsolateral compartment of the bulla. The septum is along the dorsolateral aspect of the ventral compartment and is usually convex (Fig. 8.40). Often it can be penetrated with the closed tips of a haemostatic forceps.
10. Gently extract pieces of the septum with haemostatic or alligator forceps until the dorsolateral compartment can be visualized. To prevent damage to sympathetic fibres, avoid dissection around the promontory (see Fig. 1.28).
11. Gently remove any debris or tissue with suction or forceps. Curettage is usually not required and is likely to damage the sympathetic fibres. In some cats, middle ear polyp extraction with an alligator forceps can be performed from the external ear canal by a non-sterile assistant while the surgeon manipulates the tissues through the bulla.
12. Drain placement is usually not required. Close subcutaneous tissues and skin routinely.

8.10 LASER EAR SURGERY

Laser surgery is recommended for dissection of vascular tissues. Lasers may produce a dry field compared with scalpel and scissor dissection, reduce swelling by sealing

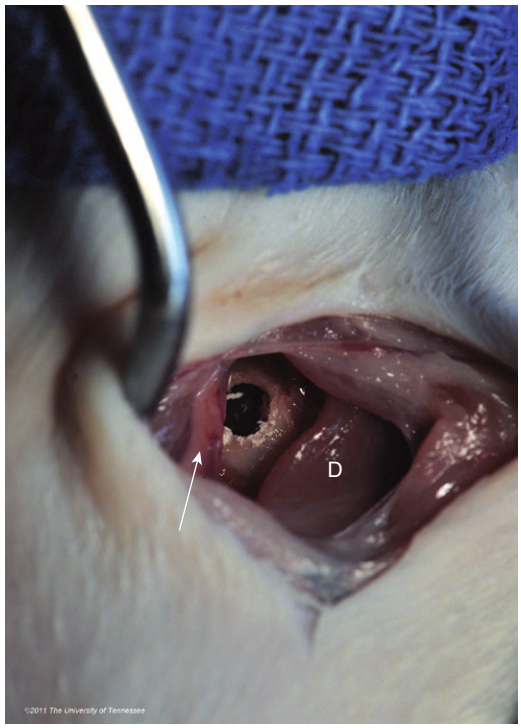


Figure 8.39 Left ventral bulla osteotomy in a cat. The ventral bulla has been perforated with a Steinmann pin and the contents gently removed with suction. The hypoglossal nerve (arrow) is medial and the digastricus muscle (D) is lateral. The inner septum is barely visible within the lateral aspect of the bulla. (Photo by Phil Snow, UTCVM) © 2012 The University of Tennessee.

lymphatics, and decrease postoperative pain. Use of the laser may increase surgical time because incisions must be made slowly. When used improperly, lasers may superheat tissues, causing peripheral (collateral) damage. Lasers do not coagulate vessels greater than 1 mm in diameter and do not provide haemostasis in areas with active blood flow. Proper safety equipment includes safety glasses, smoke evacuators, laser-safe surgical masks, and door signs to indicate that a laser is in use (Figs 8.41, 8.42). The surgeon or technician should be prepared for accidental fires initiated by heat production. The patient's eyes and endotracheal tube should be covered, and use of flammable solutions (e.g. alcohol) near the surgery site should be avoided.

8.10.1 Preparation before Laser Surgery of the Pinna

1. Under anaesthesia, evaluate the ears for otitis.
2. Place cotton in the ear canal, and clip both sides of the pinna.
3. Perform a surgical preparation with chlorhexidine scrub. Avoid alcohol, which can be ignited by the laser beam.
4. Cover the patient's eyes with moistened gauze sponges or protective goggles, and wrap the endotracheal tube with a moistened towel or sponge to prevent corneal damage and tube ignition, respectively.

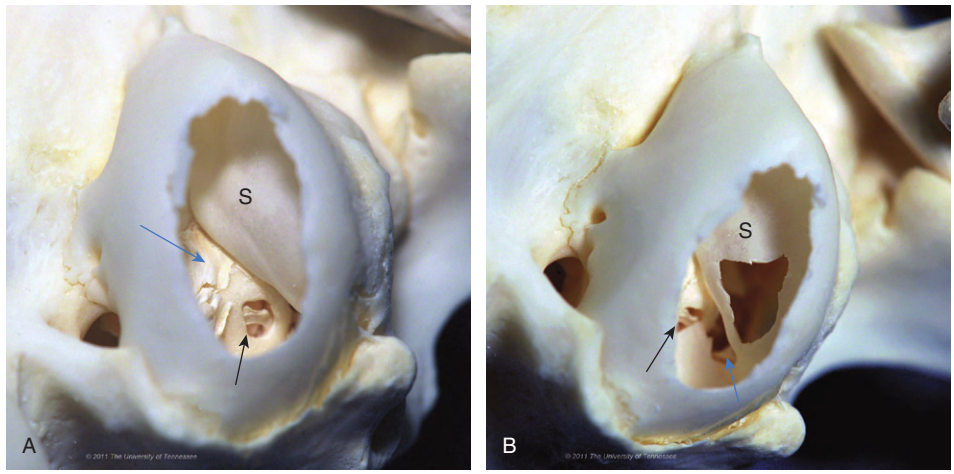


Figure 8.40 **(A)** The septum (S), which lies rostrolaterally within the tympanic bulla, separates the larger ventral compartment of the bulla from the smaller dorsal compartment where the tympanic membrane lies. The round window (black arrow), which faces laterally, is just medial to the fissure connecting the two compartments of the bulla and is lateral to the promontory (blue arrow). **(B)** The inner septum has been opened to expose the dorsolateral compartment of the bulla. The round window (black arrow) and fissure (green arrow) are visible just dorsal to the septum (S). (Photos by Phil Snow, UTCVM) © 2012 The University of Tennessee.



Figure 8.41 Laser surgery is performed in a room with appropriate ventilation. A sign should be placed on the closed door of the room when the laser is in use.



Figure 8.42 Personnel within the laser suite must wear appropriate safety glasses and masks or respirators.

8.10.2 Auricular Haematoma

Laser Fenestration Method

1. Outline the margin of the haematoma with a sterile marker. Set the CO_2 laser at 8–12 W of power, continuous mode, and use a 0.8 mm ceramic tip. Hold the tip 1–3 mm from the tissue surface when incising.
2. At the apex of the haematoma (near the pinna tip), make a 1 cm circular incision through the skin of the concave surface of the pinna into the haematoma cavity.
3. Remove the circle of skin and any attached cartilage and drain the haematoma.
4. Make multiple 1–2 mm diameter circular perforations through the concave surface of the ear into the haematoma cavity. These small fenestrations should penetrate through the skin on the inner surface of the pinna and all layers of cartilage and into the skin on the exterior of the pinna. Fenestrations should be 5–10 mm apart and distributed widely over the surface of haematoma and just beyond its margins. Flip the ear over frequently during fenestration to verify depth of the laser incisions.
5. Remove the sponges from the ears and cleanse the canals with an otic cleansing solution. Position the ear flat against the dorsum of the head, and cover the exposed inner surface with a non-adherent pad. If otitis is present, bandage the ear with the external ear canal opening uncovered to allow access as needed for topical treatment.

Laser and Suture Method

1. Make three or more circular, 0.3–0.7 cm partial thickness skin incisions in a triangular pattern into the concave surface of the haematoma.
2. Increase the depth of one of the proximal incisions into the haematoma cavity, and drain the fluid from the haematoma.
3. Continue the remaining incisions full thickness. Remove any fibrin clots and, if necessary, flush the haematoma cavity gently with sterile saline.

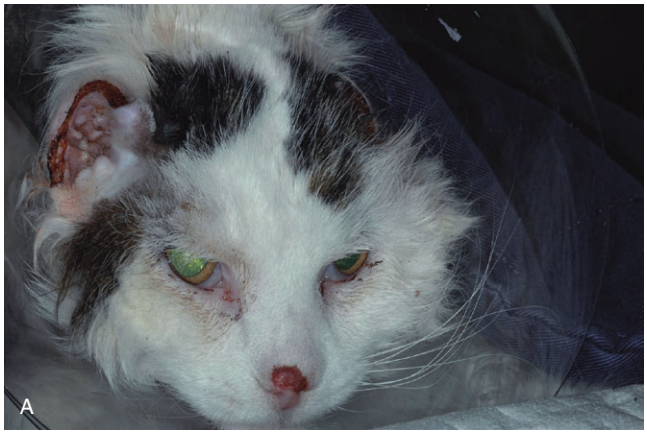
4. Suture the skin to the underlying cartilage with stented Prolene or nylon (0 to 3-0) suture on a taper or taper cut needle. Make the long axis of the mattress sutures parallel to the long axis of the ear.

8.10.3 Masses of the External Ear Canal or Pinna

1. Ablate papillomas and other benign masses with a CO_2 laser set on 8–15 W and a large focal size (≥0.5 mm). Ablate the tissue with multiple passes, removing any char between each pass. Decrease the power density or defocus the laser to provide haemostasis (Fig. 8.43).
2. Perform sharp excision (Fig. 8.44) with the CO_2 laser set at 7–9 W of continuous power using a tip size of 0.4 mm. Decrease the power density or defocus the laser to provide haemostasis.



Figure 8.43 The tissue is ablated with multiple passes of the laser; char is removed between each pass. The patient's eyes are protected with safety goggles or a damp towel during the procedure.



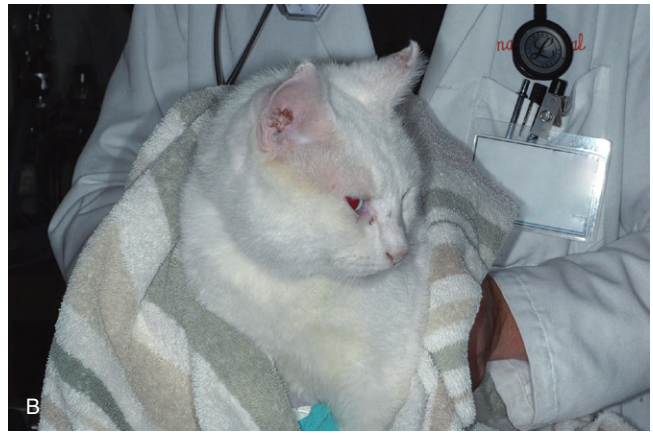


Figure 8.44 Partial pinnectomy for squamous cell carcinoma. Laser pinnectomy reduces postoperative haemorrhage (**A**). Despite second intention healing, cosmetic appearance is excellent in most cats (**B**).

VetBooks.ir

VetBooks.ir

INDEX

Numbers in **bold** indicate Figures and Tables

abiotrophic inherited deafness 139
abscesses **4**, **45**, 62, **139**, **158**, 158
acanthocytes 28, 29, **30**
actinic change 58
actinomyces 70
adenitis, sebaceous 28, 59, **87**, 90
adenocarcinomas **74**, **75**, **94**, 113
  ceruminous glands **94**, 96, **97**, **135**, **138**
adenomas 84, 113
  ceruminous glands **94**, **95**, **96**
  sebaceous glands **74**, **94**, 94, **95**, 96
adrenocorticotropic hormone (ACTH) 84
Akitas 59, **59**, 90, 68, 132
Alaskan Malamutes 61
allergic contact dermatitis 51, 53, 54
allergy **23**, 51–4, 56, 87, **88**
alopecia **23**, 24, **51**, 64–5, 81–5
  colour dilute **82**, 82
  congenital 83
  crust and scale 55, 57
alopecia areata 82
altered mentation 127, 129
alternaria 70
amikacin 131, **132**, **140**, 140
aminoglycosides **35**, 131, 140–1
ampulla 18, **19**, **20**
anagen hairs 28, **28**, 82
anisocoria **117**
annular cartilage 2, **3–4**, 4, **17**, 154, 159
annulus fibrocartilaginous 8
anthelmintics 78
antibiotics **35**, 78, 98
antihelix **2**, 2
antitragus 1, **2**, **3**
apex 1, **1**, 146, 147
arteries 2, 4, **6**, **11**
  internal carotid **6**, 10–11, 13
arthosis 140
ascending infection 107, 115
ataxia 125, **127**, **128**, 128, 130
atopy 51, 52–3, 63, 87
auditory canal *see* ear canal
auditory tube *see* Eustachian tube
auditory tympanometry 37, 49, 122, 141
aural haematoma 52, **53**
auricular cartilage 2, **3–4**, 4, 154, 159
auricular chondrosis 54
auricular haematoma 146–8, 152, 164
auriculotemporal nerve 5, **10**, 11
autoimmune disease 51, 68, 72, 75, 77, **87**, 90
  pemphigus foliaceus **87**, 90, **91**

bacteria and bacterial infections 61, 67, 100, 102
  alopecia 83
  diagnosis 25, 28, 29, 33, **34**, 35
  otitis externa 100
  otitis media 108
  otitis media interna 130
  pyogranulomas 70
balance 15, 18, **19–20**, 125–36
basal cell tumour **73**, **94**
basilar membrane 20, **21**, 136, **137**, 140
Bassetts 56, **97**
Beagles 57, **74**, 132, **138**, **139**
Bearded Collies 57, 68
Bernese Mountain Dogs 72
bilateral vestibular dysfunction 128
biopsy **23**, 31, 35, 58, 59, 60, 62
  pinch 35, **36**, 37
  punch 31, 148
blood supply 2, 4, **6**
bone vibrator or stimulator 47, 49
bony anomalies 132, **133**
bony labyrinth 15, **19**
bordetella 115
Boston Terriers **74**, 81
botfly 132
botryomycosis 70
Boxers 71, 72, **74**, 85
brainstem 125, **126**, 136, **137**
  deafness 138, 142
brainstem auditory evoked responses (BAER) 37, 45, 46–7, **48**, 49
  deafness 46–7, **48**, 49, 141–3
  PSOM 111, **112**
Bull Terriers **74**
bulla osteitis 112
bulla osteotomy **138**
  lateral 154–8, **158**, **159**
  see also ventral bulla osteotomy
Bulldogs **74**
bullous pemphigoid 75, **76**, 77
Burmese cats 132

candida yeast 33, 56
canine familial dermatomyositis 78–9
canine juvenile cellulitis 69–70, **70**
capillary ooze **26**
carbamide peroxide 131, 141
carcinoma **73**
  see also squamous cell carcinoma
caroticotympanic nerve 13
carotid canal 11
caudal auricular artery 2, **6**, **11**
Cavalier King Charles Spaniels **59**, 107, 139
  PSOM 110, **111**
cerebellum 125, **126**, 128
ceruminoliths 37, **87**, 92–3, **93**, 107, 108, **109**
ceruminous glands 2
  adenocarcinoma **94**, 96, **97**, **135**, **138**
  adenomas **94**, **95**, **96**
  hyperplasia 103, **103**
ceruminous otitis 61
cervicoauricularis muscle **5**, 159
Chesapeake Bay Retrievers 131
Chihuahuas 81
chinchillas **18**
Chinese Crested Dogs 83
chloramphenicol **140**, 141
chlorhexidine 131, **132**, **140**, 163
cholesteatoma 107, 109, **109**, **110**, 118
chorda tympani 10–11, 13
Chow Chows 68
chronic otitis externa **35**, 140, 148
chronic otitis media 115, **116**
ciclosporin **71**, 71
cilia (hair cells) 18, **20**, **21**
circling 125, **127**, 128
cisplatin **140**, 141
cladosporium 70
closed-suction drain 146, 147–8
cochlea 9, 15, 18, **20**, 136, **137**, 138–43
  BAER 47
  chinchilla **18**
  dog **17**
cochlear aqueduct 15, **19**
cochlear duct 15, 18, 20, 136, 138
cochlear nerve 18, **20**, **21**, 21, **137**
  deafness 138, 140
cochlear window *see* round (cochlear) window
Cocker Spaniels **74**, **75**, 90, 103, 132, 148
  crust and scale 58, 59, **60**
cold agglutinin disease (CAD) 78
Collies 71, 75, **76**
colour mutant alopecia/follicular dysplasia **28**, 28

Atlas of Ear Diseases of the Dog and Cat, First Edition. Sue Paterson and Karen Tobias.
© 2013 Sue Paterson and Karen Tobias. Published 2013 by Blackwell Publishing Ltd.

VetBooks.ir

comedones 26, 51, 83–5
common carotid artery **6**, **11**
computed tomography (CT) **15**, **17**, 23, 37, 38, 43–5, **45**
  chinchilla **18**
  cholesteatoma 109, **110**
  chronic otitis media 115, **116**, **117**
  middle ear 123
  middle ear growths 113, **114**
  otitis interna **130**
  otitis media **104**, **108**, **110**
  otolith **110**, 110
  peripheral vestibular dysfunction **130–1**, **133**, 134–5, **135**
  PSOM 112, **112**
  stenosis of ear canal **105**
  tympanic membrane 118, 122
concha and conchal tube 2, **4**
conductive hearing loss 47, 49, **112**, 138, 139–40, 143
congenital alopecia 83
congenital vestibular disorders 132, **133**
contact allergy or hypersensitivity 87, 99
contrast canalography 122
corticosteroids 146
cotton tip applicators 99
cotton wool 99, **100**
cranial nerve 125, **127**, 128
crista ampullaris 18, **20**
crust and scale **23**, 29, **51**, 55–62, **63**
cryptococcosis 70, 132
Cryptococcus neoformans 70–1
Ctenocephalides felis 65
cultures **23**, 31–6, 56
cupula 18, **20**
Cushing's disease **84**, **85**, 85
cutaneous adverse food reactions (CAFR) 53–4
cutaneous histiocytoma **74**
cutaneous histiocytosis 72
cutaneous lupus erythematosus 60, 72, 75, **76**
cutaneous marginal pouch 1, 2
Cuterebra (botfly) 132
cyclosporine **71**, 71
cytology **23**, 28–30, 33–5, 36, 56

Dachshunds 59, 70, 77, 81, 85
Dalmatians **74**, 139, 141
deafness 111, 138–43
  abiotrophic inherited 139
  BAER 46–7, **48**, 49, 141–3
  inherited 138, **139**, 139–40, 143
  sensorineural 47, 49, 138, 139–40, 141, 143
  vestibular disease **127**, 129, 132
Demodex spp 83, **87**, 89, **89**, **90**
  diagnosis **23**, 25–6, 28–29, **30**, 33, 89, **89–90**
Demodex canis 26, **33**, 83, **87**, 89
Demodex cati 83, **87**
Demodex corniei **25**, 25, 83
Demodex gatoi 25, 83, 89
demodicosis 28, 29, 83–4, 88, **89**
Demox felis 26
dermatitis 51, 53, 54, **54**, 56, 60–1, 62
Dermatophagoides spp 63
dermatophyte culture **23**, 27, 31–2
dermatophyte ectothrix 27
dermatophytosis **23**, 55–6
descending infection 107, 108–10
diagnosis 23–36
  advanced techniques 37–50
  alopecia 81, 82, 83, 84, 85
  crust and scale 55–62
  deafness 141–3
  demodex **23**, 25–6, 28–29, **30**, 33, 89, **89–90**
  ear canal 91, 94, 96, 97
  erythematous disease 53, 54, 55
  nodular disease 71, 72
  otitis media 109, 111, 113, 114
  papular disease 63, 65–7
  pustular disease 68, 69, 70
  ulcerative disease 75, 77–80
  vestibular dysfunction 133–6
diascopy 23, **23**, **24**
Diff-Quik **24**, 25, 29, 33
digastricus muscle **10**, 160, **161**, 162, **163**
dioctyl sodium sulfosuccinate 131, 141
direct bone conduction hearing 136, **137**
discoid lupus erythematosus (DLE) 72, 75, **76**, **87**
Dobermann Pinschers 57, 68, 81, 132
drug eruption 78

ear canal 1–5, **8**, 41–2, **44**, 87–106, 107
  allergy **87**, 87, **88**
  auditory tympanometry 49
  autoimmune disease **87**, 90
  calcification **104**
  chronic change 100, 103–6
  contact dermatitis **54**
  crust and scale 58
  CT 44, **45**
  ectoparasites **87**, 88–9, **90**
  endocrine disease **87**, 87–8
  end-to-end anastomosis 158–60
  foreign bodies **87**, 91–3
  hyperplastic disease **87**, 93–7
  idiopathic disease **87**, 90–1
  keratinization disorders **87**, 90
  lateral wall resection 148, **149**
  mineralization 41, 44, 46
  MRI 46
  neoplastic disease **87**, 93–7
  perpetuating factors 100–6
  predisposing factors 97–100
  radiography 38, **39**, 41–2, **43**, **44**
  Staphylococcal infection 67
  stenosis **105**, 106, 148, 158–8
  subtotal ablation 157–8, **159**
  total ablation **138**, 154–8, **158**
  video otoscopy 37–8
ear cleaners 98–9
ear drops **35**, **54**
ear margin seborrhoea 59–60
ear wax 32–5
ectoparasites **23**, 24–5, 51, 53, **87**, 88–9, **90**
  papular diseases 63–7
elliptical biopsy 31
end-to-end anastomosis 158–60
endocrine disease **23**, 27, 28, 51, 56, 57–8
  ear canal **87**, 87–8
endolymph 15, 18, **19**, **20**, 20–1, 136
  deafness 139, 141
endolymphatic sac 15, **19**
English Springer Spaniels 62
enophthalmos 14, **117**, 129, **129**
enterococcus 130
Enterococcus faecalis 33, **34**, **35**, 35, 98–9
entry hairs 119, **121**
environmental disease 51, 58, 77
eosinophilic granuloma syndrome 53
eosinophils 29
epithelioma **73**, **74**
epitheliotropic lymphoma (EL) 62, 70, **81**
epitympanic recess 6–9, **10**, 11, **12**, 157
erosions 29, 31, 54
erythema **23**, 23, 87, **88**
erythema multiforme complex (EM) 79–80
erythematous disease **51**, 51–5
Escherichia coli 130
ethacrynic acid 141
Eustachian tube 9–10, 12–14, **16**, **17**
  cholesteatoma 109
  nasopharyngeal polyp 113, **114**
  otitis media 107, 110, 115
  PSOM 110
Eutrombicula alfredugesi 66
excisional biopsy 31
exfoliative cutaneous lupus erythematosus 60
exfoliative dermatitis 56
external acoustic meatus 2, **7**, 8, **10**, **14**, 136, 138
  total ear canal ablation 155, 156
external acoustic meatus nerve 11
external carotid artery 4, **6**, 11
external ear 1–5, 21, 113
external ear canal 23, 32–5, 136, **137**, 165
  deafness 138, **138**, **139**, 143

facial nerve 2, 4–5, **6**, 9–10, 13, **117**, **118**
  clinical signs of damage **117**
  PSOM **111**, 111
  total ear canal ablation 154–7
  vestibular disease 125, **128**, 128–9, 132
Felicola subrostratus **26**, **64**, 64
feline immunodeficiency virus (FIV) 55, 71, 83, 100
feline leukaemia virus (FeLV) 55, 71, 83, 100
feline pox virus (cowpox) 80
feline relapsing polychondritis 54–5
feline sarcoma virus (FSV) **73**
fibromas **94**, 113
fibrosarcoma **73**
fibrosis and fibrotic change **104**, **105**, 106
fine-bore needles 37–8, **38**
  aspirates **23**, 29–30, 35
fleas 51, 65–6
fluorescein stain, dilute 122
fluoroquinolones **35**
flushing catheters 37–8, **38**
fly-bite dermatosis 66–7
follicular dysplasia 81–2
follicular dystrophy 28
food allergy 51, 53–4, 87
footpad hyperkeratosis 68
foramen lacerum 11
foreign bodies **87**, 91–3, 107, 108

foxes 63
fractures 132
French Bulldogs **133**
frostbite 77
fungal granulomas 70
fungal infections 26, 28, 31–2, 56
furosemide **140**
furuncules 29
furunculosis 55
fusidic acid **35**

gadolinium 45, **135**
gentamicin 131, **132**, **140**, 141
German Shepherds 72, **76**, 132
German Short-Haired Pointers 60
glandular hyperplasia **87**
glossopharyngeal nerve 11, 13
glucocorticoids 38, 71, 85, 146
glue ear 110–11
Golden Retrievers 57, 70, 71, 72, **74**
Gordon Setters 70
grabbing forceps 35, 37–8, **38**
Gram-negative infection 33, **34**, 35, 100, 102, 107, 108
Gram-positive infection **34**, 35, 100
granulation tissue **108**, 115, **116**, 122
granuloma disease 53, 70, 71–2
grass **87**, 107, 108, **108**
Great Danes 71
Greyhounds 81, **136**
gun dogs 63, 65, 99

haemangioma **73**
haemangiosarcoma 58, **73**, **74**
haematogenous spread of OM 107
haematoma 52, **53**, 146–8, 152, 164
haemorrhage **23**, 23, **24**
hair cells 18, 20, **20**, **21**, 21, 136, **137**
  deafness 138, 139, 140
hair follicles 2, **4**
hair plucking **23**, 26–8, 32
hamulus **10**, 14
harvest mites 66
head mange 64
head shaking 52–3, 92, 108, **114**, 146
  ectoparasites 63, 65
head tilt 125, 127–8, **130**, 130, 132–3, **135**, **136**
  cholesteatoma 109
  nasopharyngeal polyps **114**, **115**
  PSOM 111, **111**
hearing 15, 18–21, 125, 136–8
  BAER 46–7, **48**, 49
  see also deafness
helicotrema 18, **19**, 136, **137**
helix 1, 2
Heterodoxus spineger 64
Himalayan cats 60–1, **73**
histiocytoma **74**, **94**
histiocytosis 62, 72
history 115, **117**, 133–4
horizontal ear canal 2, 5, **6**, 7, 12
  ceruminolith **92**, 92–3, 108, **109**
  deafness **139**
  diagnosis 32, 35
  end-to-end anastomosis 159
  foreign bodies 108, **108**
  lateral wall resection 150
  neoplastic disease **97**
  otitis externa predisposition 97–9
  stenosis **105**
  subtotal ablation 158
  total ablation 155–6, **158**
  vertical ear canal resection 151–3
Horner's syndrome 13–14, 107, **114**, **117**
  vestibular disorders 125, **127**, 129, **129**, **131**
house dust mites 52, 63
Husky Dogs **76**
hyaline cartilage **4**
hyoid venous arch **6**, 161, **161**
hyperadrenocorticism (HAC) 28, 83, 84–5, 87–8, **88**
hyperpigmentation 52, 56
hyperplasia **23**, **87**, 93–7
hyperplastic stratified squamous epithelium 103
hypoglossal nerve 160, **161**, 162, **163**
hypoglossus muscle 160, **161**, 162
hypothyroidism 57, **68**, 87, **88**, 140
  vestibular disease 130, 132, 134
hypotrichosis *see* alopecia

idiopathic canine vestibular syndrome 133, **133**
idiopathic disease **87**, 90–1
idiopathic ear margin vasculitis 77–8
idiopathic facial dermatitis 60–1
idiopathic feline vestibular syndrome 132–3
immune-mediated disease **23**, 51, 54–5, 69–70, 78–9
immune-related disease 51, 77–80
impedance audiometry 141
impression smears **23**, **24**, 24–5, 29, 56
incisional drainage 146, 148
incus 6, **8**, 8, 136
infections 115
  ascending 107, 115
  descending 107, 108–10
  fungal 26, 28, 31–2, 56
  Gram-negative 33, **34**, 35, 100, 102, 107, 108
  Gram-positive **34**, 35, 100
  methicillin-resistant staphylococcal 67, **67**, 98
  viral 71, 80
  see also bacteria and bacterial infections; yeast infections
infectious pustular disease 51, 67–8
inflammation 28, 52, 103, 106, 107
inherited deafness 138, **139**, 139–40, 143
inner ear 15–21, 46, 125–43
  cochlea 136–43
  vestibular apparatus 125–36
internal acoustic meatus **18**
internal carotid artery **6**, 10–11, 13
internal carotid nerve 10
intertragic incisures 1, **2**, **3**, 149, **149**
ipsilateral pterygoid process **10**
Irish Setters **74**
irritant contact dermatitis 51, 53, 54, **54**

Jack Russell Terriers 55
jugular foramen 11
jugulohyoideus muscle 160, **161**, 161, 162
juvenile cellulitis **87**, 90–1

kanamycin sulfate 141
keratinization disorders 51, 58, 59, **87**, 90
keratoconjunctivitis sicca (KCS) 129, 134

Labradors 53, 57, **59**, 70, 72, **74**, 141
labyrinthitis 130
lactophenol cotton blue 55
Langerhans cells 52
lasers 31, **32**, 37–8, 106, 162–5
  auricular haematoma 148, 152
lateral bulla osteotomy 154–8, **158**, **159**
lateral wall resection 148–51
Leishmaniasis 55, 56–7
Leishmaniasis aethiopica 56
Leishmaniasis donovani 56
Leishmaniasis major 56
Leishmaniasis mexicana 56
Leishmaniasis tropica 56
lesions 37–8, 44, 46
  classes of primary **51**
  location in vestibular disease **127**, 127, **128**, 129
  pinna 23, 29–30, **30**, **31**, 31, **32**, 32, 51
  see also neoplasia
Lhasa Apsos **74**
lice 24, 26, 28, 64–5
lichenification 52, 56, 58
linguofacial vein **6**, 160, 161
Linognathus setosus 64
liquid paraffin **25**, 25–6, 27
local anaesthetic 31
locomotion abnormalities 125, 128
loop diuretics 141
lupus erythematosus 60, 72, 75, **76**
Lutzomyia spp 56

macula 18, 131
magnetic resonance imaging (MRI) 23, 37, 38, 44–6, **46**, 123
  peripheral vestibular disease 134, **135**, 135–6, **136**
  tympanic membrane 118, 122
malassezia **23**, 25, 33, 35, **102**, 108
  alopecia 83
  crust and scale 56, 58
Malassezia furfur 56
Malassezia pachydermatis **25**, **33**, 56
Malassezia sympodialis 56
malignant melanoma **94**
malleus 7, **8**, 8–9, **17**, 36, 136
manbrium of malleus 8, **8**, 12, **16**
mast cell tumours **73**, **74**, 146
mastoid process **137**
medication **87**, 91–2, 108
meiosis 14
melanoderma 82
melanoma **73**
membranous labyrinth 15, **19**, 125
meninges 15, **19**
meningioma **136**

methicillin-resistant staphylococcal infection 67, **67**, 98
metronidazole 131, **132**
Mexican Hairless Dogs 83
Microsporum canis 55
Microsporum gypseum 55
Microsporum persicolor 55
middle ear 5–14, **17**, 107–23, 138
  assessment 122–3
  chronic otitis media 115
  diagnosis 23, 35–6, 37–8, 43–4, 46–7, 49
  investigation of otitis media 115–23
  otitis media aetiology 107–15
Miniature Pinschers 81
mites 25, **25**, 26, **28**, 64, 88–9
  alopecia 83
  harvest 66
  house dust 52, 63
modiolus 18
mosquito-bite dermatosis 66–7
mosquito-bite hypersensitivity (MBH) 66–7, **67**
mucoperiosteum 115, 122
muscles 2, **4**, **5**, **10**, 159–62, **163**
  cervicoauricularis **5**, 159
  digastricus **10**, 160, **161**, 162, **163**
  hypoglossus 160, **161**, 162
  jugulohyoideus 160, **161**, 161, 162
  myohyoideus 160, **161**
  parotidauricularis **5**, 159
  sphincter colli **5**, 160, 161
  stapedius 8–9
  styloglossus 160, **161**, 162
musculotubal canal 9, **10**, **16**
mycoplasma 115
myohyoideus muscle 160, **161**
myringotomy **8**, 36, 118, 119, **120**
  PSOM 111, **112**
  technique **119**
  video otoscopy 37–8

nasodigital hyperkeratosis 56
nasopharyngeal polyps 37, 107, 113–14, **115**
  clinical signs **114**
  vestibular disorders 130, **134**
nasopharynx 9, **10**, 14, **17**, 107
neomycin 54, 99, 141
neoplasia 72–5, 122–3
  deafness 138, **138**, 140, 143
  diagnosis **23**, 28, 29, 31, 44, 46
  ear canal **87**, 93–7
  middle ear 112–13, **113**, **114**
  pinna 51, 58, 62, 72–5
  vestibular disorders 129, 130, 134–5
Neotrombicula autumnalis **26**, 66, **66**
nerves 5, 8, 10, 11, 13, 14, **117**
  auriculotemporal 5, **10**, 11
  caroticotympanic 13
  cranial 125, **127**, 128
  external acoustic meatus 11
  glossopharyngeal 11, 13
  hypoglossal 160, **161**, 162, **163**
  internal carotid 10
  postganglionic sympathetic 10, 13
  rostral auricular 11
  sympathetic **117**
  trigeminal 5, 8, 14
  tympanic 10
  vestibular 18, **20**
  vestibulocochlear **18**, **48**, 125, 136, 142
  see also cochlear nerve; facial nerve
neutrophils 29, **30**
nocardia 70
nodular disease **51**, 70–2
nodular hyperplasia **74**
nodular sebaceous hyperplasia **74**, **75**
nodules **23**, 29, 31, 56
non-steroidal anti-inflammatories 78
Notoedres cati 64
nystagmus 125–6, **127**, 128, 130, 133, **136**

oculocephalic reflex 125
oedema 54
organ of Corti 20, **21**, 21, 136, 138
ossicles 6, **8**, 11–12, 13, 21, 136, **137**
  hearing loss 47, 138, 140
osseous external acoustic meatus 2, **14**
osseous metaplasia 106
osteolysis 109
osteomyelitis 122
osteoproliferation 109
osteosarcoma **131**
osteosclerosis 109
otitis externa **4**, 37, 45, 141
  cholesteatoma 109
  conformation effects 97–8
  crust and scale 56, 58
  erythematous disease 52, 53, 54
  foreign bodies 91
  lateral wall resection 148
  nasopharyngeal polyps **114**
  neoplastic disease 97, 113
  papular disease 63
  pathogenesis to otitis media 107, 108
  perpetuating factors 100–6
  predisposing factors 97–100
  proliferative 138
  treatment effects 98–9
  triggers **87**
otitis media (OM) 11, 36, 37, 44, **46**, 100
  aetiology and pathogenesis 107–15
  calcification of ear canal **104**
  chronic 115, **116**
  clinical signs **117**
  cotton tip applicators 99
  deafness 138, 141
  diagnosis 109, 111, 113, 114
  foreign bodies 92
  investigation 115–23
  primary secretory (PSOM) 107, 110–12, **112**, 118
  secretory 139
  vestibular disorders 128–30, **130**, 132–6
otitis media interna 141
  clinical signs **117**
  vestibular disorders **128**, 128–9, **130**, 132–6
Otodectes cynotis **33**, 33, 87, 88, **89**
otoliths 110, **110**, **116**
otoscopy 119, **121**, 121
  cone 32, 35, 37
ototoxicity 129, 131, 138, 140–1, 143
oval (vestibular) window 6, 9–10, **12**, **17**, 18, 21
  deafness 138
  hearing 136, **137**
  pressure from growth 113

palpation 122
papilloma **74**, **94**, 165
papillomatosis 71
papillomavirus 71
papovavirus 71
papular disease **51**, 63–7
papules **23**, 29, 31, 56, 63–7
para-aural abscess **45**
paraneoplastic exfoliative dermatitis (PED) 62
parasites *see* ectoparasites
parotidauricularis muscle **5**, 159
pars flaccida 8, 12, 36, 87, **88**
pars tensa 8, 36, 118, **119**, **120**
Pasteurella **34**, 35, 130
pathological nystagmus 126, **127**
pattern baldness 81
Pautrier's microabscesses 62
pediculosis 64–5
pemphigus foliaceus **68**, 68–9, **69**, **87**, 90, **91**
  diagnosis **23**, 29, **30**
periauricular abscesses **4**, **139**, **158**, 158
perilymph 9, 15, **19**, 136, **137**
  deafness 138
perilymphatic duct 15
periocular lunettes 56
peripheral vestibular disorders 125, 126, **128**, 128, 129–36
  clinical signs **127**
Persian cats 55, 60–1, **73**
personal protective equipment 163–4
petro-occipital fissure 11
petrous temporal bone 8, 10, **39**
phaeohyphomycosis 70
Phlebotomus spp 56
physiological nystagmus 125, 126, **127**
pigment-associated inherited deafness 139, **139**
pinch biopsy 35, **36**, 37
pinna 1–5, 21, 51–85
  alopecia 81–5
  crust and scale 55–63
  diagnosis 23–32
  erythematous disease 51–5
  lacerations and surgery 145–6
  laser surgery 163, 164, 165
  neoplasia 72–5
  nodular disease 70–2
  papular disease 63–7
  pustular disease 67–70
  ulcerative disease 72, 76–80, **81**
pinnectomy **138**, 145, 146, **147**, **165**
plasmacytoma **74**, **94**
platysma 2, **5**, 160, 161
pollen 52
polyhexamethylene biguanide 131, **132**, **140**, 141
polyps **87**, 94, **103**, 130, **130**
  see also nasopharyngeal polyps
Poodles 59, **74**, **98**, 98

positional nystagmus 126
positive contrast canalography 41–2, **44**
postganglionic sympathetic nerves 10, 13
posture 125, **127**, 128
potassium 139, 141
potassium hydroxide 25–6, **27**, 27, 55
prednisone **135**
presbycusis 140, 143
Preyer's reflex **141**, 141
primary idiopathic seborrhoea 58, **59**, **87**, 90
primary secretory otitis media (PSOM) 107, 110–12, **112**, 118
proliferative otitis externa 138
proliferative and necrotizing otitis **87**, 91
promontory 9, **9**, **10**, 11, 13–14, **14**, **16**
  surgery **163**
  tympanic membrane 107, 109, 118, **121**, 121–2, **122**
proprioceptive deficits 125, **127**, 128
propylene glycol 54, 99, 109, 131, 141
proteus **34**, 35, 130
pruritus 52–4, 55, 58, 61, 63–6
pseudomonas **88**, 100, **101**, **102**, **109**, 109, 130
  diagnosis 33, **34**, 35
psoriasiform-Lichenoid dermatosis 62
Pugs **128**, **130**
punch biopsy 31, 148
punched out ulcers 77
puppy pyoderma 69–70
puppy strangles 69–70
pustular disease **51**, 67–70
  infectious 51, 67–8
  sterile **23**, 51, 68–70
pustules **23**, 29, **30**, 56
pyoderma **23**, 58, 61, 67, 69–70
pyogranulomatous disease **23**, 29, 71–2

rabbits 65
radiography 23, 37, 38–43, **44**, 113, 122–3
  positioning 38, **39**, 40, **40–2**, **44**
  vestibular disease 134–5
rats **20**
Reissner's membrane 20
respiratory infection 115
reticular membrane 21
ringworm **55**
rods of Corti 21
rostral auricular nerve 11
Rottweilers 72
Rough Collies 72, 78
round (cochlear) window 6, 9, 13, **15**, **17**, 18, **20**
  hearing 136
  otitis media 107
  pressure from growth 113
surgery **163**

saccule 15, 18
sacculus 15, 125
salivary glands 2, 4, 11, 160
Samoyeds 59
sarcoma **73**, **94**, **95**
sarcoptes **23**, 26, **27**, 63
Sarcoptes scabiei 63, **64**
scala media 20, 21, 136, **137**, 139
scala tympani 9, 18, **19**, **20**, 136, **137**
scala vestibuli 18, **19**, **20**, 21, 136, **137**
scapha **1**, 2
Schirmer tear test 134
Scottish Terriers **74**
scutiform cartilage 2, **5**
sebaceous adenitis 28, 59, **87**, 90
sebaceous epithelioma **74**
sebaceous gland adenocarcinoma **74**, **94**
sebaceous gland adenoma **74**, **94**, 94, **95**, 96
seborrhoea 59–60
  primary idiopathic 58, **59**, **87**, 90
secretory otitis media 139
  primary (PSOM) 107, 110–12, **112**, 118
semicircular canals 15, **17**, **18**, **20**, **137**
semicircular ducts 5, 18, 125, **126**, 139
sensorineural deafness 47, 49, 138, 139–40, 141, 143
septum 6, **7**, 13, **14**, 15, **16**, **17**
  surgery 162, 163
Shar Peis **74**, 98, **98**, **99**
Shetland Sheepdog 72, 78
Shih tzus **74**
Siamese cats **73**, 132, **136**
Siberian Husky Dogs 61, 72
silver sulphadiazine 99
skin scrapings **23**, 25–6, **27**
skulls **40**, **41**, **43**, 157
  cats **13**, **14**, **15**
  dogs **7**, **9**, **10**, **12**
Smooth Fox Terriers 132
soft tissue sarcoma **94**, **95**
soft tissue growth 113, **114**
Spaniels 65, 98, **98**
sphincter colli muscle **5**, 160, 161
Sphinx cats 83
Spilopsyllus cuniculi 65, **65**
spiral ganglion cells 140, 141
spiral lamina 18
spiral ligament 20
Sporothrix schenkii (sporothrichosis) 70
Springer Spaniels 59
squamous cell carcinoma 58, 72, **73**, **74**, **139**
  ear canal **94**, **97**, 97
  laser surgery **165**
  middle ear 113
  pinnectomy 146
Staffordshire Bull Terriers **74**
stains **24**, 25, 29, 122
Standard Poodles 59
stapedius muscle 8–9
stapes 6, 8–9, **17**, 136, **137**
Staphylococcus spp 33, **34**, 35, **67**, 67–8, **68**, 98
  crust and scale 62
  ear canal **101**
  nodular disease 70
  otitis media 115
  vestibular disorders 130
Staphylococcus aureus 67
Staphylococcus hyicus 67
Staphylococcus pseudintermedius 67
Staphylococcus schleiferi 67
Stephen-Johnson syndrome (SJS) 79
stereocilia (hair cells) 20, **21**, 21, 136, **137**
  deafness 138, 139, 140
sterile granuloma disease 71–2
sterile pustular disease **23**, 51, 68–70
sticktight fleas 51
Stomoxys calcitrans 66
strabismus 125, **127**, 127, **128**
Streptococcus spp 33, **34**, 35, 70, 115
streptomycin 131
stria mallearis 8, **8**
stria vascularis 20, **137**, 139, 140
styloglossus muscle 160, **161**, 162
stylomastoid foramen **9**, 10, **137**, 156, 157
subtotal ear canal ablation 157–8, **159**
surgery 44, 145–65
Swiss Mountain Dogs **135**
sympathetic nerve damage **117**
syringomyelia 111

T cell lymphoma 113
T helper cells 52
tape strips **23**, **24**, **25**, 56
teat cannula 146, 147
tectorial membrane **20**, **21**, 21, 136, **137**, 138
telogen hairs 28
Terriers 85
third eyelid prolapse 14
thymoma 62, **63**
ticks 66
tobramycin 131, **132**
Tonkinese cats 132
topical vestibulotoxicity 131
total ear canal ablation 154–8, **158**
toxic epidermal necrolysis (TEN) 79
tragus 1, 2
trauma 13, **52**, 63, 135, 140
  deafness 138, 140
  ear canal changes **105**, 106
  hair loss 27, **27**
  surgery 145, 158, **159**
  vestibular disorders 129, 132, 133
Trichodectes canis **64**, 64
trichography 26–8, 55, 59
Trichophyton erinacei 55
Trichophyton mentagrophytes 55
triethanolamine 141
trigeminal nerve 5, 8, 14
TrisEDTA 131, **132**, **140**, 141
trombicula 26
tubus auris 2
tympanic bulla 36, 109, 119, 122–3
  CT scan 44, **45**
  granulation tissue 115, 122
  growths 113, **114**
  middle ear (cat) **12**, **13**, 13, **14–17**
  middle ear (dog) 6, **7**, **10**, 11, **17**
  MRI 46
  otitis media 107, 115, **116**, 119, **120**
  otoliths **110**, **116**
  peripheral vestibular disorders 128, **129**, **130**, 130, **131**, 131–2
  PSOM 111, 112
  radiography 38, **39**, 40–1, **42–3**
tympanic cavity 6–7, 9–10, 12, 13, **17**
tympanic membrane 5, 6–9, 12–13, **16**, 21, 118–22
  assessment of patency 121–2, **122**
  ceruminolith 92, **93**
  cotton tip applicators 99
  deafness 138, 141

VetBooks.ir

tympanic membrane (*continued*)
diagnosis 35–6, 37, 42–3, 49
foreign bodies 108, **109**
hearing 136, **137**
intact abnormal 118, **119**
intact normal 118, **118**
myringotomy **119**, **120**
otitis media 107, 108, 118–22
pseudomonas otitis 100, **101**, **102**
PSOM 111, **111**, 118
radiography 42–3
rupture 119–21, 138
tears 119, **121**
tympanic nerve 10
tympanic plexus **10**, 11, 13–14
tympanometry 37, 49, 122, 141
tympano-occipital fissure 11, 13

ulcerative disease **51**, 72, 76–80, **81**
ulcers 23, 29, 31
ear canal 98–9, **100**, **101**, 102
irritant contact dermatitis **54**, 54
ultraviolet (UV) light 58
umbo 8, 92
utricle 15, 18
utriculosaccular duct 15
utriculus 15, 125

vaccines 78
vasculitis **23**, 77–8
ventral bulla osteotomy **7**, **11**, 160–1, **162**
cats 12, **14**, **16**, 161–2, **162**, **163**
vertical ear canal 2–3, **3**, 5, 32, 35, 52
deafness **139**
lateral wall resection 148, 149, 150, 151
resection 151–4
subtotal ablation 158, **159**
total ablation 154, **158**
vestibular apparatus 125–36
effects of medication **132**
location of lesions **127**
vestibular aqueduct 15
vestibular disorders 125–9
clinical signs **127**
effects of medication **132**
otitis media 107, 110, 113
peripheral dysfunction 129–36
vestibular nerve 18, **20**
vestibular window *see* oval (vestibular) window
vestibule 15, 18, **20**
vestibulocochlear nerve **18**, **48**, 125, 136, 142
vestibule-ocular reflex 125
video otoscopy **16**, 35, 36, 37–8, **99**
cholesteatoma 109
lateral wall resection 148
tympanic membrane 118, 121
viral infections 71, 80
Vizslas 59

Weimaraners 71
West Highland White Terriers 56, **75**
wetting 99
Whippets 81
white cats 139
Wood's lamp examination 32
Wright's stain **24**, 25, 29

yeast infections 25, 28, 33, 70, 102
crust and scale 55, 56, 61
otitis externa 99, 100, 102
Yorkshire Terriers 55, **69**, **75**, 82, **138**

zinc-responsive dermatoses 61