Practical Guide to Canine and Feline Neurology

3rd Edition

Curtis W. Dewey • Ronaldo C. da Costa

with website

WILEY Blackwell

PRACTICAL GUIDE TO CANINE AND FELINE NEUROLOGY

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Third Edition

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Dedication

Alexander de Lahunta, DVM, PhD, DACVIM (Neurology), DACVP.

As neurologists, Ronaldo and I shudder to think where we would be professionally without the myriad and substantial contributions that Alexander ("Sandy") de Lahunta has made to our specialty. He has–in a career spanning nearly half a century–laid the framework for our understanding of neuroanatomy and neuropathology. It is a testament to this man's legendary and iconic status in veterinary medicine overall that any veterinarian who opens this book will immediately feel respect and gratitude for "Dr. D" and know that he deserves all the accolades we can bestow upon him. And if you would like to read about the accolades that Dr. D has earned, you should go online; they are far too numerous to fit on a textbook dedication page. Dr. D's contributions to our understanding of embryology, anatomy, neurology, and neuropathology are voluminous and ongoing. His passion has been and remains fulfilling the role of teacher. As one of his former students, I can personally attest to his unequalled skill in this arena. I can also attest to the fact that Dr. D has kept in touch with many of his students after they graduated and moved forward with further educational endeavors and careers. Years after I left Cornell as a student, I would hear of Dr. D telling his current students about something I had published in a journal. It meant a lot to know that someone I revered so highly was proud of my accomplishments. Ronaldo and I are proud of Alexander de Lahunta, as all veterinarians should be, and feel incredibly fortunate that he influenced our career paths. We dedicate this edition to someone who has positively and permanently changed the face of veterinary neurology and veterinary medicine in general—Dr. Alexander de Lahunta.

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About the Editors

Curtis W. Dewey, DVM, MS, DACVS, DACVIM (Neurology), Associate Professor and Section Chief of Neurology/Neurosurgery, Cornell University. Dr. Dewey was a faculty neurologist at Texas A&M University (1995–2001) and a staff neurologist at Long Island Veterinary Specialists (2001–2006) prior to returning to his alma mater in 2006. He has authored and coauthored numerous peer-reviewed journal articles and many textbook chapters. He is a nationally and internationally recognized speaker and has served on the editorial board of a number of veterinary journals (Veterinary Surgery, Journal of the American Animal Hospital Association, Compendium on Continuing Education for the Practicing Veterinarian). He has also served as an ad hoc reviewer for many other journals. He has served on the ACVIM (Neurology) Residency Training Committee (2005–2008; committee chair 2007–2008), and the ACVIM Taskforce on Neurosurgical Training of Neurology Residents (2004–2010; committee chair 2007–2010). Dr. Dewey has been a VIN (Veterinary Information Network) consultant since 2004. He consults regularly with Long Island Veterinary Specialists (LIVS) and Veterinary Specialists and Emergency Service (VSES) of Rochester. He has been a member of the Board of Directors of the New York Veterinary Foundation since 2008. Dr. Dewey is a member of the AVMA and VECCS. His main areas of research include seizure control and the surgical management of congenital brain disorders. Dr. Dewey was recently the recipient of the 2014 Hills ACVECC Jack Mara Scientific Achievement Award.

Ronaldo Casimiro da Costa, DMV, MSc, PhD, Dipl. ACVIM (Neurology), Associate Professor and Service Head, Neurology and Neurosurgery, The Ohio State University College of Veterinary Medicine. Prior to joining the Ohio State University in 2008, Dr. Ronaldo C. da Costa was a faculty member at the Federal University of Paraná in Brazil (1997–2008), the same school where he obtained his DMV degree. Following that, he got his Masters in Surgery in Brazil, and completed his residency and PhD at the Ontario Veterinary College, University of Guelph. He has authored and coauthored dozens of peer-reviewed journal articles and several book chapters in authoritative textbooks. He was also the Guest Editor of a Veterinary Clinics of North America Small Animal Practice focused on spinal diseases. Dr. da Costa actively serves the profession as a member of the ACVIM, AVMA, and other associations, a reviewer of several national and international veterinary journals, and as the founding President of the Brazilian Veterinary Neurology Association. He has an active clinical and research program on spinal diseases, primarily cervical spondylomyelopathy, and has received over 30 awards for academic, research, and teaching excellence. Dr. da Costa has lectured extensively nationally and internationally, and is passionate about teaching. He has received eight teaching awards including the Pfizer Norden Excellence in Teaching Award (highest college teaching award) and the Excellence in Graduate Teaching Award at Ohio State. He was a finalist for the National Pfizer Norden Award Excellence in Teaching Award.

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Preface

In the preface to the second edition of *A Practical Guide to Canine and Feline Neurology*, I mention the increasingly difficult task of keeping up with the rapidly expanding knowledge base in the field of small animal neurology and neurosurgery. As the time approached to consider working on the third edition, I became increasingly anxious about this endeavor. I am proud of this book and truly believe it has helped many veterinary students, general small animal practitioners, interns, residents and specialists; however, and perhaps because of the book's success, the thought of writing a third edition struck me as less the labor of love that the previous edition had been and more a herculean task, with all the feelings of trepidation that engendered. I realized that this transformation from positive to negative was rooted in my concern that this textbook's next iteration—in order to stay true to my vision of maintaining a comprehensive, up-to-date, visually attractive, and clinically useful reference source—was beyond my capability to create as a sole editor. I came to the conclusion that this book had outgrown me as a sole editor, and that I needed help. I chose Ronaldo da Costa for this monumental task and—fortunately for me and anyone who reads this book—he agreed to be co-editor.

Ronaldo's insight and passion for this project reinvigorated my outlook and drive to create the best edition of this textbook yet. His resourcefulness and attention to detail are also very evident in the pages. This time around, both Ronaldo and I pooled comments we had heard about the second edition, in order to make the third edition better than the second. This edition is obviously in color (mostly), which is great, but looks aren't everything (it's true, despite what the media portrays). There are quite a few new features of this edition that I am sure will be very helpful.

You will notice some new contributing authors (in addition to Ronaldo, of course), some new chapters, and some new images. A few of the exciting (I think) additions include a differential diagnosis chapter (by Ronaldo and me), a chapter devoted solely to magnetic resonance imaging (Ronaldo and Silkie Hecht), and a chapter dealing with movement disorders (Simon Platt). These additions, along with approximately 400 new figures and dozens of videos, will hopefully greatly enhance the reader's experience.

As with the first and second editions, we requested assistance with images from a number of colleagues and, as before, these individuals came through for us. You will notice that there are quite a few new images provided by Diane Shelton, Jen Bouma, and Scott Fellows. We also have a number of fantastic new illustrations by Tim Vojt, Medical Illustrator, and beautiful new pictures by Jerry Harvey, Medical Photographer, both from the Ohio State University. A number of our colleagues have expressed interest in assisting with the next edition. The fact that these individuals volunteered their involvement for the fourth edition is quite a compliment. And we will likely be contacting you about your impending involvement very soon.

Ronaldo and I hope you like this edition and that you find it "user friendly" in teaching, clinical practice, or both. And if you have been referring to this book as "Dewey," please refer to it now and in the future as "Dewey/da Costa." I think that this has a nice alliterative ring to it.

Acknowledgments

Something like this that you have in your hands could not happen without the help of countless individuals, people that cared for us way before we were something in life! We would like to thank our entire families, starting with Curtis' parents (Pam and Maurie), wife (Janette), and kids (Jordan, Isaiah, Ethan, Carver, Sole, and Jolie), and Ronaldo's parents (Sirlei and Ivo), wife (Luciana) and kids (Felipe, and Rafaela). Without their love, support, and patience (!) over the years, this would not have come to fruition.

On the professional side we both had several mentors who guided us in our professional growth as veterinary neurologists. I (Curtis) have had the privilege of having been trained and inspired by legends of the veterinary profession at Cornell University, the University of Georgia, and the University of California-Davis. I (Ronaldo) would like to thank the faculty members at the Federal University of Paraná, Federal University of Santa Maria, and the Ontario Veterinary College, University of Guelph, especially Dr. Joane Parent for everything!

We are indebted to all contributors—Linda Barter, Mary Tefend Campbell, David Dorman, Julie Ducoté, Daniel Fletcher, Thomas Fletcher, Silke Hecht, Janice Huntingford, Karen Kline, Paula Martin-Vaquero, Simon Platt, Jacques Penderis, Bruno Pypendop, Sean Sanders, Lauren Talarico, and Bill Thomas—who generously offered their time and expertise to make this book a comprehensive and up-to-date resource. We are also very thankful for the images and advice offered by Diane Shelton for the neuropathy and myopathy chapters.

Obviously without the inspiration and support of our friends, colleagues, technicians, and residents at the Cornell University and the Ohio State University (too many to be named!) this book would not have been completed. Two people in particular were key to making this third edition what it is: Paula Sharp at Cornell and Tim Vojt at Ohio State. We cannot thank them enough for their time and expertise. Additionally, the videos you see could not be possible without the assistance of Heather Myers and Amanda Disher. We are also very grateful to our patients and their guardians who allowed us to care for and learn from them. Ultimately, this book only exists because of them.

Thanks also to the staff at Wiley-Blackwell, especially Nancy and Catriona, for bearing with us through this process.

Finally, I (Ronaldo) want to personally thank Curtis for inviting me to join him on this journey. It is truly an honor to work with him on a book that I used extensively as resident and neurologist, and have recommended to countless students and veterinarians. By working on this book over the past few years, it became clear that no one edits a textbook for financial reasons! Those that have the vision to do this, do so sacrificing their personal and professional time to benefit their profession and their patients. Curtis had the vision to do this 12 years ago and I applaud him for that. This third edition is an evolution of his dedication and commitment to the veterinary profession. I cannot thank him enough for giving me the opportunity to assist him in such a noble task.

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/dewey/neurology

The website includes:

- Videos
- Link to the University of Minnesota's Canine Brain Atlas

The password for the companion website is the last word in the caption for Figure 2.22.

CHAPTER 1

Signalment and History: The First Considerations

Curtis W. Dewey & Ronaldo C. da Costa

Introduction

When presented with a patient that is suspected of having a neurologic disorder, the signalment (i.e. breed, age, and sex) and history are often helpful in guiding the clinician toward the most likely diagnosis. It is important to recognize, however, that this information is *adjunctive* to the neurologic examination. Properly weighting the importance of signalment and history will help avoid "tunnel vision" when devising diagnostic plans and implementing treatment strategies.

Signalment[1–3, 5]

The information in Table 1.1 and Table 1.2 provides a summary of suspected and confirmed breed predilections for various neurologic disorders. Knowledge of breed predilections can be very helpful when considering differential diagnoses, especially for uncommon presentations (e.g. neuropathies in juvenile patients). The clinician should be aware of the limitations of breed predilection tables, however. Newly discovered breed predilections or undiscovered breed predilections will not necessarily be represented in a table. In other words, breed predilection tables tend to increase in size with successive textbook editions. Also, breeds other than those reportedly predisposed to a particular disorder may occasionally be affected by that disorder. Finally, certain rare disorders may have only one or a few members of a certain breed reported in the literature. Since some of these disorders are inherited (e.g. lysosomal storage diseases), it may be assumed that the breed is at risk, despite low numbers of actually confirmed cases.

Certain disease categories tend to be more likely with specific age groups. In general, neoplasia (e.g. brain tumor) is more common in older patients. Congenital disorders (e.g. hydrocephalus) are more commonly encountered in juvenile patients. As with other aspects of patient signalment, there are no "absolutes" in regard to age for the various neurologic disorders encountered in clinical practice. Some congenital disorders tend to cause clinical dysfunction in adult patients (e.g. Chiari-like malformation (CLM)) and some neoplasms are typically encountered in young patients (e.g. nephroblastoma of the spinal cord).

There are very few neurologic disorders with sex predilections. One example would be muscular dystrophy in Golden Retrievers, an X-linked heritable disease.

History[4]

Obtaining a concise and accurate medical history as it pertains to a specific neurologic complaint is often crucial to guiding the diagnostic plan. It is important to allow the pet owner to elaborate on pertinent historical details; it is equally important to dissuade the pet owner from delving into historical details that have little or nothing to do with the chief clinical complaint. For example, an intricate account of events concerning a cranial cruciate ligament repair from 10 yrs ago is unlikely to be of value in a patient that presents for head-pressing and generalized seizure activity. Alternatively, pet owners often omit pertinent historical details. A pet owner may not necessarily think, for instance, that a change in the sound of their myasthenic dog's bark (dysphonia) is in any way related to the pelvic limb weakness that prompted them to seek medical advice. Although definitely related to the chief complaint, dysphonia may be regarded by the owner as an unrelated and clinically unimportant observation. In such instances, it is up to the clinician to ask specific questions that may help to elucidate the nature of the patient's neurologic disorder.

It is very important to get a specific history that does not involve *interpretation* of signs by the owner but rather descriptive facts related to the owner's *observation* of signs only. This is a common mistake in clinical neurology. For example, a client may observe a dog getting disoriented, falling into lateral recumbency, and paddling for a few seconds. This event could either be an acute vestibular episode or a seizure. Owners will likely interpret this event as a seizure. If the clinician accepts the owner's interpretation of the event as a seizure, he/she could follow an

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Companion Website: www.wiley.com/go/dewey/neurology

Table 1.1 Breed-associated neurologic abnormalities of dogs.

Afghan Hound	Acquired (idiopathic) laryngeal paralysis
	Hereditary myelopathy (leukodystrophy)
	Narcolepsy/cataplexy
	Retinal degeneration
Airedale Terrier	Cerebellar abiotrophy
	Cerebellar hypoplasia
	Congenital myasthenia gravis
	Degenerative lumbosacral stenosis
Akita	Acquired myasthenia gravis
	Congenital deafness
	Congenital vestibular disease (bilateral)
	Glycogenolysis (type III)
Alaskan Husky	Gangliosidosis (GM1)
	Mitochondrial encephalopathy (Leigh's disease, subacute necrotizing encephalopathy)
Alaskan Malamute	Hereditary polyneuropathy
	Myelodysplasia
	Muscular dystrophy
	Osteochondromatosis of the vertebrae
American Bulldog	Ceroid lipofuscinosis
American Eskimo dog	Congenital deafness
Australian Blue Heeler	Congenital deafness
Australian Cattle dog	Ceroid lipofuscinosis
	Congenital deafness
	Dermatomyositis
	Mitochondrial encephalomyelopathy
	Myotonia congenita
	Polioencephalomyelopathy
Australian Kelpie	Cerebellar abiotrophy
Australian Shepherd	Ceroid lipofuscinosis (CLN 6)
	Congenital deafness
Basset Hound	Cervical spondylomyelopathy (bony stenosis)
	Degenerative disc disease (type I)
	Globoid cell leukodystrophy (Krabbe's disease)
	Glycoproteinosis (Lafora's disease)
Bavarian Mountain dog	Cerebellar abiotrophy
Beagle	Agenesis vermis cerebellum
	Congenital deafness
	Congenital vestibular disease
	Cerebellar abiotrophy
	Globoid cell leukodystrophy (Krabbe's disease)
	Glycoproteinosis (Lafora's disease)
	Idiopathic epilepsy
	Intervertebral disc disease (type I)
	Methionine deficiency-related spinal myelinopathy
	Narcolepsy
	Necrotizing vasculitis (steroid meningitis, Beagle pain syndrome)
Beagle mix	Gangliosidosis (GM1)
Belgian Sheepdog	Congenital nystagmus
	Muscular dystrophy
Belgian Shepherd (Groenendael)	Muscular dystrophy
Belgian Shepherd (Malinois)	Degenerative myelopathy
	Degenerative lumbosacral stenosis
	Leukodystrophy/spongy degeneration (encephalomyelopathy; Belgian Shepherd (Malinois)/Shepherd mixed-breed dogs)

Table 1.1 (*Continued*)

Belgian Shepherd (Tervuren)	Idiopathic epilepsy
	Muscular dystrophy
Bern Running dog	Cerebellar degeneration
Bernese Mountain dog	Aggression
	Cerebellar abiotrophy
	Degenerative myelopathy
	Epilepsy
	Hepatocerebellar degeneration
	Histiocytic sarcoma
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Meningitis/meningomyelitis (necrotizing vasculitis)
Bichon Frise	Atlantoaxial instability
	Caudal occipital malformation syndrome
	Congenital deafness
	Idiopathic tremor syndrome (steroid responsive)
Blue Tick Hound	Globoid cell leukodystrophy
Boerboel	Cervical spondylomyelopathy
Border Collie	Cerebellar abiotrophy
	Ceroid lipofuscinosis
	Congenital deafness
	Fibrocartilaginous embolic myelopathy
	Idiopathic epilepsy
	Sensory neuropathy
Border Terrier	Spongiform leukoencephalopathy
Borzoi	Cervical spondylomyelopathy
	Congenital deafness
Boston Terrier	Brain tumor (gliomas)
	Cerebellar abiotrophy
	Congenital deafness
	Congenital hydrocephalus
	Congenital vertebral malformation (hemivertebrae)
	Intracranial arachnoid cyst
	Muscular dystrophy
	Myelodysplasia
	Vermian hypoplasia
Bouvier des Flandres	Distal sensorimotor polyneuropathy
	Hereditary laryngeal paralysis
	Muscular dystrophy
	Pharyngeal/esophageal myopathy
Boxer dog	Autoimmune polymyositis (+/– paraneoplastic)
	Congenital deafness
	Corticosteroid-responsive (aseptic) meningitis
	Degenerative myelopathy
	Disseminated idiopathic skeletal hyperostosis (DISH)
	Head-bobbing (suspected dyskinesia)
	Neuroaxonal dystrophy
	Neuronal vacuolation
	Pilonidal (dermoid) sinus
	Primary brain tumor (glioma, meningioma)
	Progressive axonopathy
	Sensory neuropathy
	Spondylosis deformans
Briquet Griffon Vendéen	Spinal muscular atrophy (motor neuron disease)

Table 1.1 (*Continued*)

Brittany Spaniel	Cerebellar abiotrophy (late onset)
	Muscular dystrophy
	Sensory ganglioradiculitis
	Spinal muscular atrophy
	Spinocerebellar degeneration
Brussels Griffon	Chiari-like malformation (CLM)
Bull Mastiff	Cerebellar abiotrophy
	Cervical spondylomyelopathy
	Extradural synovial cyst
	Leukodystrophy/spongiform degeneration
Bull Terrier	Cerebellar abiotrophy
	Congenital deafness
	Hereditary laryngeal paralysis
	Hyperkinesis
	Tail chasing
Cairn Terrier	Globoid cell leukodystrophy
	Hydrocephalus
	Portosystemic shunt (hepatic encephalopathy)
	Spinal muscular atrophy (motor neuron disease)
Cardigan Welsh Corgi	Congenital deafness
	Sensory ganglioradiculitis
Catahoula Leopard dog	Congenital deafness
Cavalier King Charles Spaniel	Chiari-like malformation (CLM)
	Cerebellar infarct
	Congenital deafness
	Dorsolateral vertebral canal stenosis and compression at C2–C3
	Episodic muscle hypertonicity ("falling cavaliers"—probable dyskinesia)
	Femoral thromboembolism
	Fly chasing behavior
	Idiopathic epilepsy
	Primary secretory otitis media
Chihuahua	Atlantoaxial instability
	Ceroid lipofuscinosis
	Congenital deafness
	Congenital hydrocephalus
	Muscular dystrophy
	Necrotizing meningoencephalitis
	Neuroaxonal dystrophy
Chinese Crested	Cerebellar abiotrophy
Chow Chow	Cerebellar hypoplasia
	Congenital deafness
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Myotonia congenita
Clumber Spaniel	Cerebellar abiotrophy
	Mitochondrial myopathy
Cocker Spaniel	Cerebellar abiotrophy
	Ceroid lipofuscinosis
	Congenital deafness
	Congenital vestibular disease (English)
	Cryptococcosis (American)
	Hydrocephalus
	Idiopathic facial nerve paralysis
	Intervertebral disc disease (type I)
	Juvenile epilepsy
	Leukodystrophy/spongiform degeneration

Table 1.1 (*Continued*)

	Multisystem neuronal degeneration (red-haired)
	Muscular dystrophy
	Myopathy (lipid storage, mitochondrial, phosphofructokinase deficiency)
	Myotonia congenita
Collie (rough-coated)	Cerebellar abiotrophy
	Dermatomyositis
	Optic nerve hypoplasia
	Sensory trigeminal neuropathy
Collie (scotch)	Congenital deafness
	Dermatomyositis
	Distal polyneuropathy
Collie (smooth-coated)	Congenital deafness
	Dermatomyositis
	Neuroaxonal dystrophy
	Spinal muscular atrophy
Coton de Tuléar	Cerebellar abiotrophy (two forms)
Dachshund	Cerebellar abiotrophy
	Ceroid lipofuscinosis
	Congenital deafness (dappled)
	Glycoproteinosis
	Idiopathic epilepsy
	Intervertebral disc disease (type I)
	Mucopolysaccharidosis (type III; wire-haired)
	Myasthenia gravis (congenital, acquired)
	Narcolepsy/cataplexy
	Neuronal glycoproteinosis (Lafora's disease)
	Sensory neuropathy (long-haired)
Dalmatian	Ceroid lipofuscinosis
	Cervical spondylomyelopathy
	Congenital deafness
	Episodic muscle hypertonicity ("cramp")
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Laryngeal paralysis/polyneuropathy complex
	Leukodystrophy/spongy degeneration
Doberman Pinscher	Cervical spondylomyelopathy
	Congenital deafness
	Congenital vestibular disease (uni or bilateral)
	Dancing Doberman disease
	Idiopathic head tremor
	Idiopathic self-mutilation (sensory neuropathy)
	Immune mediated myositis
	Narcolepsy/Cataplexy
Dogo Argentino	Congenital deafness
	Laryngeal paralysis/polyneuropathy complex
Dogue de Bordeaux	Cranial thoracic stenosis
English Bulldog	Cerebellar abiotrophy
	Congenital deafness
	Congenital vertebral malformation (Hemivertebra)
	Hydrocephalus
	Idiopathic head tremor
	Sacrococcygeal malformation
	Spina bifida

(*continued*)

Table 1.1 (*Continued*)

English Foxhound	Methionine deficiency-related spinal myelinopathy (Hound ataxia)
English Pointer	Cerebellar abiotrophy
	Sensory neuropathy (automutilation)
	Spinal muscular atrophy
English Setter	Ceroid lipofuscinosis
	Congenital deafness
Fila Brasileiro	Intervertebral disc disease (type II)
Fox Terrier	Congenital deafness
	Myasthenia gravis (congenital)
	Spinocerebellar degeneration
French Bulldog	Arachnoid diverticulum
	Congenital deafness
	Congenital vertebral malformation (Hemivertebrae)
	Idiopathic head tremor
Gammel Dansk Honsehund	Congenital myasthenic syndrome (presynaptic)
German Shepherd dog	Acquired myasthenia gravis
	Autoimmune polymyositis
	Cervical spondylomyelopathy
	Congenital deafness
	Congenital megaesophagus
	Congenital vestibular disease
	Cranial thoracic disc disease (protrusion)
	Degenerative lumbosacral stenosis
	Degenerative myelopathy
	Fibrotic myopathy
	Giant axonal neuropathy
	Hereditary laryngeal paralysis (white coat)
	Idiopathic epilepsy
	Intervertebral disc disease (type II)
	Masticatory myositis
	Mitochondrial myopathy
	Mucopolysaccharidosis
	Nephroblastoma
	Neuroaxonal dystrophy
	Spinal muscular atrophy (motor neuron disease)
German Shorthaired Pointer	Coccygeal muscle injury
	Gangliosidosis (GM2)
	Hemivertebra
	Pyogranulomatous meningoencephalomyelitis
	Sensory neuropathy
Golden Retriever	Acquired myasthenia gravis
	Eosinophilic meningoencephalitis
	Extraocular myositis
	Horner's syndrome
	Hypomyelinating polyneuropathy
	Idiopathic epilepsy
	Multiple cartilaginous exostoses
	Multisystem axonopathy and neuronopathy
	Muscular dystrophy
	Myasthenia gravis
	Primary brain tumor (meningioma)
	Sensory neuropathy
Gordon Setter	Cerebellar abiotrophy
Great Dane	Cervical spondylomyelopathy
	Inherited (noninflammatory/central core) myopathy
	Congenital deafness

Table 1.1 (*Continued*)

	Congenital myotonia
	Disseminated idiopathic skeletal hyperostosis (DISH)
	Distal symmetric polyneuropathy
	Extradural synovial cyst
	Fibrocartilaginous embolic myelopathy (FCE)
	Myasthenia gravis
	Nemaline myopathy
	Primary orthostatic tremor
	Spinal muscular atrophy (Great Dane crosses)
Great Pyrenees (Pyrenean Mountain dog)	Congenital deafness
	Laryngeal paralysis/polyneuropathy complex
	Optic nerve hypoplasia
Greyhound	Cervical disc disease
	Congenital deafness
	Congenital megaesophagus
	Corticosteroid (aseptic) responsive meningitis
	Degenerative lumbosacral stenosis
	Exertional myopathy
	Fibrocartilaginous embolic myelopathy
	Thalamic infarct
Harrier	Cerebellar abiotrophy (Finnish)
	Methionine deficiency-related spinal myelinopathy
Hound	Methionine deficiency-related spinal myelinopathy
	Polyradiculoneuritis
Hovawart	Degenerative myelopathy
Ibizan Hound	Axonopathy (central and peripheral)
	Congenital deafness
Irish Setter	Acquired (idiopathic) laryngeal paralysis
	Cerebellar abiotrophy
	Ceroid lipofuscinosis
	Congenital megaesophagus
	Hereditary quadriplegia and amblyopia
	Idiopathic epilepsy
	Laryngeal paralysis (acquired idiopathic)
	Lissencephaly
Irish Terrier	Muscular dystrophy
Irish Wolfhound	Cervical spondylomyelopathy
	Fibrocartilaginous embolic myelopathy (juvenile)
	Spinal epidural empyema
Italian Greyhound	Cervical intervertebral disc disease
	Congenital deafness
Italian Spinone	Cerebellar abiotrophy
Jack Russell Terrier	Congenital deafness
	Congenital myasthenia gravis
	Hereditary ataxia
	Intracranial arachnoid cyst
	Mitochondrial encephalopathy
	Myokymia/neuromyotonia
	Myotonia congenita
	Neuroaxonal dystrophy
	Sensory neuropathy
Japanese Chin	Atlantoaxial instability
Japanese Spaniel	Gangliosidosis (GM2)
Japanese Spitz	Muscular dystrophy
Keeshond	Idiopathic epilepsy

Table 1.1 (*Continued*)

Kerry Blue Terrier	Cerebellar abiotrophy
	Degenerative myelopathy
	Multisystem degeneration
Kuvasz	Congenital deafness
Labrador Retriever	Acquired (idiopathic) laryngeal paralysis
	Cerebellar abiotrophy
	Congenital deafness
	Exercise intolerance-collapse syndrome
	Idiopathic epilepsy
	Labrador Retriever (central) axonopathy
	Labrador Retriever myopathy
	Leukodystrophy/spongy degeneration (encephalomyelopathy)
	Lumbosacral stenosis
	Myasthenia gravis (acquired)
	Myotonia congenital
	Narcolepsy/cataplexy
	Organic aciduria
	Reflex myoclonus
Lagotto Romagnolo dog	Cerebellar abiotrophy
	Idiopathic epilepsy
Leonberger dog	Laryngeal paralysis/polyneuropathy complex
	Leukoencephalomyelopathy
Lhasa Apso	Congenital hydrocephalus
	Lissencephaly
Lurcher Hound	Hypomyelination/dysmyelination (dysmyelinogenesis)
Malinois Shepherd cross	Spongiform degeneration (gray matter)
Maltese	Chiari-like malformation (CLM)
	Congenital deafness
	Congenital hydrocephalus
	Idiopathic (steroid responsive) tremor syndrome
	Necrotizing meningoencephalitis
	Organic aciduria
Mastiff	Cerebellar abiotrophy
	Cervical spondylomyelopathy
	Extradural synovial cyst
Miniature Pinscher	Atlantoaxial subluxation
	Congenital deafness
	Idiopathic tremor syndrome
	Mucopolysaccharidosis (type 2)
Miniature Poodle	Congenital deafness
Newfoundland	Myasthenia gravis
	Polymyositis
Norwegian Hound (Dunker)	Congenital deafness
Norwich Terrier	Episodic muscle hypertonicity
Nova Scotia Duck Tolling Retriever	Congenital deafness
	Idiopathic epilepsy
	Steroid responsive meningitis arteritis
Old English Sheepdog	Cerebellar abiotrophy
	Congenital deafness
	Mitochondrial myopathy
	Muscular dystrophy
Papillon	Congenital deafness
	Neuroaxonal dystrophy
Pekingese	Atlantoaxial instability
	Congenital hydrocephalus
	Intervertebral disc disease (type I)
	Optic nerve hypoplasia

Table 1.1 (*Continued*)

Pembroke Welsh Corgi	Degenerative myelopathy
	Dermatomyositis
	Intervertebral disc disease (type I)
	Sensory ganglioradiculoneuritis
Pit Bull Terrier	Congenital deafness
Plott Hound	Mucopolysaccharidosis (type 1)
Pointer	Congenital deafness
	Spinal muscular atrophy
Pomeranian	Atlantoaxial instability
	Chiari-like malformation (CLM)
	Congenital hydrocephalus
	Globoid cell leukodystrophy
	Intracranial arachnoid cyst
Poodle (Miniature)	Atlantoaxial instability
	Chiari-like malformation (CLM)
	Cerebellar abiotrophy
	Degenerative myelopathy
	Glycoproteinosis
	Intervertebral disc disease (type I)
	Leukodystrophy/spongy degeneration (brain)
	Narcolepsy/cataplexy
	Optic nerve hypoplasia
	Sphingomyelinosis
	Spinal cord leukodystrophy
Poodle (Standard)	Idiopathic epilepsy
	Organic aciduria (neonatal encephalopathy)
	Polymicrogyria (neuronal migration disorder)
Poodle (Toy)	Atlantoaxial instability
	Congenital hydrocephalus
Portuguese Water dog	Gangliosidosis (GM1)
Pug dog	Arachnoid diverticulum
	Chiari-like malformation (CLM)
	Congenital vertebral malformation (hemivertebra)
	Degenerative myelopathy
	Intracranial arachnoid cyst
	Necrotizing meningoencephalitis
Puli	Congenital deafness
Queensland Blue Heeler	Ceroid lipofuscinosis
Rat Terrier	Muscular dystrophy
Rhodesian Ridgeback	Cerebellar abiotrophy
	Congenital deafness
	Degenerative myelopathy
	Dermoid (pilonidal) sinus
	Myotonia congenital
Rottweiler	Cervical spondylomyelopathy
	Congenital deafness
	Distal sensorimotor polyneuropathy
	Laryngeal paralysis-polyneuropathy complex
	Leukoencephalomyelopathy
	Myopathy (distal)
	Neuroaxonal dystrophy
	Neuronal vacuolation
	Spinal arachnoid cyst
	Spinal muscular atrophy (motor neuron disease)

(*continued*)

Table 1.1 (*Continued*)

Russian Wolfhound	Optic nerve hypoplasia
Saint Bernard	Acquired (idiopathic) laryngeal paralysis
	Congenital deafness
	Episodic dyscontrol (rage syndrome)
	Idiopathic epilepsy
	Narcolepsy/cataplexy
Saluki	Ceroid lipofuscinosis
	Leukodystrophy
	Spinal muscular atrophy (motor neuron disease)
	Spongiform degeneration (gray matter)
Samoyed	Cerebellar abiotrophy
	Cerebellar hypoplasia/lissencephaly
	Congenital myasthenia gravis
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Leukodystrophy/spongiform degeneration
	Muscular dystrophy
	Myotonia congenital (Samoyed cross-breed)
Schnauzer (Giant)	Congenital deafness
	Narcolepsy/cataplexy
Schnauzer (Miniature)	Congenital megaesophagus
	Fibrocartilaginous embolic myelopathy
	Hyperlipidemia (seizures)
	Idiopathic adipsia
	Idiopathic epilepsy
	Intervertebral disc disease (Type I)
	Muscular dystrophy
	Myotonia congenita
Scottish Deerhound	Primary orthostatic tremor
	Vertebral articular process (facet) hypertrophy
Scottish Terrier	Cerebellar abiotrophy
	Congenital deafness
	Episodic muscle hypertonicity (Scotty cramp)
	Leukodystrophy/spongy degeneration (fibrinoid leukodystrophy/Alexander's disease)
	Sensory ganglioradiculitis
Sealyham Terrier	Congenital deafness
Shar Pei	Congenital megaesophagus
Shetland Sheepdog	Congenital deafness
	Dermatomyositis
	Hyperlipidemia (seizures)
	Mitochondrial encephalopathy (Kearnes–Sayre syndrome)
	Spongiform encephalopathy
Shih Tzu	Atlantoaxial instability
	Intervertebral disc disease
	Intracranial arachnoid cyst
Shiloh Shepherd dog	Vertebral articular process (facet) hypertrophy
Shropshire Terrier	Congenital deafness
Siberian Husky	Congenital deafness
	Degenerative myelopathy
	Hereditary laryngeal paralysis
	Sensory ganglioradiculoneuritis
Silky Terrier	Leukodystrophy/spongy degeneration

Table 1.1 (*Continued*)

Smooth-coated Fox Terrier	Congenital myasthenia gravis
	Hereditary ataxia
Soft-coated Wheaten Terrier	Congenital deafness
	Degenerative myelopathy
	Dyskinesia (movement disorder)
Springer Spaniel	Congenital deafness
	Congenital myasthenia gravis
	Episodic dyscontrol (rage syndrome)
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Fucosidosis
Staffordshire Terrier	Chiari-like malformation (CLM)
	Cerebellar abiotrophy
	Myotonia congenita
	Organic aciduria (L-2-hydroxyglutaric aciduria)
Sussex Spaniel	Congenital deafness
	Mitochondrial myopathy
Swedish Lapland dog	Glycogenosis type II
	Spinal muscular atrophy (motor neuron disease)
Sydney Silky Terrier	Glucocerebrosidosis
Terrier Mix	Multiple cartilaginous exostoses
Tibetan Mastiff	Hypertrophic neuropathy
Tibetan Spaniel	Congenital deafness
Tibetan Terrier	Ceroid lipofuscinosis
	Congenital deafness
Toy Poodle	Congenital deafness
Walker Hound	Congenital deafness
	Mononeuropathy
Weimaraner	Cerebellar hypoplasia
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Spinal dysraphism
West Highland White Terrier	Congenital deafness
	Corticosteroid responsive (idiopathic) tremor syndrome
	Globoid cell leukodystrophy
	Organic aciduria (L-2-hydroxyglutaric aciduria)
Whippet	Congenital deafness
	Sensory neuropathy
Wire-haired Fox Terrier	Cerebellar abiotrophy
	Congenital deafness
	Congenital megaesophagus
	Lissencephaly
Yorkshire Terrier	Atlantoaxial instability
	Chiari-like malformation (CLM)
	Congenital deafness
	Congenital hydrocephalus
	Intervertebral disc disease (type I)
	Microvascular hepatic dysplasia
	Mitochondrial encephalopathy
	Myokymia/neuromyotonia
	Necrotizing leukoencephalitis
	Portosystemic shunt (hepatic encephalopathy)
Yugoslavian Sheepdog	Ceroid lipofuscinosis

Table 1.2 Breed-associated neurologic abnormalities of cats.

Abyssinian	Acquired myasthenia gravis
Balinese	Sphingomyelinosis (Niemann–Pick disease, type A)
Birman	Distal polyneuropathy
	Leukodystrophy/spongy degeneration
Burmese	Congenital vestibular disease
	Hypokalemic myopathy
	Meningoencephalocele
Cornish Rex	Congenital deafness (white coat)
Devon Rex	Congenital deafness (white coat)
	Muscular dystrophy
Domestic Short-haired cat	Acquired (idiopathic) laryngeal paralysis
	Ceroid lipofuscinosis
	Globoid cell leukodystrophy (Krabbe's disease)
	Gangliosidosis (GM1)
	Gangliosidosis (GM2)
	Hyperoxaluria
	Mannosidosis
	Metachromatic leukodystrophy
	Mucolipidosis II (I-cell disease)
	Mucopolysaccharidosis (type I) (Hurler's syndrome)
	Mucopolysaccharidosis (type VI) (Maroteaux–Lamy syndrome)
	Muscular dystrophy
	Neuroaxonal dystrophy
	Sphingomyelinosis (Niemann–Pick disease, type C)
	Spinal muscular atrophy
Domestic Tri-colored cat	Neuroaxonal dystrophy
Egyptian Mau	Leukodystrophy/spongy degeneration
Exotic Short Hair	Congenital deafness (white coat)
Himalayan	Esophageal hypomotility
	Fibrotic myopathy
	Pendular nystagmus (congenital)
Korat	Gangliosidosis (GM1)
	Laryngeal paralysis
	Lissencephaly
Manx	Congenital deafness (white -coat Manx)
	Sacrocaudal (sacrococcygeal) dysgenesis
Norwegian Forest cat	Glycogenosis (type IV)
Persian	Cerebellar abiotrophy (late onset)
	Congenital deafness (white coat)
	Mannosidosis-alpha
Rex	Myopathy
Scottish Fold	Congenital deafness (white coat)
Siamese	Cerebellar abiotrophy
	Ceroid lipofuscinosis
	Congenital vestibular disease
	Gangliosidosis (GM1)
	Hypomyelination/dysmyelination (dysmyelinogenesis)
	Mucopolysaccharidosis
	Muscular dystrophy
	Myasthenia gravis
	Pendular nystagmus (congenital)
	Sphingomyelinosis
Somali	Acquired myasthenia gravis
Sphynx	Muscular dystrophy
Turkish Angora	Congenital deafness (white coat)

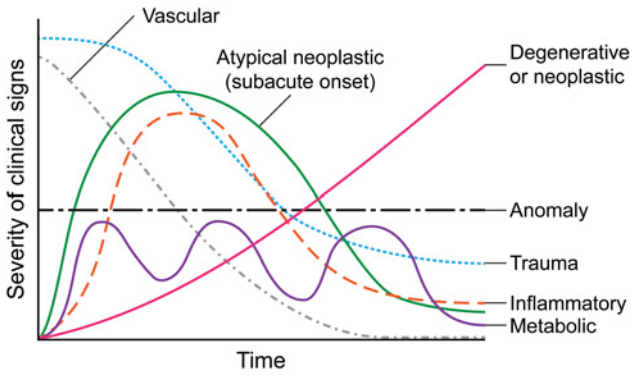

Figure 1.1 Sign–time graph of neurologic diseases. This applies to the majority of cases but there are exceptions in essentially all categories. (The Ohio State University. Reproduced with permission.)

erroneous diagnostic approach. It is important to ask the client to simply state the signs he/she observed, without interpretative connotations, leaving the interpretation of all signs to the clinician.

For any episodic event or signs seen only intermittently, it is very helpful to have a video recording of the event. In this day, video recording is easily available, and in cases where the history is unclear and the neurologic signs inconclusive, it is important to review videos showing the events/episodes to decide on the diagnostic approach.

The neurologic history should allow the clinician to obtain information regarding the possible etiologies. In general, there are expected time course patterns characteristic of certain categories of neurologic disease. Ischemic/vascular and traumatic disorders tend to have peracute onsets (within minutes to a few hours) and often progress minimally or not at all after the initial 24 hrs of onset of clinical signs. Inflammatory/infectious disorders tend to have acute onsets (hours to days) with fairly rapid progression if not aggressively treated. Neoplastic and degenerative disorders often display insidious onset of clinical dysfunction (days to several months) with slower progression of clinical signs (Fig. 1.1). Some degenerative disorders (e.g. type II disc disease) may progress slowly over several years. Many anomalous disorders are characterized by static disease courses, that is the clinical abnormality is recognized at a young age and the disease is nonprogressive. Finally, there are some neurologic disorders that are typically episodic in nature, such as idiopathic epilepsy. As with signalment information, the nature of disease onset and progression is often helpful in ranking differential diagnoses in terms of likelihood for a specific patient, but should be considered as a rough guideline only. There are numerous and notable exceptions to the expectations outlined above. For example, spinal lymphoma in cats is characterized by acute onset of clinical signs.

The history can also provide therapeutic and prognostic information. For example, a large-breed dog with progressive proprioceptive ataxia and paraparesis that received treatment with

corticosteroids and showed no improvement would have degenerative myelopathy as a higher diagnostic consideration, as opposed to one that responded favorably to steroid treatment. Similarly, the duration of clinical signs could provide prognostic consideration. The prognosis for a deep pain negative (absent nociception) paraplegic dog for 2 wks is significantly worse than a dog that has similar signs for 12 hrs.

Listed below are examples of questions that are provided to students at the Ohio State University to guide them in the history taking of patients with neurologic signs.

General questions applicable for most conditions

- When did you first observe the signs?
- Did they appear quickly or slowly (acute or chronic)?
- Are the signs progressing?
- How is the behavior/personality at home? Did you notice any change?
- Have you noticed any mentation changes at home (e.g. quiet, dull, somnolent)?
- Is he/she or was he/she on any medication (try to learn dose and frequency)?
- Have you had any tests (blood work, radiographs, etc.) done for this problem?
- Have you noticed any other sign?
- Does he/she have, or has he/she had, any other medical problems?
- Has he/she had any vomiting, diarrhea, coughing, sneezing?
- How is he/she eating or drinking? What does he/she eat?
- Is he/she updated on vaccines?
- Is he/she indoors/outdoors? Did you travel with him/her?

Questions pertinent to spinal problems (gait problems)

- What is the problem (present complaint)?
- When did you first observe the signs?
- Which limb(s) is (are) affected?
- Did the signs appear quickly or slowly (acute or chronic)?
- Are the signs progressing?
- Do you think he/she is in pain? If so, where?
- If yes, why do you think he/she is in pain?
- Any possibility of trauma? How?
- Has he/she had any similar episodes?
- Are you giving him/her any medicine for this problem?
- Have you noticed any response to treatment(s)?
- Have you had any tests (blood work, radiographs, etc.) done for this problem?

Questions pertinent to seizures and episodic events

When phrasing the questions, be careful to not repeat and reinforce the idea of a specific event like a seizure. Refer to any episodic event as "episodes" or "events."

- Can you please describe the *event* that you observed in details (describe the entire event, i.e. signs before, during, and after the event)?
- How was the muscle tone during the event (e.g. flaccid/floppy or rigid/stiff)?
- Did you notice anything happening on his/her face (e.g. drooling, facial/eyelid twitching)?
- Was the head involved in the episode (e.g. tremors, tilting)?
- Did you observe any evidence of lateralizing signs (one eye/limb more affected)?
- Have you seen any drooling, urination, or defecation associated with the event?
- Was he/she responsive and aware during the event?
- When was the event first noted?
- What is the frequency of these events?
- How long do these events last?
- Are they increasing in frequency or duration?
- How is your dog after the event (evidence of postictal signs)?
- Are the events associated with anything (stress, sleeping, feeding, etc.)?
- How is their behavior/personality at home? Did you notice any change?
- Have you noticed any mentation changes at home (e.g. quiet, dull, somnolent)?
- Is he/she on any anticonvulsant, or any other, medication (try to learn specific drug, dose, and frequency)?
- If on anticonvulsants, ask for results of serum levels.
- Have you noticed any other signs?
- Is he/she indoors/outdoors? Any possible toxin or drug exposure?
- Did you travel with him/her?
- Any family history of the same event?

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CHAPTER 2

Performing the Neurologic Examination

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Introduction

A thorough neurologic examination can be performed in 10 to 15 min. The main components are evaluation of mental status and behavior, gait and postural reactions, cranial nerves, spinal reflexes, palpation, and pain perception. General observation of mental status, posture, attitude, and gait is performed while taking the history. Once the history is clarified, the remainder of the examination is completed. Based on the presenting complaint, it may be necessary to modify portions of the examination. For example, a tetraplegic patient after being hit by a car is not subjected to postural reactions for fear of exacerbating a possible unstable cervical injury. Start with procedures least likely to upset the patient. Disagreeable or painful procedures, such as palpating painful areas, are left until the end of the examination. If the clinician upsets the patient early on, it may be difficult to complete the examination. Also, once pain is elicited, the patient will often anticipate further painful stimuli, making it difficult to determine if other procedures are truly painful. The purposes of the various procedures are explained to the client as the examination proceeds. This lessens the client's distress when he or she observes unfamiliar procedures performed on a pet. Some abnormalities will be blatantly obvious, whereas others will be subtle. A subtle abnormality is still an abnormality. There is a tendency for subtle abnormalities to be chalked up to anything but a neurologic lesion; trust your neurologic findings.

Tools for performing the neurologic examination

A pleximeter (rubber hammer) is used to test myotatic reflexes. Other instruments, such as scissors, are not recommended because these do not provide a consistent stimulus and appear less professional to the client. A hemostat is often useful when testing for nociception (deep pain perception) or eliciting a cutaneous trunci reflex. A strong light source is necessary to elicit pupillary light reflexes in excited dogs and cats. A cotton-tipped applicator or a piece of cotton to cover a hemostat is useful to evaluate the nasal sensation. Finally, a moistened cotton-tipped applicator stick is recommended for performing the corneal reflex. A light touch with your fingertip is acceptable, but if a client is watching you, it may appear that you are poking the pet in the eye.

Performing the neurologic examination[1–12]

A. Mental status and behavior (Video 1)
   1. Before handling the patient, let the patient have the run of the examination room, if ambulatory, and observe the patient's reaction to the surroundings.
   2. Mental status should be evaluated in terms of both level and content of consciousness.
      a. Level of consciousness
         1. Alert—the patient responds appropriately to environmental stimuli.
         2. Depressed/obtunded—the animal is drowsy but arousable. Depressed/obtunded dogs and cats are typically inattentive and display little spontaneous activity.
         3. Stuporous—the patient is in a sleep state, but arousable with a strong stimulus.
         4. Comatose—the patient is unconscious and cannot be aroused even with painful stimuli.
      b. Content of consciousness
         1. Refers to the quality of consciousness
         2. Dementia/delirium—the patient has an alert level of consciousness, but exhibits abnormal behavior and responds inappropriately to stimuli.
      c. Mental status is a function of the ascending reticular activating system (ARAS) that extends over the entire length of the brain stem to activate the cortex. Brainstem disease can cause changes in mental status.
   3. Abnormal behavior is identified by comparing the patient's behavior to expected behavior for animals of a similar breed and age. The client is often able to bring

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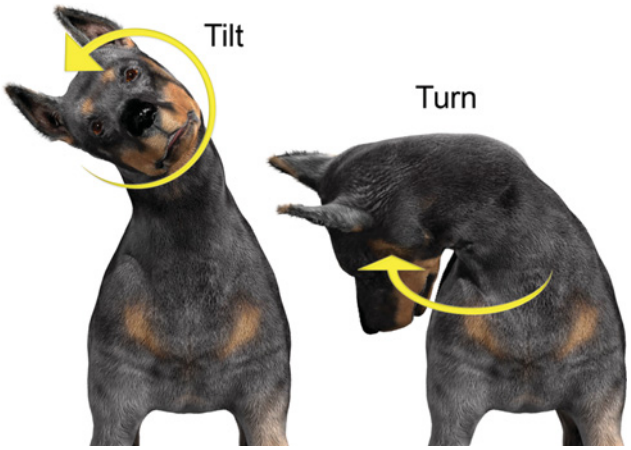


Figure 2.1 A head tilt posture should be differentiated from head turn. Head tilt indicates vestibular disease, whereas head turn suggests a forebrain (thalamocortical) lesion. (The Ohio State University. Reproduced with permission.)

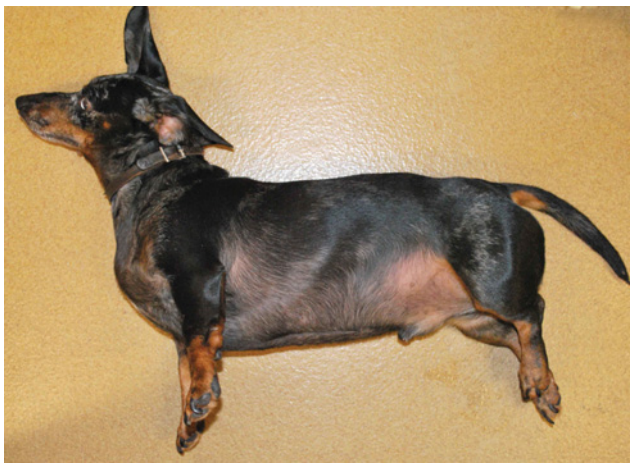


Figure 2.2 Schiff–Sherrington posture in a Dachshund with thoracolumbar intervertebral extrusion.

subtle changes in behavior to the veterinarian's attention. Behavioral changes may be an indication of forebrain (cortical) disease.

B. Attitude/posture
   1. Attitude refers to the position of the eyes and head in relation to the body. Abnormal head position is often manifested as a head tilt or a head turn (Fig. 2.1). In a patient with a head tilt, one ear is held lower than the other. It is also important to make sure that the eye in the affect side is also lower than the other, because sometimes ear diseases can cause a dropped ear without an associated head tilt. Unilateral vestibular dysfunction will often cause a head tilt. When an animal develops a head turn, the head is held level, but the nose is turned right or left. Animals with forebrain lesions may tend to turn their heads and circle in one direction. (Videos 7 and 8).
   2. Posture is the position of the body with respect to gravity. Abnormal postures, such as a wide-based stance, are common in dogs and cats with neurologic disease. Several classic abnormal postures indicative of neurologic dysfunction have been described.
      a. Decerebrate rigidity—due to a brain-stem lesion and characterized by extension of all limbs and sometimes opisthotonus (dorsiflexion of the head and neck). A decreased level of consciousness (often stupor or coma) usually accompanies this posture.
      b. Decerebellate rigidity—due to an acute cerebellar lesion and characterized by opisthotonus, thoracic limb extension, and flexion of the hips. Consciousness is not impaired due to lack of brain-stem involvement.
      c. Schiff–Sherrington posture—frequently encountered in veterinary practice and caused by a lesion in the thoracic or lumbar spinal cord segments. Extension of the thoracic limbs (best appreciated in lateral recumbency) with paralysis of the pelvic limbs characterizes Schiff–Sherrington posture (Fig. 2.2).
      d. Kyphosis, lordosis and scoliosis—abnormal spinal postures frequently observed. A kyphotic posture is commonly seen with painful conditions of the thoracolumbar vertebral column. Scoliosis may be seen with congenital malformation and in cases of caudal occipital malformation (Chiari-like syndrome and syringomyelia). Lordosis is infrequently seen and reflects weakness of the epaxial musculature.
      e. Abnormal limb postures—plantigrade or palmigrade postures are used to describe an abnormal posture of the pelvic or thoracic limb, respectively (Fig. 2.3). These postures are frequently seen in cases of neuromuscular diseases, primarily polyneuropathies, but can also be seen in patients with musculoskeletal diseases.

C. Gait (Video 2)
   1. Lameness
      a. Limb pain can cause a limp when the patient tries to bear weight on a painful limb and then quickly plants the contralateral limb to relieve the pain. As a result, the stride of the painful limb is often shortened. When a single limb is severely painful, it is often carried. This is in contrast to a paretic limb, which is often dragged. Lameness is usually caused by orthopedic disease, but some neurologic lesions—such as attenuation or inflammation of a nerve root or spinal nerve by intervertebral disc extrusion or nerve sheath tumor—can cause lameness. This form of lameness is often referred to as a "root signature."
      b. Patients with bilateral limb pain, such as hip disease or ruptured cruciate ligaments, may not walk at all or

(a) (b)

Figure 2.3 (A) Palmigrade posture in a cat. (B) Plantigrade posture in a dog.

have short-strided, stilted gaits. This can mimic weakness due to neurologic disease.

c. Lower motor neuron (LMN) weakness can cause a short-strided gait in the affected limb(s).

2. Ataxia—inability to perform normal, coordinated motor activity that is not caused by weakness, musculoskeletal problems, or abnormal movements, such as tremor. There are three types:
   a. Sensory or proprioceptive ataxia (Videos 11, 26 and 27)
      1. Loss of the sense of limb and body position due to interruption of ascending proprioceptive pathways (primarily unconscious proprioception).
      2. Characterized by clumsiness and incoordination, resulting in a wide-based stance and a swaying gait. This type of ataxia is often seen in association with paresis. The stride of the affected limb(s) is often longer than normal and the toes may drag or scuff the ground.
      3. Caused by a lesion affecting primarily the white matter of the spinal cord (unconscious proprioceptive pathways).
   b. Cerebellar ataxia (Videos 9 and 21)
      1. Inability to regulate the rate and range of movement (unconscious proprioception).
      2. Characterized by dysmetria, especially hypermetria—an overreaching, high-stepping gait.
      3. Caused by cerebellar disease or selective dysfunction of spinocerebellar tracts (less likely).
   c. Vestibular ataxia (Videos 8, 19 and 20)
      1. Unilateral vestibular lesions cause leaning and falling to one side. Other signs of vestibular disease, such as head tilt and abnormal nystagmus, may be evident.
      2. With bilateral vestibular dysfunction, the patient maintains a crouched position, is reluctant to move, and exhibits side-to-side head movements, without an obvious head tilt (since both vestibular receptors and nuclei are affected).
3. Paresis/paralysis

   Paresis is a partial loss of voluntary movement. This is manifested as a decreased rate or range of motion, increased fatigability, decreased muscle tone, or limited ability to perform certain motor acts. Paralysis (plegia) is a complete loss of voluntary movement. Paresis or paralysis indicates a lesion of either the upper motor neuron (UMN) system or the lower motor neuron (LMN) system. It is not possible to discriminate between UMN weakness and LMN weakness based solely on the severity of the weakness.
4. Abnormal movements
   a. Tremor—a rhythmical, oscillatory movement localized to one region of the body or generalized to involve the entire body. A terminal tremor, or intention tremor, occurs as the body part nears a target during goal-oriented movement. This is most evident as a head tremor when the patient attempts to sniff an object, eat, or drink. A postural tremor occurs as the limb or head is maintained against gravity.
   b. Myotonia—delayed relaxation of muscle following voluntary contraction. Myotonia is manifested as muscle stiffness that is relieved by exercise. Attacks of myotonia may culminate in recumbency with rigid extension of the limbs. Some patients with myotonia will display "dimpling" of sustained indentation of affected muscle when percussed.
   c. Myoclonus—a brief, shock-like muscle contraction producing a quick, jerking movement of a body part.

(a) (b)

Figure 2.4 Proprioceptive positioning is evaluated with the patient supported in a standing position in the pelvic (A) and thoracic limbs (B). Proper support of the patient is essential. The dorsal surface of the paw is placed on the floor without pushing it down. The patient should immediately replace the paw to a normal position.

D. Postural reactions (Video 3)

Postural reactions test the same neurologic pathways involved in gait, namely the proprioceptive and motor systems. Their main value is detecting subtle deficits or inconspicuous asymmetry that may not be obvious during the observation of gait. Postural reactions are also useful in discriminating between orthopedic and neurologic disorders. Frequently it is only necessary to perform two postural reaction tests: proprioceptive positioning and hopping.

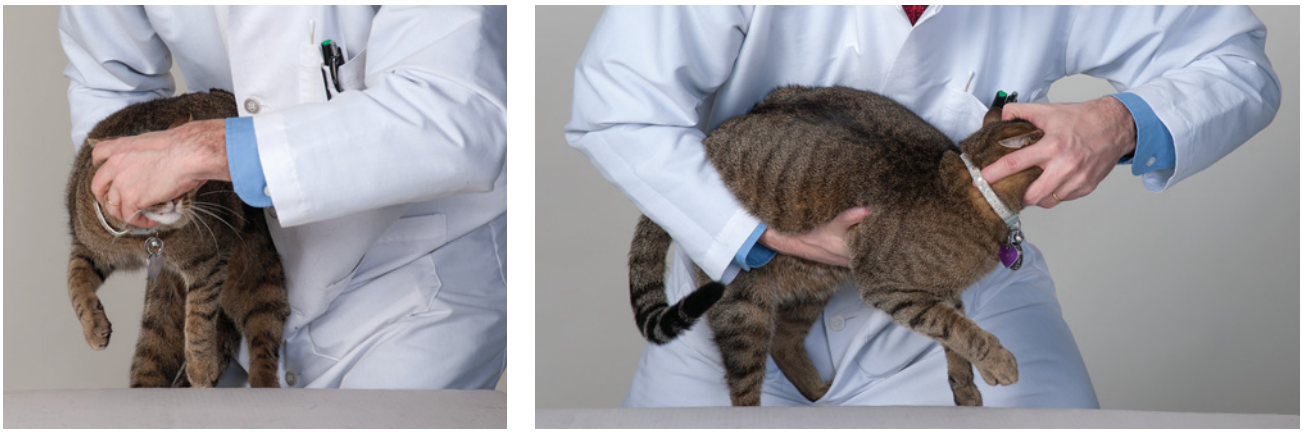
1. Proprioceptive positioning
   - a. Support the animal to avoid body tilt and turn one paw over so that the dorsal surface is in contact with the ground. The patient should immediately return the foot to a normal position (Fig. 2.4).
   - b. When properly supported, most patients with orthopedic disease will have normal proprioceptive positioning. On the other hand, proprioceptive pathways are often compromised early in the course of neurologic diseases, so defects in proprioceptive positioning may be detected before there are obvious signs of weakness.
2. Hopping
   - a. Hold the patient so that the patient's weight is supported by one limb and move the animal laterally. The amount of support and the technique are different in small dogs or cats (Fig. 2.5 A and B), compared to large dogs (Fig. 2.6 A and B). Normal animals will hop on the limb while keeping the foot under their body for support.
   - b. Each limb is tested individually and responses on the left and right are compared. This is a sensitive test for subtle weakness or asymmetry.
3. Placing response
   - a. The nonvisual (tactile) test is performed first. Cover the patient's eyes, pick the animal up, and move it toward the edge of a table. When the paw touches the table, the animal should immediately place the limb forward to rest the paw on the table surface (Fig. 2.7).
   - b. Visual placing is tested similarly, except the patient's eyes are not covered. The normal response is to place the paws on the surface as the table is approached, before the paws make contact with the table. This test may detect visual deficiencies.
4. Hemiwalking, wheelbarrowing, and extensor postural thrust
   - a. These tests can be performed if other postural reactions are equivocal.
   - b. For hemiwalking, hold up the limbs on one side of the body and move the patient laterally (similarly to the technique demonstrated in Fig. 2.6). The normal reaction is as described for the hopping response.

(a) (b)

Figure 2.5 The hopping response in small dogs and cats is tested by lifting and supporting the patient such that most of its weight is borne on one limb. The patient is moved laterally to test the thoracic limb (A) and pelvic limb (B). Note how the support and position of the hands differ.

(a) (b)

Figure 2.6 The hopping response in large dogs can be tested by just lifting the contralateral limb and moving the patient laterally to test the thoracic limb (A) and pelvic limb (B). It is unnecessary to try to hop medially or cranially.



(a) (b)

Figure 2.7 Tactile placing is tested by covering the patient's eyes and moving the patient toward the edge of a table. The test can be brought toward the table frontally (A), or laterally (B). Normal dogs and cats will place their paw on the table as soon as they contact the edge of the table.

c. Wheelbarrowing in the thoracic limbs is done by supporting the patient under the abdomen so that the pelvic limbs do not touch the ground and moving the patient forward (Fig. 2.8). Normal animals will walk with symmetrical, alternate movements of the thoracic limbs.

d. Extensor postural thrust is tested by lifting the patient by the thorax and lowering the pelvic limbs to reach the floor. Normal patients will move the pelvic limbs caudally as soon as they touch the floor (Fig. 2.9).

E. Cranial nerves (CN) (Fig. 2.10, Table 2.1) (Video 4)

1. CN I (olfactory nerve) is not routinely tested. After ascertaining patency of the nostrils, cover the patient's eyes and present a morsel of food beneath the nose, observing for normal sniffing behavior. Irritating substances, such as ammonia or isopropyl alcohol, should not be used, because they stimulate trigeminal nerve endings in the nasal passages and produce false results.

Figure 2.8 Wheelbarrowing is tested by supporting the patient under the abdomen so that the pelvic limbs do not touch the ground and moving the patient forward.

Figure 2.9 Extensor postural thrust is tested by supporting the patient under the thorax and lowering it until it touches the ground.

2. CN II (optic nerve)

a. Note pupillary size and any anisocoria before actually testing the pupillary light reflex (Fig. 2.11). There should be a direct and consensual pupillary light reflex in each eye (Fig. 2.12).

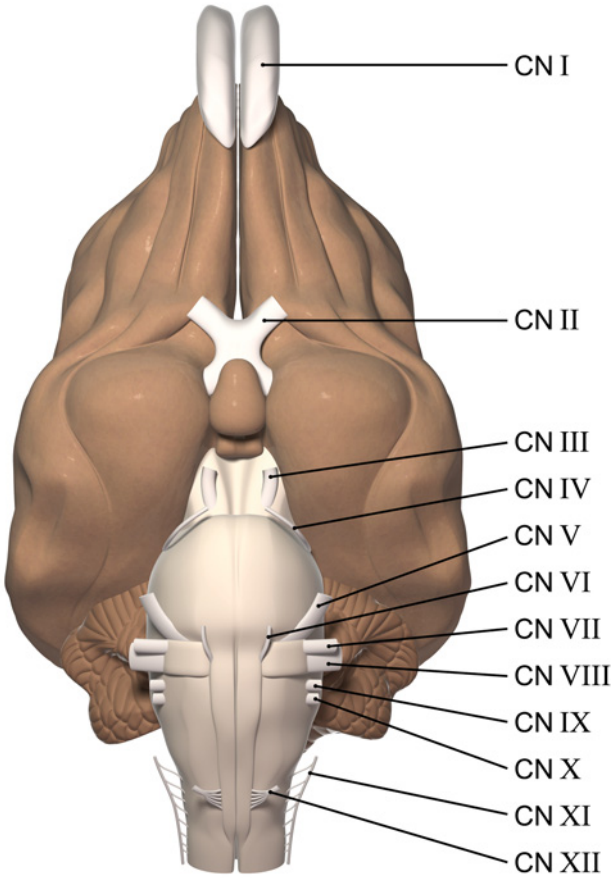


Figure 2.10 Ventral aspect of the canine brain, showing the relative anatomic positions of the cranial nerves. (The Ohio State University. Reproduced with permission.)

Table 2.1 Cranial nerves and their function

Cranial nerve	Function/Innervation
CN I	Olfaction
CN II	Vision
CN III	Somatic motor to most of the extraocular muscles (dorsal, medial, ventral rectus; ventral oblique; levator palpebrae superioris)
	Parasympathetic innervation to pupil (pupillary light response)
CN IV	Somatic motor to dorsal oblique muscle of the eye
CN V	Somatic motor to muscles of mastication
	Somatic motor to tensor tympani muscle
	Sensory to most of face
CN VI	Somatic motor to lateral rectus and retractor bulbi muscles (extraocular)
CN VII	Somatic motor to muscles of facial expression
	Somatic motor to stapedius muscle
	Parasympathetic innervation to salivary glands (mandibular, sublingual)[a] and lacrimal, palatine, and nasal glands[b]
	Sensory to inner pinna
	Sensory (mechanoreception, thermal) and taste to rostral 2/3 of tongue (chorda tympani nerve)[c]
CN VIII	Vestibular function and hearing
CN IX–XI	Parasympathetic innervation of viscera (CN X)
	Parasympathetic innervation to salivary glands (parotid and zygomatic, CN IX)[d]
	Sensory and taste to caudal 1/3 of tongue (CN IX)
	Sensory innervation of pharynx (CN IX and X)
	Somatic motor for laryngeal and pharyngeal function (nucleus ambiguus)
CN XII	Somatic motor to extrinsic and intrinsic tongue muscles

[a] Postganglionic axon in CN V, mandibular branch (after mandibular and sublingual ganglia).
[b] Postganglionic axon in CN V, maxillary branch (after pterygopalatine ganglion).
[c] Chorda tympani nerve joins lingual branch of mandibular branch of CN V near middle ear.
[d] Postganglionic axon in CN V, mandibular branch (after otic ganglion).

b. Menace response. Move your hand toward the patient's eyes in a threatening manner, observing for a blink response (Fig. 2.13A). Make sure you test the ability to blink before menacing the patient. The menacing hand should stop about half to one foot away from the patient's face. This would avoid generating air currents that would stimulate the ophthalmic branch of the trigeminal nerve and cause false positive results. By covering the contralateral eye, you can test the nasal (medial) and temporal (lateral) visual fields of each eye. The efferent part of this reaction is controlled by the facial nucleus and nerve (CN VII). The menace response may be deficient in puppies and kittens (less than 12 wks) due to cerebellar immaturity.

c. The menace response evaluates the ipsilateral optic and facial cranial nerves, as well as the ipsilateral cerebellum (ipsilateral) and the contralateral forebrain (thalamocortex) (Fig. 2.13B).

d. Visual following. Drop cotton balls or move a toy or ball in front of the patient and observe if the patient's eyes and head follow the object.

3. CN III (oculomotor nerve), IV (trochlear nerve), and VI (abducent nerve) are considered together because they control eye movements. CN III also mediates pupillary constriction (parasympathetic function), which is evaluated by the pupillary light reflex.

a. Strabismus may be obvious or can be detected by shining a light on the cornea. When the eyes are aligned, the light reflection is on the same area in each eye.

b. Observe spontaneous eye movements when the patient looks about. Move the patient's head from side to side and up and down to induce horizontal and vertical nystagmus.

c. Oculocephalic reflex, vestibulo-ocular reflex or physiologic nystagmus is elicited by moving the patient's head side to side and up and down. Normal physiologic nystagmus has a fast phase in the direction of the head movement (Fig. 2.14A). This test evaluates

Figure 2.11 The size and pupillary symmetry are tested by holding a good light source one to two feet away from the patient's face so that the light shines equally on both eyes.

Figure 2.12 The pupillary light reflex is elicited by shining a bright light in each eye. Normally, there is a brisk constriction of the ipsilateral (direct pupillary light reflex) and contralateral pupil (indirect or consensual pupillary light reflex). There is no need to cover the contralateral eye when testing the direct pupillary light reflex.

cranial nerves VIII (sensory) and III, IV, VI (motor) (Fig. 2.14B). In small dogs or cats, primarily those with significant cervical pain, the test can be performed moving the entire patient sideways (Fig. 2.15).

d. To induce the corneal reflex, touch the cornea with a cotton-tipped applicator moistened with saline. Corneal sensation depends on the ophthalmic branch of the trigeminal nerve. The normal response is a retraction of the globe, mediated by the abducent nerve (CN VI).

4. CN V (trigeminal nerve)
   a. Motor portion—the temporalis and masseter muscles are visualized and palpated to detect any swelling, atrophy, or asymmetry (Fig. 2.16). If there is bilateral weakness, the patient may not be able to close the mouth.
   b. Sensory portion
      1. Ophthalmic branch—this branch of CN V can be evaluated via the corneal reflex (discussed above) and by specifically touching the medial canthus of the eyelid region during the palpebral reflex (Fig 2.17A). The efferent part of this reflex is dependent on normal function of the facial nucleus and nerve (CN VII).

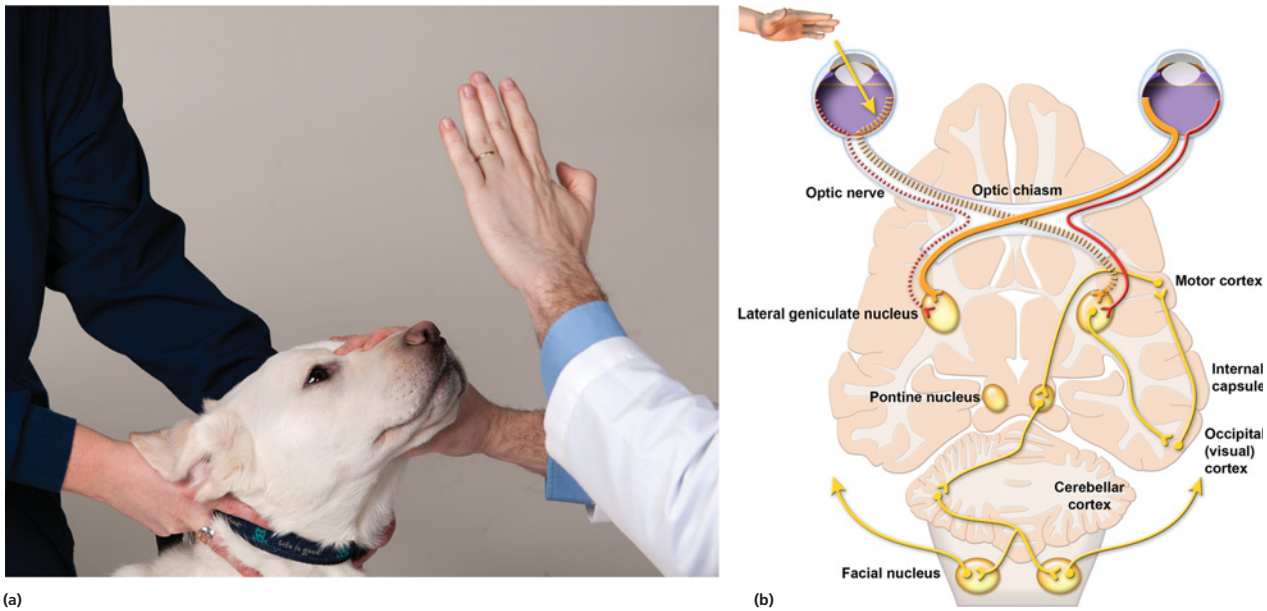


Figure 2.13 (A) The menace response is elicited by making a threatening gesture at the eye, which should induce a blink. Be careful to not get too close to the eye. (B) Pathways and structures involved in the menace response—optic (II) and facial (VII) nerves, as well as the cerebellum and thalamocortical (forebrain) regions. (The Ohio State University. Reproduced with permission.)