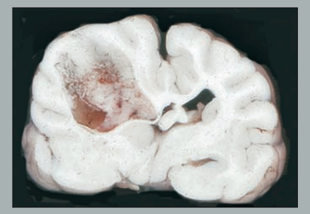
This book is a comprehensive compilation of small animal neurology in all its clinical aspects.

The first part of the book provides a detailed explanation of the clinical neurological investigation and the additional investigations (such as laboratory tests, electrodiagnostics and neuroradiology) that are necessary for the diagnosis of neurological disease. A discussion of the most important areas of neurosurgery and the basis of neurogenetics and acupuncture complete the general part of the book.

The second part of the book deals with the clinical aspects of neurology. Individual diseases are considered according to the different segments of the nervous system – from the peripheral to the central nervous system.

Using 585 clinical, radiographic, MRI and CT pictures as well as numerous drawings and diagnostic illustrations, the authors present a large variety of visual aids from their specialty areas and provide detailed information on the diagnosis and therapy of neurological disease in the dog and cat.

In the field of neurology, the assessment of movement is a central element in the diagnosis of disease. The book's CD-ROM demonstrates, using video clips, the course of a clinical neurological investigation and also a number of well-chosen neurological case studies – a valuable didactic medium for self-study.



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Small Animal Neurology

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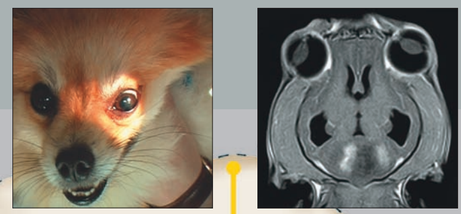
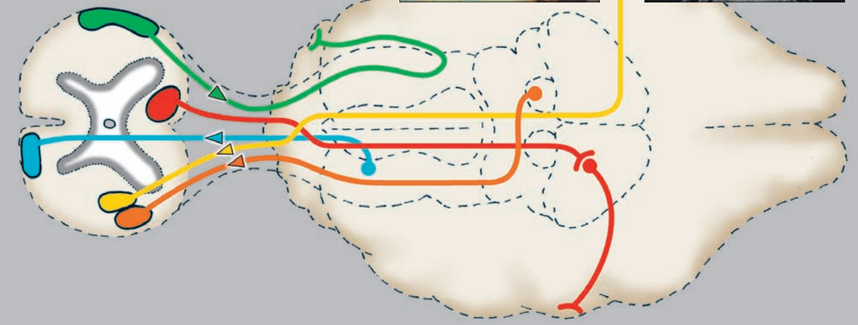
André Jaggy (ed.)

Small Animal Neurology

An Illustrated Text

Associate editor
Simon R. Platt





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English translation
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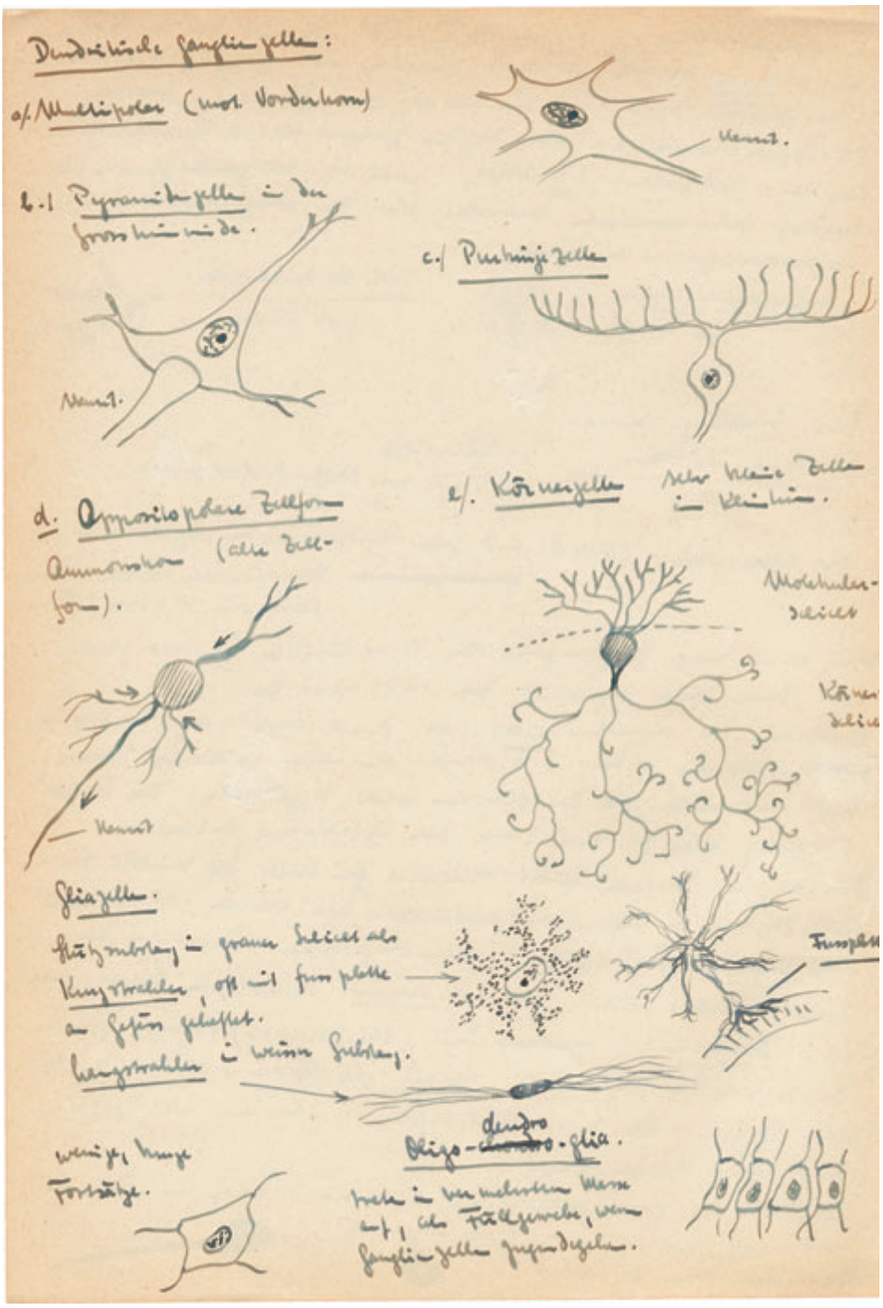
Frontispiece: Hand-drawn sketch, neurophysiology introductory course: part 2

To the memory of Rudolf Frankenhauser



"Everything should be made to be as simple as possible, but no simpler"
ALBERT EINSTEIN

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Preface to the English edition

It has been a great honour to edit the English language version of this extremely comprehensive veterinary neurology and neurosurgery text. This is a textbook which could proudly sit next to any of authoritative neurology books of our time. The attention to detail and its clarity in presentation make this a phenomenal reference for veterinary students, clinicians in general practice, neurologists in training, and board certified neurologists all around the world.

In editing this book for a wider English speaking audience, I was able to draw on my experience of clinical neurology in referral practice and academic environments in Europe and in the USA. Such experience has allowed me to understand the views and needs of students and clinicians both in learning and undertaking veterinary neurology from many walks of life. The benefit of the time I have been glad to spend sharing my views with students, interns, residents, and colleagues', learning from them as I go, has helped me undertake this editing role and it is to them that I dedicate this text.

It is a testament both to the authors of this book and the tremendous worldwide advances made in veterinary neurology that only minor additions and amendments were necessary ensuring that disease predispositions, clinician preferences and drug availabilities were geographically addressed. Diagnostic capabilities vary within small geographical areas, let alone between countries, and between the variety of practices and hospitals where we all work. However, this textbook has successfully addressed this issue without the need for any adaptation for the English speaking audience. To me, as I read through this work, it served as tremendous evidence of the globalisation of veterinary neurology and a phenomenal inspiration. We should all be proud, wherever we feel we are in this profession, of the contributions we have made to help in whatever small way to accomplish this.

It would be unfair to identify any of the chapters within this book above another. However, in identifying the unique and therefore outstanding aspects of this book, the chapters on neuroimaging, neurosurgery, neuropharmacology and electrodiagnosis deserve special attention by the reader. The inclusion of MRI based neuroanatomy and frequently used drug appendices are exceptional.

I would like to thank Dr. Andre Jaggy for his friendship and insight over the years and most of all for this excellent opportunity to be involved with this outstanding book.

Athens, October 2009
Simon R. Platt

Preface to the second German edition

»Im eigenen Auge schau mit Lust
Was Plato von Anbeginn gewusst!
Und will Dir's nicht von selbst gelingen
*So wird Purkinje Dir es bringen.«**

JOHANN WOLFGANG VON GOETHE

The text and figures in the chapters of this second edition have been revised and amended. We have remained true to our maxim of describing the organic diseases so that their weighting, classification, or grouping caters for both the student as well as the practicing veterinarian.

In addition, two renowned specialists – Prof. Spreng and Dr. Sigrist – were won over to allow inclusion of their profound knowledge about emergency medicine into the redesigned Chapter 12. Using simple flow diagrams and clear statements, they have been able to present the complexity of neurological emergencies in a didactically precise manner. Furthermore, the interested reader will find an excellent presentation of magnetic resonance images and macrosections in Appendix 1 (under the title »Comparative sectional anatomy of the canine and feline brain«). The experts – Prof. Lang and Prof. Vandevelde in cooperation with Drs. Gassner, Rossi, and Konar – have made a solid contribution to the understanding of neurological diagnostics with their work here. A clear overview of the most common sequences in MRI diagnostics is given and their implementation is explained in an understandable manner using clinical examples.

I wish to give my thanks to all the coauthors for their contributions. The revision of this book has also caused the publishers an especially large amount of work; therefore, I wish to give a big thanks to Dr. Ulrike Oslage, without whom the second edition would not have been so quickly completed. I would also like to especially thank Bettina Sodemann for her exertions and careful editing.

I wish to send heartfelt thanks also to all the readers and friends whose critical comments have led to the removal of inaccuracies or mistakes. Indeed, we would be thankful for any future critical comments.

Bern, Spring 2007
André Jaggy

* *»With your own eye show with pleasure, What Plato knew from the start! And if you cannot do it on your own, So will Purkinje teach you.«*

Preface to the first German edition

This book unites a highly motivated and predominantly internationally renowned group of neurologists, radiologists, internists, surgeons, anaesthetists, cardiologists, pathologists, geneticists, behavioural therapists and parasitologists. It was my job, as professor of neurology and neurosurgery at the Vetsuisse Faculty in Bern, to plan, organise and edit the different chapters so that the book is homogeneous – which is most evident in the clinical chapters (12–19).

Most of the neurologists come from the **Bern School of Clinical Animal Neurology**, which was set up by Prof. Marc Vandevelde and then continued by myself in the late 1980s. I am grateful to Prof. Vandevelde and my early mentor – Prof. John Oliver, University of Georgia, USA – as both of them taught me how important it is to always combine clinical experience with relevant research results. I have tried to fulfil this premise with respect to the knowledge in my area of research – epilepsy in the dog – and later in my tuition of students and the further education of veterinarians, both nationally and internationally.

This book consists of two parts. The first part includes the process of the neurological examination, an introductory chapter on neuropathology as well as a somewhat detailed discourse of adjunct examination methods such as electro-diagnosis, laboratory investigations and radiology; without which neurology could not be conceivable. Detailed information about anaesthesia, pharmacology and rehabilitation is also included. The first part is rounded off by chapters on neurosurgery, neurogenetics and acupuncture. The second part of the book is dedicated to the clinical aspects of neurology and is subdivided according to the different sections of the nervous system: from the peripheral structures such as nerves and muscles to the higher centres of the brain.

The practised reader will quickly notice that the different chapters are subdivided according to the acronym VITAMIN D and then follow the respective incidences of the diseases.

Despite its extensiveness, this book does not under any circumstances purport to be all encompassing. It is the authors' intentions that the book describes the most important aspects of neurological disease. The literature listed in the text enables the reader to easily acquire in-depth information about the individual facets. The diagnosis of neurological disease is not conceivable without the examination methods shown in great detail in the illustrations. However, the authors see in these methods primarily a means of confirming a previously formulated tentative clinical diagnosis. The latter is achieved as a rule from a careful anamnesis, a thorough clinical investigation and a precise localisation. May this book serve in the further development of this knowledge.

A CD-ROM has been produced as an accompaniment to the book to provide a better understanding of the neurological examination and so, the anatomical localisation of the lesion. Nine neurological cases for self-evaluation have been included. My thanks go to Dr. Fabrice Hamann and Dr. Sam Jaggy, who both did so much for the realisation of this project.

The authors also wish to thank Dr. Ulrike Oslage of the Schlütersche Verlagsgesellschaft for the careful design and production of this book.

Many people have helped in the acquirement of literature and pictures, in the critical appraisal of the chapters and the careful production of the appendices. Our especial thanks go to Tim Bley, Yvonne Reimer, Ales Tomek, Martin Konar and Patrick Kirchner. We also wish to particularly thank Stan Demiere for the graphics and line drawings. In addition, special thanks should go to my colleague, Johann Lang for the radiographs, CT and MRI pictures.

I feel a need to thank all those who have helped us in various ways in our work: the academic and technical co-workers at the Clinic for Small Animal Medicine; the veterinarians and specialists who referred patients to us – without these, we as a predominantly referral practise would be on a very weak footing – the students who challenge us daily, who criticize and bring us back to reality, who give meaning to our teaching and provide us with pleasure. Merci …

Bern, October 2004
André Jaggy

For Danielle
My thanks to her for her love, support and understanding
as well as for my sons, Thomas, Stefan and Samuel.

Abbreviations

5-HT	5-hydroxytryptamine (serotonin)
AASL	atlanto-axial subluxation
ACE	angiotensin converting enzyme
ACh	acetylcholine
ACP	acupuncture
ACTH	adrenocorticotrophic hormone
AEP	auditory evoked potentials
ALAT	alanine aminotransferase
ALD	acral lick dermatitis
ALT	alanine aminotransferase
ANA	antinuclear autoantibodies
ANS	autonomic nervous system
AP	alkaline phosphate
aPPT	activated partial thromboplastin time
AST	aspartate aminotransferase
ATP	adenosine triphosphate
BAEP	brain stem auditory evoked potentials
BD	Borna disease
BDV	Borna disease virus
BID	twice daily
BSE	bovine spongiform encephalopathy
cAMP	cyclic adenosine monophosphate
CBASS	completely balanced steady state
CBC	complete blood count
CBF	cerebral blood flow
CCSM	cervical caudal spondymyelopathy
CD	cognitive dysfunction
cGMP	cyclic guanidine monophosphate
CK (= CPK)	creatine kinase (= creatine phosphokinase)
CMAP	compound muscle action potential
CMO	craniomandibular osteoarthropathy
CMO_2	cerebral oxygen consumption
CN	cranial nerve
CNS	central nervous system
CPP	cerebral perfusion pressure
CRC	corrected reticulocyte count
CRH	corticotrophin releasing hormone
CSF	cerebrospinal fluid
CT	computed tomography
cTSH	canine thyroid stimulating hormone
CVP	central venous blood pressure
DD	differential diagnosis
DIC	disseminated intravascular coagulation
DISH	disseminated idiopathic skeletal hyperostosis
DLSS	degenerative lumbosacral stenosis
DV	dorsoventral
EAP	electroacupuncture
ECT	emissions computer tomography
ED_{50}	effective dose 50
EEG	electroencephalogram / electroencephalography
EMG	electromyogram / electromyography
EPA	tissue-type plasminogen activator
EPSP	excitatory postsynaptic potential
ERG	electroretinogram
Ext	extensors
FeLV	feline leukaemia virus
FET	functional electrical therapy
FIP	feline infectious peritonitis
FIPs	fibrillation potentials
FIV	feline immunodeficiency virus
FLAIR	fluid light attenuation inversion recovery
Flex	flexors
FLM	fasciculus longitudinalis medialis
FR	reticular formation
free T_4 or fT_4	free biologically active thyroxin fraction
FSE	feline spongiform encephalopathy
GABA	γ-aminobutyric acid
Gd	gadolinium
Gd-DTPA	gadolinium diethylenetriaminepenta-acetic acid
GFAP	glial fibre protein
GGT or γ-GT	gamma glutamate transferase
GI	gastrointestinal
GLDH	glutamate dehydrogenase
GME	granulomatous meningoencephalitis
GOBF	giant onion bulb formation
Hct	haematocrit
HE	hepatoencephalopathy
HU	Hounsfield units
HVSF	high voltage, slow frequency (activity)
Hz	Hertz
ICP	intracranial pressure
IgA	immunoglobulin A
IgG	immunoglobulin G
IgM	immunoglobulin M
IM	intramuscular
IP3	inositol triphosphate
IPSP	inhibitory postsynaptic potential
IR	inversion recovery
IU/kg	international units / kilogramme body weight
IV	intravenous
LAD	leucocyte adhesion protein deficiency
LDH	low density lipoproteins
LMN	lower motor neuron
LMNS	lower motor neuron system
LSD	lysosomal storage disease
LVFA	low voltage, fast activity
MAC	minimal alveolar concentration
MAO	monoamine oxidase
MAOB	monoamine oxidase B
MAP	mean arterial blood pressure

MCE	multiple cartilaginous exostoses
MCHC	mean corpuscular haemoglobin concentration
MCV	mean corpuscular volume
MEP	miniature endplate potential
MHC	major histocompatibility complex
mNCV	motoric nerve conduction velocity
MP	myotonic potential
MPS	mucopolysaccharidosis
MPSS	methylprednisolone sodium succinate
MR	magnetic resonance
MRI	magnetic resonance imaging
mRNA	messenger RNA
NCV	nerve conduction velocity
NMDA	N-methyl-D-aspartate
NMR	nuclear magnetic resonance
NSAID	non-steroidal anti-inflammatory drug
OAAM	occipito-atlanto-axial malformation
OCD	osteochondritis dissecans
$PaCO_2$	arterial carbon dioxide partial pressure
PaO_2	arterial oxygen partial pressure
PCR	polymerase chain reaction
PET	positron emission tomography
PLR	pupillary light reflex
PMMA	polymethylmetacrylate
PMP	pseudomyotonic potentials
PNCV	peripheral nerve conduction velocity
PNS	peripheral nervous system
PO	per os, orally
PSS	portosystemic shunt
PSW	positive sharp waves
PU /PD	polyuria/polydipsia
Pw	proton weighted
QID	four times daily
REM	rapid eye movement
RMI	reticulocyte maturation index
RNA	ribonucleic acid
RNS	repetitive nerve stimulation
RPI	reticulocyte production index
RT-PCR	reverse transcriptase polymerase chain reaction
SA	stimulation artefact
SBM	submaximal stimulation
SID	once daily
SSRI	selective serotonin reuptake inhibitor
SSS	sick sinus syndrome
SPECT	single photon emission tomography
SPM	supramaximal stimulation
SRME	steroid-responsive meningoencephalo-myelitis
STIR	short T1 inversion recovery
$t_{1/2}$	elimination half-life
T1	T1 relaxation time
T1w	T1-weighted
T2	T2 relaxation times
T2w	T2-weighted
T_4	serum thyroxin
TBE	tick-borne encephalitis
TCM	traditional Chinese medicine
TENS	transcutaneous electrical nerve stimulation
TID	three times daily
tPA	tissue plasmogen activator
TPA	tissue polypeptide antigen
TRH	thyrotropin releasing hormone
TSH	thyroid stimulating hormone
UE	uraemic encephalopathy
UMN	upper motor neuron
UMNS	upper motor neuron system
V	volt
VD	ventrodorsal
VLDH	very low density lipoprotein
WHO	World Health Organisation

V
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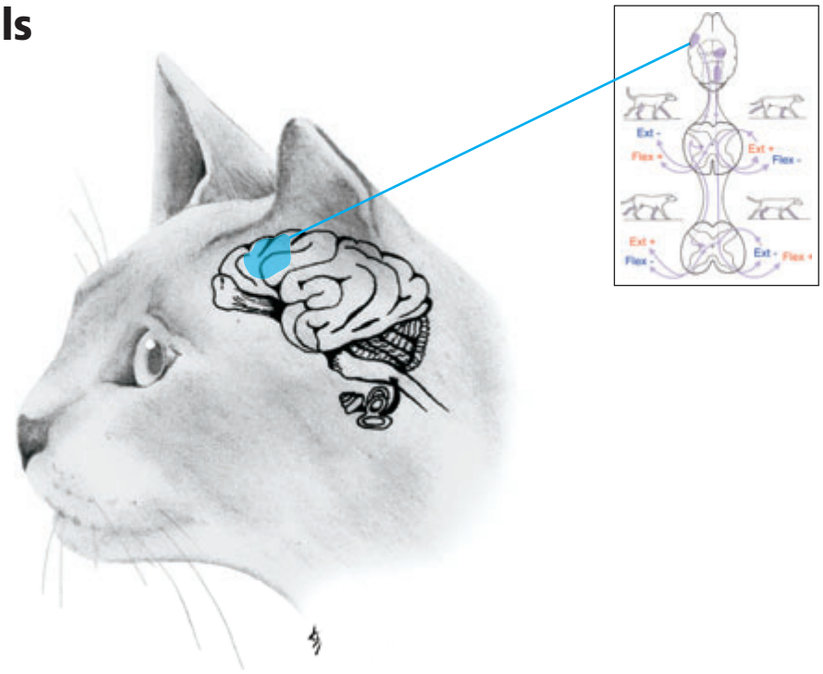
N
D

Instructions for the Reader

The authors have followed the acronym VITAMIN D for the clinical diagnosis of neurological diseases. Due to their colour coding, these tables can be quickly found in the respective chapters. If no clinically relevant disease has been documented, the letter for that group of diseases has been left out.

1 Neurological Examination of Small Animals

André Jaggy
Bernhard Spiess



The neurological examination is the basis of clinical neurology and serves to recognize abnormal clinical signs. Identifiable pathological symptoms form the fundamental framework of neurological syndromes.

The aims of the examination are:

- Confirmation and differentiation of neurological and non-neurological abnormalities.
- Localisation of the lesion in the central (CNS) or peripheral (PNS) nervous system.
- Determination of the severity of the lesion(s).
- Development of a differential diagnosis and prognosis.

The neurological examination, therefore, forms an integral part of the clinical examination. After the determination of the localisation and the suspected diagnoses, more specific investigations can be undertaken to establish the exact cause of the disease which will help to predict a more accurate prognosis.

1.1 Signalment

Some neurological diseases are more common in specifc breeds (e.g. disc herniation in the Dachshund, epilepsy in the Golden Retriever, syringomyelia in the Cavalier King Charle's spaniel or hydrocephalus in the Chihuahua), while others are more common at certain ages (e.g. hereditary and infectious disease in puppies and young dogs, degenerative or neoplastic changes in older / geriatric animals). In some diseases, a sex predisposition has been observed (see Appendix 2). In addition, functionally related disturbances in the CNS due to hypoglycaemia can occur in dogs trained for specific purposes (e.g. police or hunting dogs) or in toy breeds on poor diets.

1.2 Anamnesis

The neurological examination always begins with a determination of the history of the case (anamnesis). This is the initial and most important element on the way to determining the differential diagnosis and in certain cases can be used to predict the diagnosis. The observations and comments of the owner or care-giver are essential for the interpretation of all further examinations, but should be judged carefully. Certain clinical signs can be better delineated / defined by well aimed questioning. The onset and course of the disease (acute, chronic, intermittent, progressive, non-progressive, relapsing) must be taken into consideration. An acute onset of clinical signs can indicate (a) toxicosis, (b) trauma, (c) vascular insult, (d) inflammatory disease, or (e) neoplasia. In comparison, a

chronic progression can be indicative of a degenerative, neoplastic or infectious aetiology. It is important to determine whether or not the process is progressive (as in degenerative myelopathy in the German Shepherd dog), non-progressive (as in traumatic lesions) or relapsing (often seen with disc herniations). The description of the initial clinical signs as well as of the subsequent disease development can help to determine a focal (such as vascular or neoplastic disease) vs. a multifocal process (such as inflammation).

Some diseases, such as idiopathic epilepsy in the Golden and Labrador Retrievers, occur in families. In such cases, information about the patient's origin (family tree analysis) can provide the basis for a genetic cause of the disease. Information about possible behavioural disturbances and/or changes in personality should be determined in discussion with the owner/care-giver. This information can be important in the determination of the lesion localisation and the differential diagnosis. For instance, the acute occurrence of unmotivated anxiety attacks or untargeted aggression can indicate either a functional psychomotor epilepsy (temporal lobe epilepsy) or a space-occupying lesion in the cerebrum.

Sometimes there is a relationship between the animal's nutritional status and neurological symptoms; for example, hypervitaminosis A or thiamine deficient encephalopathy, most commonly seen in the cat. The initial origin of the animal (kennel, import, stray, etc.) is important. Previous medical diseases, concurrent medications, toxin exposure as well as the animal's vaccination and worming/parasite status should all be taken into consideration.

1.3 General examination

When there is suspicion of a neurological disease, examination of the extraneural body functions is essential. Accordingly, the general clinical examination plays an integral part in the neurological investigation. Numerous clinical signs can indicate a primary organic disease outside of the CNS or PNS. Even when the patient has been presented with well-defined neurological signs, a systemic cause of the dysfunction (e.g. liver insufficiency, endocrine disease or sepsis) should be investigated.

The differentiation between a focal and a generalised disease process or a primary vs. a secondary disturbance can in some cases be achieved during a general examination. For example, infectious diseases which affect the nervous system are often associated with clinical signs occurring in other organ systems (e.g. respiratory or gastrointestinal symptoms with distemper or toxoplasmosis).

Clinical experience teaches the veterinarian to use routine laboratory investigations and more specialised tests both in a targeted and an economic manner. A useful rule is that the longer a case remains unclear, the more specialised investigations need to be implemented.

1.4 Neurological status

1.4.1 Neurological examination methods

The neurological examination must be undertaken methodically. A logical sequence should be established so that incorrect conclusions are not reached and so that the results can be easily assessed. It is important to write down all the results so that no test or result is forgotten (Table 1.1).

The examination undertaken should be as complete as possible but may be varied from case to case, depending on the size, breed and personality of the animal. Using the following sequence of tests, both the CNS and the PNS are systematically examined from the "higher" to the "lower" centres. The neurological examination can be broken down into several important subdivisions including observation (information about mental status, behaviour, posture and movement), palpation (muscle mass, muscle tone, pain), cranial nerve function, spinal reflex function, superficial sensory function and postural reaction testing.

Manipulations that cause either excitement or pain should not be undertaken at the start of the examination (e.g. the testing of flexor reflexes or deep pain). In order to gain the trust of the animal, it is sometimes advisable to undertake the examination in a playful manner. If the animal is frightened or puts up a fight, then many reflexes and reactions are difficult to interpret.

1.4.2 The sequence of the neurological examination

While questioning the owner or care-giver, the level of consciousness, behaviour, posture and movement of the animal should be carefully observed. The behaviour and movement can be examined in depth at a later time (see Chaps. 1.4.3.2 and 1.4.3.4).

1.4.3 The main aspects of the examination

1.4.3.1 Mental status

The state of consciousness, the animal's behavioural patterns and the ability of the patient to interact with its environs are assessed. Small differences in standard behaviour can be indi-

Table 1.1: Neurological Examination

1. Consciousness: normal / abnormal; obtunded / stupor / coma _____

2. Behaviour: normal / abnormal _____

3. Posture: normal / abnormal _____

4. Gait: normal / abnormal _____

5. Cranial nerves

left		right
_____	II Vision	_____
_____	Menace response (II, VII)	_____
_____	II Cotton ball test	_____
_____	II + III PLR	_____
_____	Stimulation left	_____
_____	Stimulation right	_____
_____	III, IV, VI Strabismus	_____
_____	VIII + III, IV, VI Nystagmus	_____
_____	V Sensory	_____
_____	V Chewing	_____
_____	VII Facial	_____
_____	V, VII Corneal	_____
_____	V, VII Palpebral	_____
_____	IX, X Swallowing	_____
_____	X Sensory	_____
_____	XI Neck muscles	_____
_____	XII Tongue	_____
Muscle palpation		_____
Otoscopy		_____
Ophthalmoscopy		_____
_____	II Fundus	_____
Comments:		_____

6. Postural reactions

left		right
_____	*Hopping*	
_____	Front	_____
_____	Back	_____
_____	*Knuckling*	
_____	Front	_____
_____	Back	_____
_____	*Righting*	
_____	Extensor Postural Thrust	_____
_____	*Wheelbarrow*	_____
_____	*Visual placing*	_____
_____	*Neck extension*	_____
_____	*Placing test*	_____
_____	Tactile	_____
_____	Optical	_____
_____	*Neck reactions*	_____

7. Spinal reflexes

left		right
	Forelegs	
_____	Ext. carpi radialis	_____
_____	Flexor	_____
	Hindlegs	
_____	Patellar	_____
_____	Tibialis cranialis	_____
_____	Flexor	_____
	Other	
_____	Perineal	_____
_____	Vulvourethral	_____
_____	Panniculus	_____

8. Sensation

_____	Superficial	_____
_____	Deep pain	_____
_____	Hyperaesthesia	_____
_____	Hypaesthesia	_____
_____	Analgesic zone	_____

Comments: _____

Evaluation: –2 absent; –1 reduced; 0 normal; +1 increased
PLR: pupillary light reflex

vidual, breed or family specific. Labrador Retrievers, Golden Retrievers and Persian cats are in general very cooperative and sociable. In contrast, other breeds are rather exuberant and difficult to examine. The history of the animal's actions and the owner's opinion will help to classify its behaviour as being normal or not.

Anatomy and physiology

The reticular formation, an extensive nuclear area in the brain stem, extends from the medulla oblongata to the diencephalon and receives information via the majority of the sensory tracts (exteroception, interoception and proprioception), which it then diffusely projects to the cerebral cortex (Fig. 1.1). The functions of the reticular formation are as follows: maintaining consciousness, reflex and reaction readiness, maintenance of alertness and the control of sleep-related activity levels

A focal or diffuse lesion in the brain stem or a diffuse bilateral lesion in the cerebral cortex can lead to an interruption in this feedback control system; the cause of which can be primary or secondary.

Examination and assessment

The level of consciousness can be determined by observing the animal and from the answers to the following questions: Is the animal attentive? Does it react with its environment? Does it react to various stimuli (calling, touching or pain)?

The four levels of consciousness are:

- Normal.
- Obtunded: the animal is awake but disinterested. Almost every disease can be the cause of this state.
- Stuporous: the animal is unconscious and can only be aroused by the application of strong stimuli (e.g. pain). This state is mainly observed in association with an interruption between the reticular formation and the cortex.
- Comatose: the animal exhibits a deep loss of consciousness and does not react even to painful (noxious) stimuli. A coma is often observed when there is a complete interruption between the reticular formation and the cortex. A lesion of the brain stem is the cause in the majority of these cases.

1.4.3.2 Behaviour

Behaviour is the result of a very complex series of physiological processes, whose anatomic features lie in the cerebral cortex and the limbic system.

Anatomy and physiology

Behaviour is controlled by the limbic system and is the result of the interactions of stimuli from the environment and those which originate from within the body itself (Fig. 1.2). Behavioural disturbances can either be of a primary (in association with primary CNS dysfunction) or a secondary nature (e.g. subsequent to systemic disease).

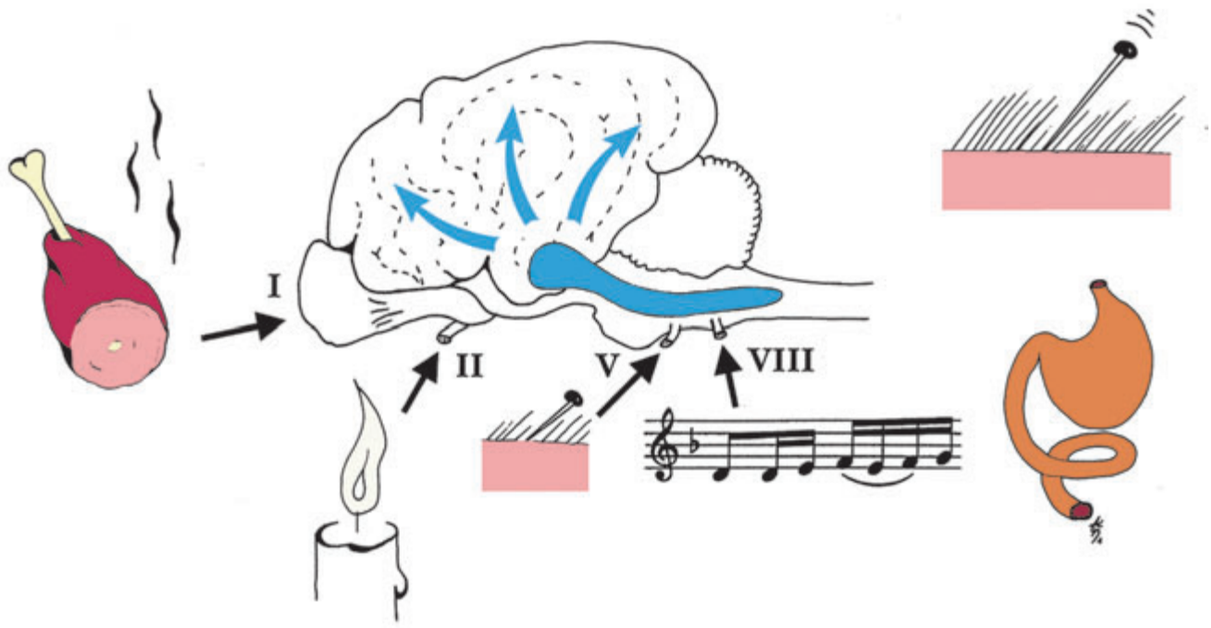


Fig. 1.1
Consciousness. The reticular formation, a fine network that extends from the upper cervical spinal cord to the thalamus, is primarily responsible for consciousness. The rostral part of the brain stem forms a key part in this relay system. It transmits stimuli directly (without an interchange) or indirectly (with an interchange) from the periphery to the cortex, which is neurophysiologically subordinate to it.

Examination and assessment

Fear, aggressiveness, shyness or disorientation are examples of abnormal behaviour. Often these are associated with chewing, licking, yawning and compulsive pacing movements. These signs can rarely be associated with the dysfunction of a single anatomical structure, as the manifestations of disease of the cortex and limbic system are very similar. An indispensable part of the examination is a discussion with the owner or care-giver targeted at elucidating any changes in behaviour as well as prolonged observation in a relaxed environment.

1.4.3.3 Posture

A healthy animal takes on a normal body posture when a standing as it counters the effects of gravity on its body weight. The limbs are in extension and a the line running through the pelvis, back, and chest is parallel to the ground, while the head is held in balance with the neck.

Anatomy and physiology

A reflex arc ensures normal posture. The afferent pathway is the sensory part and this transmits the necessary information from various receptors in the limbs and body, from the eyes, and from the vestibular system via sensory nerves to the CNS. The vestibular receptors are mainly responsible for the appreciation of movement and changes in the position of the head. Receptors in the limbs (e.g. stretch receptors) report on the tension within the muscles, tendons, and the joint capsules. Such information is sent partly via the cerebellum and the brain stem. It is interpreted in the cortex and then transmitted via the efferent (motor) tracts to the α- and γ-motor neurons that innervate the neck, body, and limb muscles. The cerebellum and the vestibular apparatus are also responsible for the maintenance of normal body posture.

Examination and evaluation

When observing the animal, the posture of the head, neck, trunk, and limbs are evaluated. In many cases, variations from the norm are first seen during movement (dynamic posture).

The characteristics of abnormal posture are:

- Head tilted (principally with vestibular lesions).
- Head turned sideways / pleurothotonus (mainly with cerebral lesions).
- Head and neck tilted sideways / torticollis (chiefly with brain stem lesions).
- Head and neck flexed downwards (especially with vestibular brain stem lesions or cervical lesions).
- Kyphosis (especially with spinal cord lesions in the thoracolumbar region; Fig. 1.3a).
- Scoliosis (lateral curvature of the spine; Fig. 1.3c) and lordosis (ventral curvature of the spine; Fig. 1.3b) in diseases of the spinal column.

Reduced muscle tone in a limb or more than one limb results in a wide-based gait or knuckling of the feet (especially with lesions affecting the lower motor neurone system [LMNS]).

Increased muscle tone in one or more limbs is conspicuous as a hyperextension of the affected limbs (stiffness, spasticity). This is seen particularly with lesions of the upper motor neurone system (UMNS).

Opisthotonus (extension of the head and neck) is observed with lesions of the rostral brain stem.

Decerebrate rigidity, an involuntary extension of all the extremities in response to external stimuli, arises with lesions in the rostral brain stem / mesencephalon / pons. In some cases, this can occur in combination with opisthotonus.

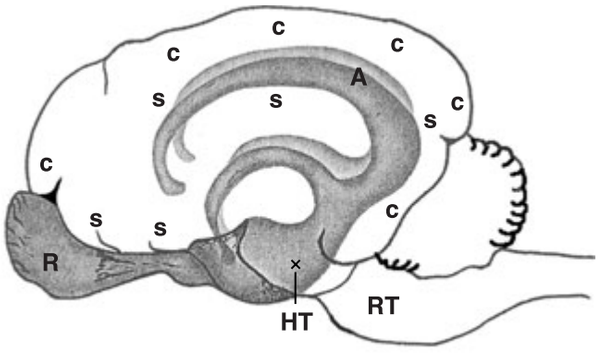


Fig. 1.2
The limbic system consists of the rhinencephalon (R), the hypothalamus (HT), the hippocampus (A), the subcortical (s) and cortical (c) centres and part of the reticular formation (RT). This system is responsible for the complex behaviour of animals.

1.4.3.4 Movement

Anatomy and physiology

Movement with displacement of the body from one place to another in principle requires the movement of the body's centre of gravity forwards, to the side or backwards. This process is regulated by the **afferent** (spinocortical) and **efferent** (corticospinal) tracts (Fig. 1.4a) as well as the **locomotion centres** (Fig. 1.4b).

Afferent nerves

The afferent sensory pathways are categorized according to their target and modality, into spinocortical and the spinocerebellar pathways (Fig. 1.4a).

Efferent nerves

In order to understand the concept of the efferent motor pathways, it is necessary to explain the concepts of the lower motor neuron (LMN) and the upper motor neuron (UMN) (Fig. 1.4a).

The LMN

The lower motor neurons originate as cell bodies in the ventral horn of the grey matter of the spinal cord and the motor centres in the brain stem. There are two types of neurons, α and γ, each of which innervate the striated muscle. Both of these types of neurons can be stimulated or even inhibited at the segmental, intersegmental and suprasegmental levels. The summation of these stimuli determines whether or not the LMN is sufficiently activated to elicit a contraction of the musculature. As the stimulation of the LMN results in muscle contraction, a lesion in the LMN causes a flaccid paresis or paralysis, with reduced or absent reflexes and neurogenic muscle atrophy. Such clinical-neurological phenomena do not occur in lesions of the UMN. Anatomically, the LMN system is composed of the ventral horn (α-motor neurons), the ventral root, spinal nerves, the peripheral nerves, the neuromuscular end-plate and the muscles (target organs).

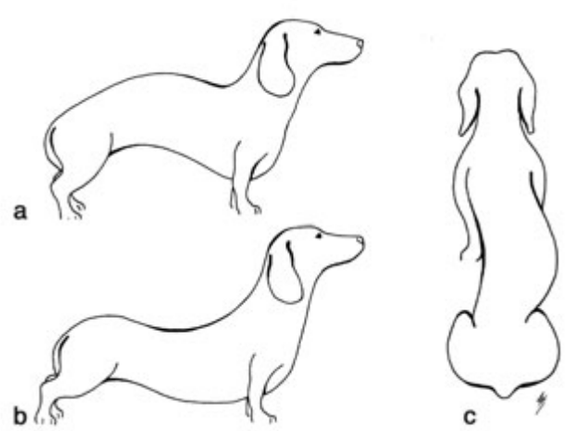


Fig. 1.3a–c
Abnormal body positions: kyphosis (a), lordosis (b) and scoliosis (c).

The UMN

The upper motor neurons (UMN) are suprasegmental neurons, which directly or indirectly influence the LMN or LMNS. In human beings, the UMN is found in the primary motor cortex and affects the LMN or LMNS via the UMNS (corticospinal tract).

The UMNS is more extensive in veterinary neurology as it includes not only **neurons in the cortex**, but also ones in the **basal nuclei**, the **brain stem** and the **cerebellum**. The UMNS influences the LMN via interneurons. The overall effect of the UMNS is an inhibition of the LMN. A lesion in the UMN or UMNS results in a loss of this inhibition (dysinhibition) of the LMN. If the segmental reflex arc remains

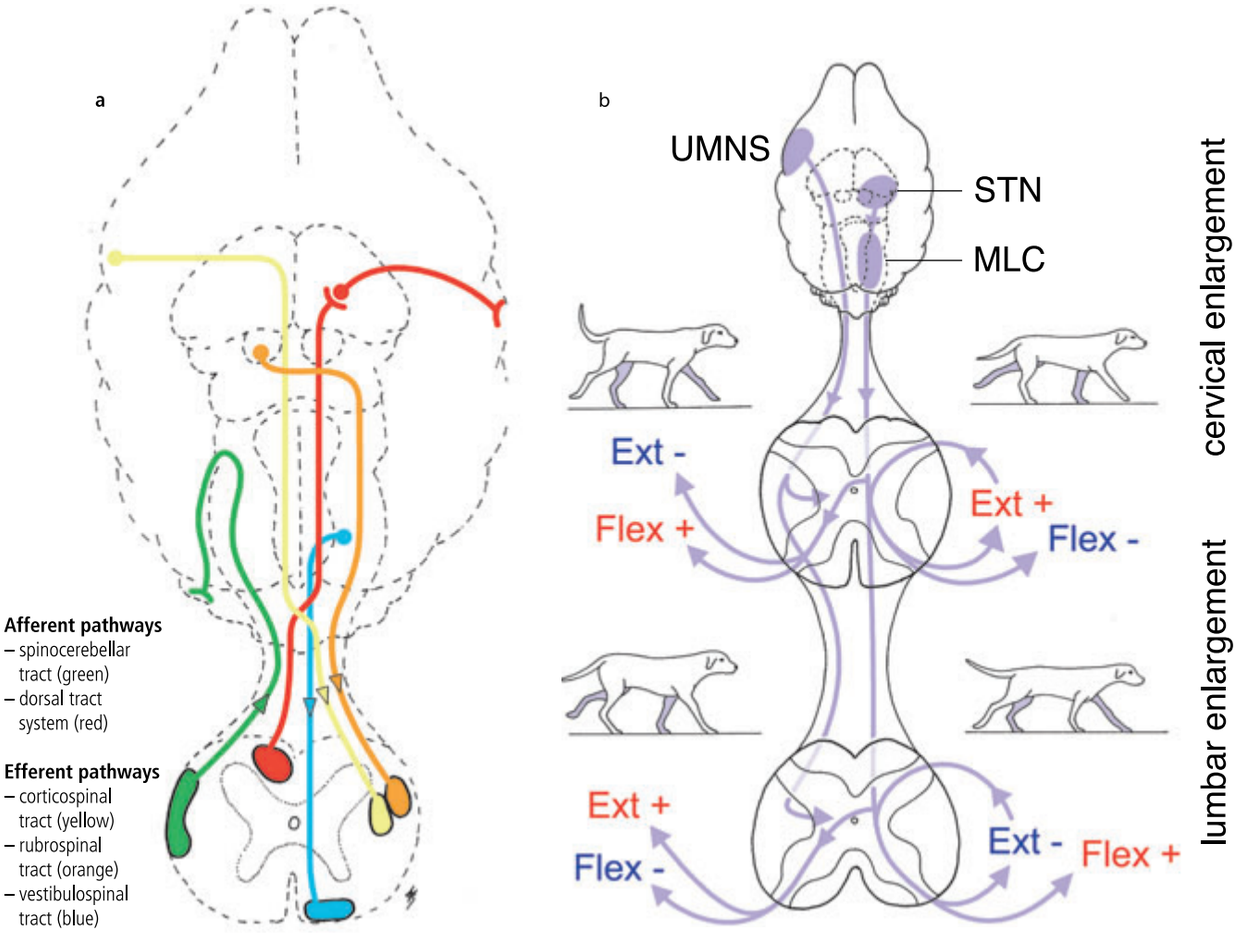


Fig. 1.4a, b
(a) Ascending and descending pathways. The ascending sensory pathways are divided into two main groups according to their target and their modality. In the first group, information reaches the consciousness via the spinocortical tracts, which end in the cerebrum. The second group which deals with the stimuli "subconsciously" end in the cerebellum via the spinocerebellar tracts. This information is very important for the feedback of reflex activation and modulation during movement. In addition to these primary endings, the ascending tracts give off collateral branches, which have synapses at the level of the spinal cord (and that enter the dorsal horn for the activation or inhibition of segmental or intersegmental reflexes) and the brain stem (via the dorsal spinocerebellar tracts to the reticular formation and which are important in the brain stem reflexes). The descending pathways are divided into an upper motor neuron system (UMNS) and a lower motor neuron system (LMNS).
(b) Physiological gait consists of an afferent and an efferent part coming from the cortex, thalamus (subthalamic nuclei, STN), brain stem (mesencephalic locomotion centre, MLC) and the spinal locomotion centres. Each limb has its own pacemaker, which is located in the cervical enlargement for the forelimbs and the lumbar enlargement for the hindlimbs. These two spinal enlargements are coordinated with each other and are influenced by a motor centre in the mesencephalon, which in turn is influenced by a higher centre in the subthalamus. Stimulation of this system results in an alternating flexion (swing phase) and extension (support phase) of the limbs. Together these two dynamic elements form a single step. The flexor and extensor reflexes are proof for the presence of the pacemaker.

intact, the reflexes can be exaggerated (hyperreflexia). The muscle tone can either be normal or increased (spastic). The degree of paresis depends on the extent of the lesion affecting the UMNS.

In addition to an understanding of the LMN and UMN systems, an understanding of the concept of the mechanisms responsible for locomotion, is important in the appreciation of the processes involved in movement.

Locomotion centres
The locomotion centre for the forelegs lies in the cervical enlargement, whilst that for the hindlegs is in the lumbar enlargement (Fig. 1.4).

Relays at the level of a single limb
The stimulation of the skin afferents of a limb results in an activation of the motor neurons of the flexors and an inhibition of the motor neurons of the extensors in the same limb. As a result, the limb is flexed.

Stimulation of the afferent muscle spindles causes an activation of the motor neurons of the extensor muscles and an inhibition of the feedback system of the antagonistic flexor muscles in the same limb. The limb is then extended (support phase).

Relays at the level of a pair of limbs (thoracic or pelvic)
If a limb is in the initial swing phase (extensors –; flexors +), for example in the left leg, then the contralateral limb influenced via an internal feedback circuit comprising segmental reflex arcs, will be in the support phase (extensors +; flexors –). In this manner, it is possible for the animal to balance out its centre of gravity. The crossed extensor reflex indicates damage to this internal feedback circuit; with lesions of the thoracolumbar spinal cord, stimulation of the skin of one pelvic limb, results in an extension of the contralateral limb, in addition to flexion of the stimulated limb. In chronic cases of paraplegia, due to lesions at the level of the thoracolumbar spinal cord, every form of external stimulus can initiate an alternating motor response in the limbs.

In an animal with paraplegia due to a thoracolumbar lesion, it is certainly possible for an incoordinated and involuntary gait (spinal walking) to arise. In spinal walking, the cervical pacemaker centre is stimulated by a displacement of the centre of gravity resulting in the thoracic limbs taking a step forwards. The concurrent displacement of the pelvic region then stimulates the pacemaker centre of the pelvic limbs, and so they then take a pace forwards, albeit in an incoordinated manner

Relays at the level of both pairs of limbs
The coordination of the thoracic limbs with the pelvic limbs is regulated through the cervical and the lumbar enlargements (pacemaker centres). For the production of such movements, both the afferent and the efferent pathways work in consortium with the propriospinal tracts.

Higher centres of locomotion
The mesencephalic locomotion centre (MLC) is responsible for the coordination of spontaneous movement and lies symmetrically and bilaterally at the level of the rostral mesencephalon. Electrical stimulation of this centre leads to both normal and spontaneous movements. Directed spontaneous movement is not possible after experimental sectioning of the rostral mesencephalon. As the subthalamic nuclei (STN) are responsible for the determination or orientation of spontaneous movement, these nuclei are superior to the MLCs.

If a lesion is in the STN itself, then the animal's gait is almost normal; what is missing in such patients is an ability to determine their movements. Often, such patients present with the urge to be always be on the move.

In conclusion, all the important relay stations responsible for the normal sequence of movements involved in gait lie in the subthalamus, the mesencephalon and the spinal cord.

Influences on the locomotion centres
The complex systems described above are physiologically inferior to the cerebral cortex, the cerebellum and the vestibular apparatus. The cerebral cortex is responsible for "will" and decision-making, the cerebellum for the fine tuning of movement and muscle tone, and the vestibular apparatus is responsible for balance and sustaining muscle tone in conjunction with the cerebellum. The influence of these structures on the locomotion centres takes place via the descending motor pathways.

Corticospinal tracts
The motor pathways are subdivided according to their course and their localisation. They are divided into the pyramidal and the extrapyramidal tracts. The **pyramidal tracts** originate in the cerebral motor cortex and extend as "pyramids" to the medulla oblongata. Some tracts leave the main "pathways" at the level of the brain stem to go directly to the brain stem nuclei (corticolbulbar tracts). The rest of the fibres cross over at the level of the medulla and then descend through the spinal cord as the corticospinal tracts (in the lateral funiculi). These tracts synapse via interneurons on α-motor neurons. A lesion of this system results in spastic paralysis. The corticospinal tracts exert their greatest influence **on the distal limb musculature** and are most highly developed in primates, but have minimal function in companion veterinary species.

Extrapyramidal tracts
These tracts have their greatest influence on the proximal limb musculature. They are made of polysynaptic circuits of differing complexities. The **basal ganglia**, **subthalamic nuclei** and the **substantia nigra** (in the mesencephalon) comprise the largest part of this system. The nuclei are connected with the cortex via polysynaptic loops. These regulatory circuits have a direct influence on the nuclei in the mesencephalon and the reticular nuclei in the pons and medulla. The descending tracts of these brain stem and vestibular nuclei have an indirect influence on the LMN.

The descending tracts can be characterised according to their functionality. There is a direct connection between the functional effects of the LMNS and its phylogenetic development. The phylogenetically oldest system is responsible for posture. It has a greater positive effect on the anti-gravity muscles (extensors) than on the gravity muscles (flexors). These tracts contain vestibulospinal and recticulospinal "transmission lines" and are found in the ventral funiculus. The phylogenetically newer motor system is responsible for the initiation and modulation of spontaneous and rhythmic motor actions (running, jumping, etc.). To achieve this, the extensor tonus must be partially reduced, which is made possible via the lateral funiculi (corticospinal, rubrospinal and lateral reticulospinal tracts).

The corticospinal tracts are far better developed in human beings than in animals. Their animal counterparts are the ru-

brospinal tracts. The red nucleus is directly affected by the motor cortex via the phylogenetically younger cortico-rubrospinal tracts. Older tracts influence the red nucleus indirectly via the motor cortex and the basal nuclei. The rubrospinal tracts cross caudal to the red nucleus and descend via the lateral funiculi to the segmental interneurons in the dorsal horn of the spinal cord. The vestibulospinal tract is one of the phylogenetically oldest parts of the motor system. The vestibular nuclei receive only a little information from the cortex. They are mainly stimulated by the peripheral vestibular apparatus (inner ear) and modulated by the cerebellum.

Examination and assessment

Abnormality of the gait can be seen as proprioception disturbances, paresis or paralysis, circling movements, ataxia and/or dysmetria.

Disturbances in proprioception

Proprioception is the mechanism involved in the self-regulation of posture and movement through an awareness of the position of the limbs in space. A deficit results in an abnormal placing of the feet or knuckling of the limbs, which is not necessarily seen with every step taken. It can also result in one of the three types of ataxia.

Paresis or Paralysis

Paralysis or plegia is defined as the complete inability to activate one or more muscles. Paresis is an incomplete paralysis. The affected limbs show either inadequate or no contraction of their musculature. Depending on the clinical signs, the following terms are used: **monoparesis** (paresis of one limb); **paraparesis** (paresis of both hindlimbs); **tetraparesis** (paresis of all four limbs); **hemiparesis** (paresis of an ipsilateral pair of limbs); and **claudication** (limping or lameness). The cause of paresis or paralysis lies in a failure of the motor function of a nerve or its target organ, e.g. the muscle. The lesion can lie either in the UMNS or the LMNS. One, therefore, speaks of a UMN or LMN paresis or paralysis.

Depending on the tone in the musculature, paresis or paralysis can be divided into three forms: spastic (increased tone = UMN or central lesion), flaccid (reduced tone = peripheral or myogenic [LMN unit] lesion, or a rarely a chronic central lesion resulting from disuse of the limb(s)) and intermittent paresis or paralysis.

Circling

Clinically, circling movements can be defined as a drifting to one side with either large or small diameters, or as a turning motion. The direction of the displacement is towards the side of the lesion in the majority of cases, but not all. Circling can be seen with prosencephalic or brainstem lesions. Concurrent torticollis and/or head tilt occur mainly in association with lesions in the vestibular system.

Ataxia

A disturbance in the coordination of movement or collaboration of muscle groups is called ataxia. Ataxia can, but not always, be associated with paresis or involuntary movements depending on the localisation of the causative lesion. The degree of the ataxia present may be described as slight, medium or severe ataxia, although this is qualitative, and may be focal or generalised in its distribution. Depending on the location of the responsible lesion, ataxia can be subdivided into peripheral/spinal (proprioceptive), cerebellar, or vestibular forms. The clinical picture depends on the type and form of the ataxia, though it is always important to look for incoordinated movements. A base wide stance, crossing over of the limbs, shortened or elongated stride length are all typical of the majority of the different forms of ataxia. The responsible lesion can be neuro-anatomically located utilising the additional clinical signs present in the patient, and the type of ataxia can be defined. For example, when the cerebellum is affected hypermetria, intention tremors and loss of the menace response may be seen in addition to the ataxia.

Dysmetria

Dysmetria describes stride lengths that are either too big (hypermetria) or too small (hypometria). Often "goose-stepping" strides can be observed: the stride is stopped suddenly and the patient sways. Dysmetria of the head is best seen when the patient drinks or eats and is often termed an intention tremor. The distance to the target (food bowl) is usually overestimated or even underestimated. This form of ataxia can occur with lesions of the cerebellum or the cerebellar pathways.

1.4.4 Postural reactions

Postural reactions entail complex normal regulatory processes in which motor, sensory and coordination pathways are involved. Testing of the postural reactions enables the examination of not only the afferent pathways but also the efferent ones, too. Such testing is also useful in registering subtle abnormalities or for a clearer recognition of a neurological problem. It is very important to compare all four limbs with each other because these tests often show differences between the right and left half of the body, which can assist with developing a differential diagnosis list

1.4.4.1 Hopping on one leg or a pair of legs

Peripheral nerves, the spinal cord, brain stem, cerebellum and cerebrum are all involved in hopping. The hopping test is undertaken as follows: the patient is held so that it stands on one leg or a pair of legs. The patient is then pushed to one side (Fig. 1.5).

Fig. 1.5
Hopping on a foreleg.

Fig. 1.6
Knuckling of the left hindleg.

An **extension of the supporting leg(s)** followed by a quick correction whilst being pushed to one side is considered as being normal. Abnormal reactions include a **reduced speed of reaction** (mainly a proprioception deficit), an **abnormal placement of the foot** (motor deficit), **hypometria** defined as a reduced correction reaction (lesion in the spinal cord [C6–T2]) and **hypermetria** (cerebellar lesion, spinocerebellar pathways).

Patients with lesions in the cervical spinal cord cannot hop or stand normally on the ipsilateral pair of limbs.

1.4.4.2 Knuckling

With the knuckling test, both the sensory afferent pathways and the motor efferent pathways in the limb are tested. The former are responsible for the perception of the position of the limb in space, while the latter are responsible for the normal placement of the foot. The distal part of the limb is flexed, so that the dorsal surface of the metacarpus (or metatarsus) lies on the floor. The animal should put its limb back in its normal position within a second. It is remarkable that the musculoskeletal diseases which cause severe disturbances in gait are associated with little or no disturbances in this reaction (Fig. 1.6).

1.4.4.3 Righting reaction

With this test, the ability of the patient to take on a normal position in the gravitational field is assessed. Of the four systems which play a part in maintaining a balanced posture, the visual and the vestibular systems are activated first of all, followed by the proprioceptive and the motor systems (Fig. 1.7).

Fig. 1.7
Righting reaction.

Fig. 1.8
Extensor postural thrust reaction.

Fig. 1.9
Wheelbarrow.

Fig. 1.10
Wheelbarrow with head held high.

There are two methods of testing the righting reaction:

1. The animal is held up by the pelvis and the placement of its head with respect to the body is observed, while the forelegs are well flexed. A body-head angle of 45° is expected in normal cases. If the vestibular system is to be tested, the patient should be blindfolded, although this is rarely necessary.
2. The animal is placed in lateral recumbency and then observed as it gets up. Healthy animals that do not have a one-sided lesion of the vestibular system should first roll onto their sternum and then get up. This is incoordinated or even impossible in an animal with a disease of its vestibular system.

1.4.4.4 Extensor postural thrust reaction

In this two-stage test, the animal is held behind the shoulder blades and lifted so that it no longer touches the floor and then it is carefully placed back on the floor again. **Hyperextension** of the limbs should be observed. In the second part of the test, the animal is pushed gently backwards, so that an **alternating flexion and extension of the hindlimbs** is elicited. Lesions in the spinocortical, corticospinal and the vestibulocerebellar pathways cause disturbances in the movement processes and such clinical signs should be interpreted as pathological (Fig. 1.8).

1.4.4.5 Wheelbarrow test or walking on the forelimbs

Lifting of the patient, whereby the abdomen is supported so that the animal's hindlegs no longer touch the floor, enables the animal's ability to walk on its forelimbs to be judged. Normally, the head is carried parallel to the floor and the animal moves its legs forwards in symmetrical, short and alternating steps (Fig. 1.9). Pathological findings are a hesitant gait, knuckling or buckling of the limbs and a slowing down of the beginning or intention phase of the movement. Severe lesions in the cervical spinal cord cause a severe flexion of the head such that the nose touches the floor. To ensure that the patient can only correct its position using proprioceptive information alone, the wheelbarrow test is done with the ani-

Fig. 1.11
Visual placement test.

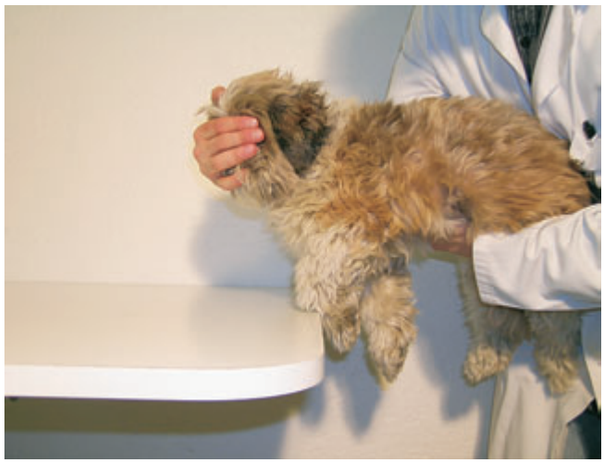


Fig. 1.12
Tactile placement test.

mal's head in extension (head held high) (Fig. 1.10). A slight increase in tone of the foreleg extensors is to be seen. This technique is particularly useful in animals with cerebral lesions as they usually exhibit a normal gait.

1.4.4.6 Placement test

This test has both a visual and a tactile component, which are undertaken one after the other. The visual placement test is very good for testing visual-assisted integration of movement. The animal is held up by its thorax and slowly pushed towards a barrier (table edge or top of a wall). The normal reaction is a coordinated stepping on the barrier without there having been any contact (Fig.1.11).

The tactile placement test is done in a similar manner though the animal is either blindfolded or has its head held in hyperextension. The thoracic limbs are then brought into contact with the edge of the table at the level of the carpus. The animal should immediately pick up its feet and lay them on the table. This test can be repeated with each limb individually, but as an adaptation (quick learning of the reaction) can take place, it is suggested that the animal should be turned around once between each test (Fig.1.12).

Knuckling, awkward or a lack of foot placement, excessive placement movements, excessively slow or a hesitant correction of movements are all to be interpreted as abnormal reactions.

1.4.4.7 Tonic neck reactions

These tests are infrequently done because they are difficult to interpret. The complex reactions to these movements are initiated by the receptors in the cervical muscles and by a fine coordination of the vestibular apparatus with the neck musculature and the receptors in the neck.

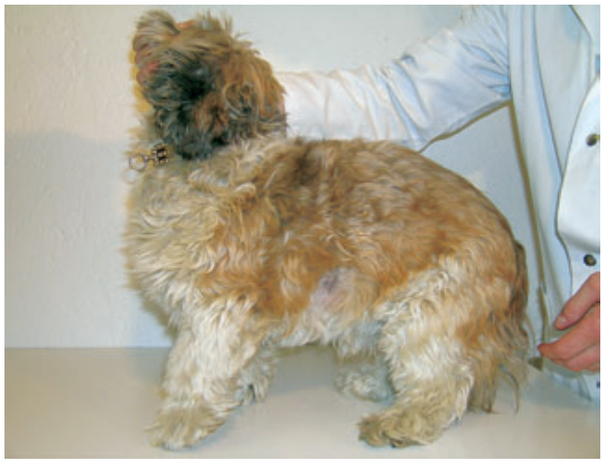


Fig. 1.13
Tonic neck reaction (hyperextension of the forelegs and hyperflexion of the hindlegs).

This test assesses the normal reaction of a flexion of the pelvic limbs and an extension of the thoracic limbs associated with raising the animal's head. Abnormal reactions are asymmetrical movements, stretching or abnormal bending of one or more limbs (Fig. 1.13).

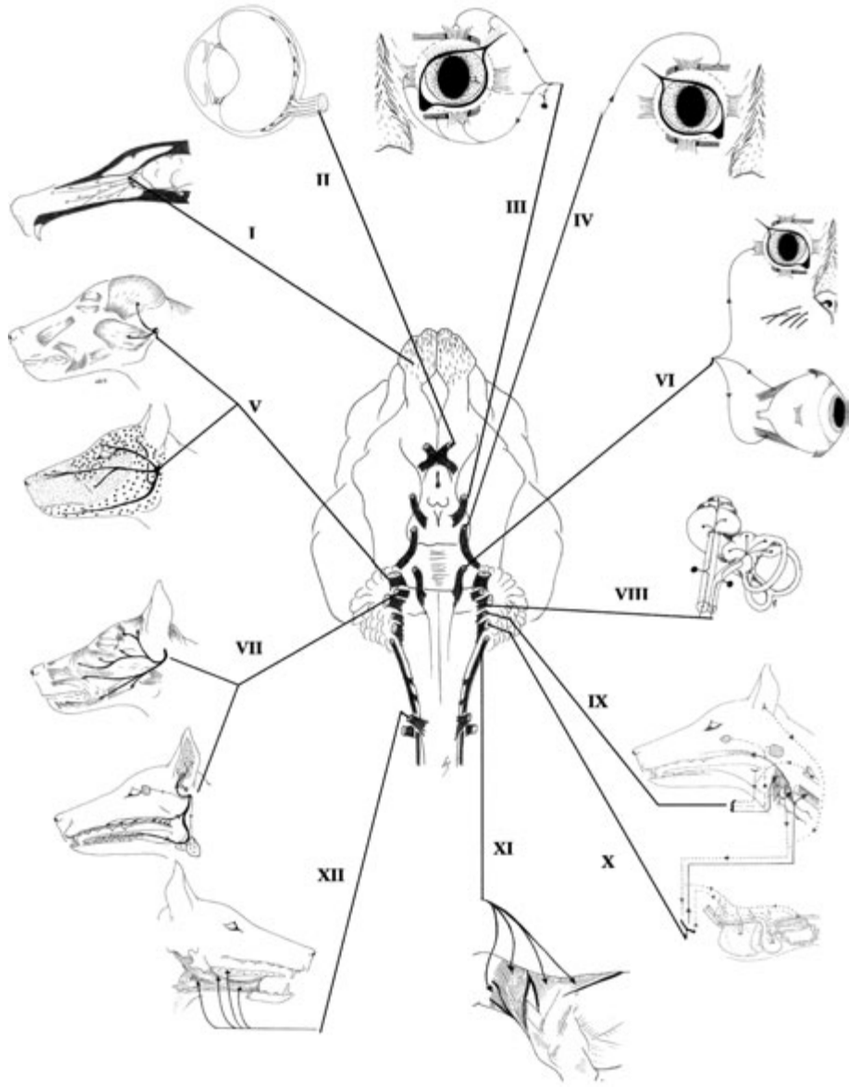


Fig. 1.14
Schematic overview of the twelve pairs of cranial nerves (I–XII). The majority of the cranial nerves originate in the midbrain, pons and medulla oblongata. The two exceptions are the olfactory nerve and the optic nerve.

If the head is bent downwards, a normal semiflexion of the thoracic limbs and an extension of the pelvic limbs occur. If the head is rotated to one side, then an extension of the limbs on the same side can be seen.

Cervical spinal lesions cause an abnormal flexion of all limbs during these tests in most cases.

1.4.5 Investigation of the cranial nerves

1.4.5.1 Cranial nerve function

The cranial nerves (CN) are conventionally numbered from I to XII. They originate either in the brain stem (III–X, XII) or outside it (I, II, XI). Simple examination methods can be used to investigate the functions of all the cranial nerves, which are comprised of motor and/or sensory fibres (Fig. 1.14). Parasympathetic fibres run within CN III (Fig. 1.15), VII, IX and X, while the sympathetic fibres lie separately (Fig. 1.16).

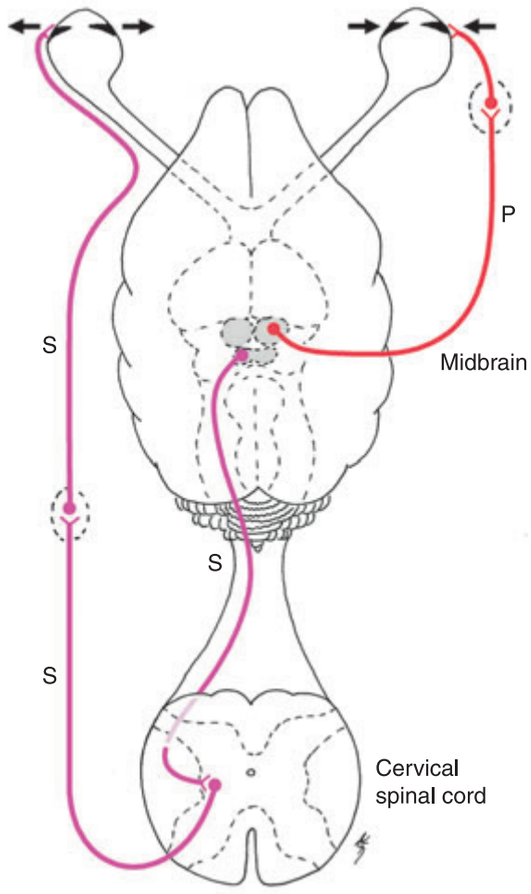


Fig. 1.15
Parasympathetic (P) and sympathetic innervation (S) of the pupils. (Pupil dilatation ← →; pupil constriction → ←).

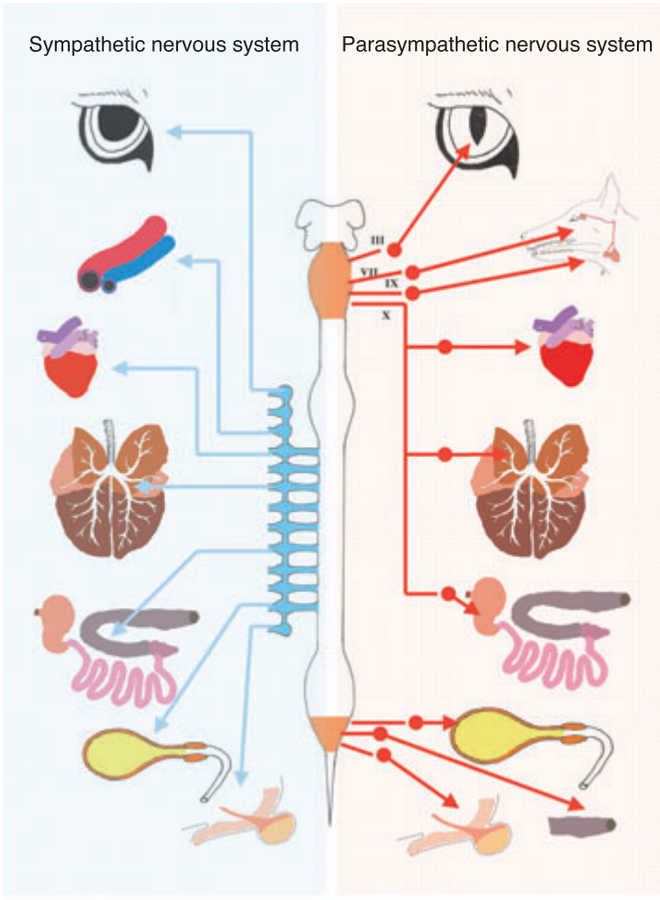


Fig. 1.16
Parasympathetic (red) and sympathetic (blue) innervation of the organs.

I. Olfactory nerve (sense of smell)

Anatomy and physiology

The chemical stimuli are transferred via the olfactory bulb to the olfactory part of the cerebral cortex for the conscious perception of smell.

Examination and assessment

The anamnesis (the observations of the owner or care-giver) with respect to the animal's behaviour in the presence of food or scents is most informative.

The objective assessment of the sense of smell is very difficult. In the dog, repulsive or pleasant test substances can be used. It should be ensured that substances are not used that will irritate the mucous membranes (ammonia or tobacco smoke), otherwise the receptors of the trigeminal nerve (CN V) will be stimulated causing a behavioural "defence" reflex to occur. Food, alcohol fumes or cloves are well suited to test whether a total loss in the sense of smell has occurred (**anosmia**). An incomplete loss, in contrast, cannot be diagnosed with any certainty.

Tumours or infectious disease are the most common causes of anosmia. However, this clinical sign is considered to be very rarely associated with a neurological lesion and so is not frequently tested.

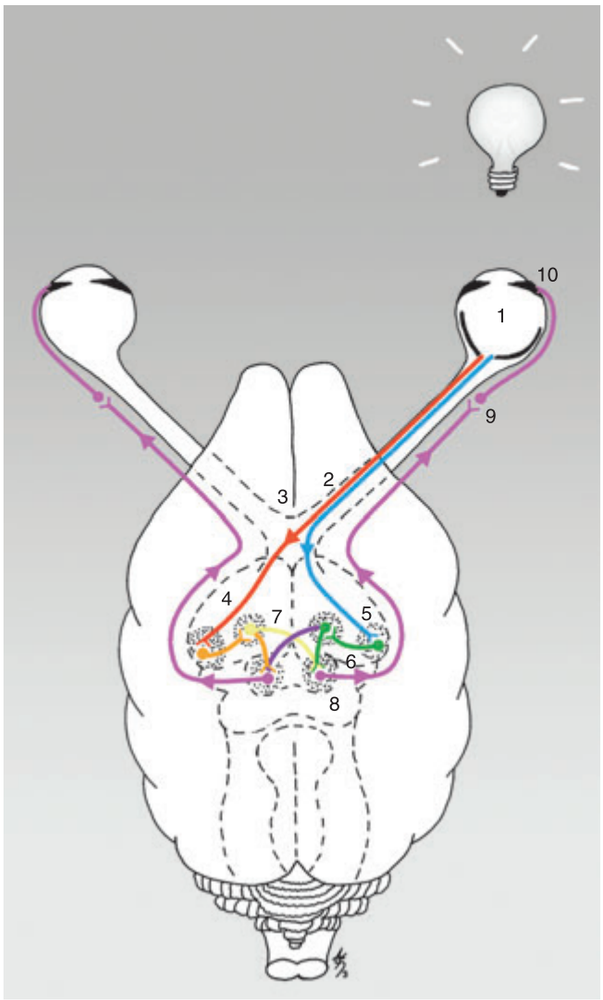


Fig. 1.17
Pupillary light reflex. The electrical impulses are transmitted in part by anatomical structures similar to those involved in the menace response. These are the temporal and nasal retina (1), the optic nerve (2), the optic chiasma (3) and the optic tract (4).The latter tract has synapses with the pretectal nuclei and further on with the parasympathetic nuclei (Edinger-Westphal nuclei, 5) of the oculomotor nerve. The majority of the optic fibres responsible for the pupillary light reflex come from the temporal side of the retina, do not cross over and run via pretectal fibres to the lateral (direct reflex arc, 6) and contralateral (indirect reflex arc, 7) Edinger-Westphal nuclei. This is the reason why the reactions of the direct pupillary light reflex are stronger than those of the indirect reflex. The preganglionic parasympathetic neurons of CN III (8) lie in the rostral part of the midbrain ventral to the mesencephalic aqueduct. These fibres go ventral to the crus cerebri, pass through the dura mater and join up with CN IV and VI. Afterwards, they then pass through the orbital fissure. The parasympathetic part of CN III has its synapses in the ciliary ganglion (9). Short postganglionic fibres go to the ciliary and the sphincter pupillae muscles (10). The sympathetic fibres innervate the dilator pupillary muscles. The reflex arc originates in the hypothalamus and has its first synapses in the midbrain. From there, it continues on to the preganglionic neurons in the spinal cord between T1–T3 (Fig. 1.15). After leaving the spinal cord, these neurons run within the vagosympathic trunk to the cervical ganglia. The postganglionic fibres travel with the internal carotid artery, then pass through the middle ear, to finally innervate the smooth muscle, glands and blood vessels in the head and neck.

II. Optic nerve (sense of sight)

Anatomy and physiology

Light stimulates the photoreceptors of the retina and subsequently, the neurons of the retina and then highly specialised ganglion cells. The axons of the latter cells form the optic nerve. The majority of fibres of CN II cross over at the optic chiasm and then run as the optic tracts to the lateral geniculate body of the thalamus. The neurons from this body project via the optic radiation to the optic cortex, where the stimulus is perceived at a conscious level.

In animals, the majority of the optic fibres cross over at the level of the optic chiasm to the contralateral side, whereas in primates only those fibres which have their origin in the nasal half of the retina cross over. In dogs, the majority of the nasal fibres and about 50% of the temporal fibres cross over to the contralateral side. In animals within the same genus, about 75% of the fibres cross over, while in cats it is roughly 65%. From a clinical point of view, the majority of animals behave as if all the fibres crossover. Therefore, the left and right fields of vision are examined separately, and a patient with a one-sided lesion of the cerebral cortex will appear clinically to be blind only on the contralateral side.

The reflex arc for the pupillary light reflex partially contains the same anatomical-physiological structures as the pathways for sight (Fig. 1.17).

Examination and assessment

The sense of sight is responsible for vision and the pupillary light reflex. A number of neurological tests are used to examine these processes: the menace response, the pupillary light reflex, the cotton ball test, the visual placement test and an ophthalmological examination (see Chap. 1.5). Observation of the patient while navigating around obstacles is a rather unreliable test as other neurological abnormalities can affect the results.

Menace response

Both eyes are investigated separately. A sudden movement with the hand is made towards one eye. The normal response is a closing of the eyelids; this is not a reflex (Fig.1.18). This response is not present in animals under 10 to 12 weeks of age and **has to be learnt**. The hand movement should not be too harsh or the tactile receptors of the cornea will be stimulated, thereby initiating the corneal reflex.

Diffuse cerebellar lesions maybe associated with a loss of the menace response. Such patients are not blind and they have a normal palpebral reflex.

Pupillary light reflex

The pupillary light reflex tests, amongst other things, the function of the peripheral visual pathways. First of all, the size and symmetry of the pupils are examined. Physiologically, the pupils are the same size in both eyes. If the pupils are of different sizes, it is called anisocoria (mydriasis = enlarged pupil; miosis = small pupil).

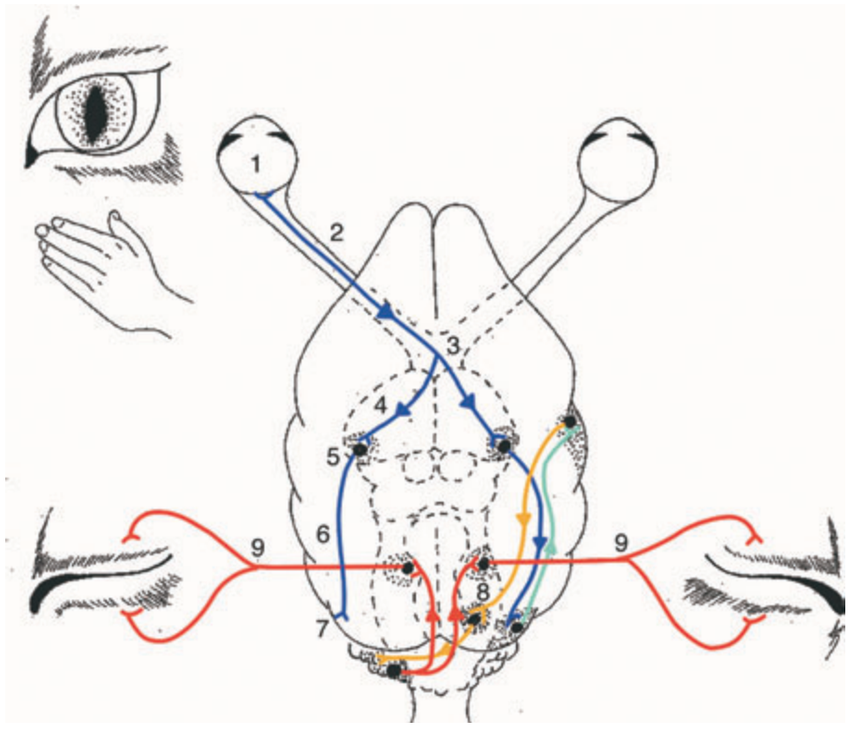


Fig. 1.18
menace response. This "reflex" arc consists of the receptors in the retina (1), the optic nerve (2), the optic chiasma (3), the optic tract (4), the lateral geniculate body (5), the optic radiation (6), the visual cortex (7), the crossed descending pathways to the brain stem (8) and the facial nerve (9).

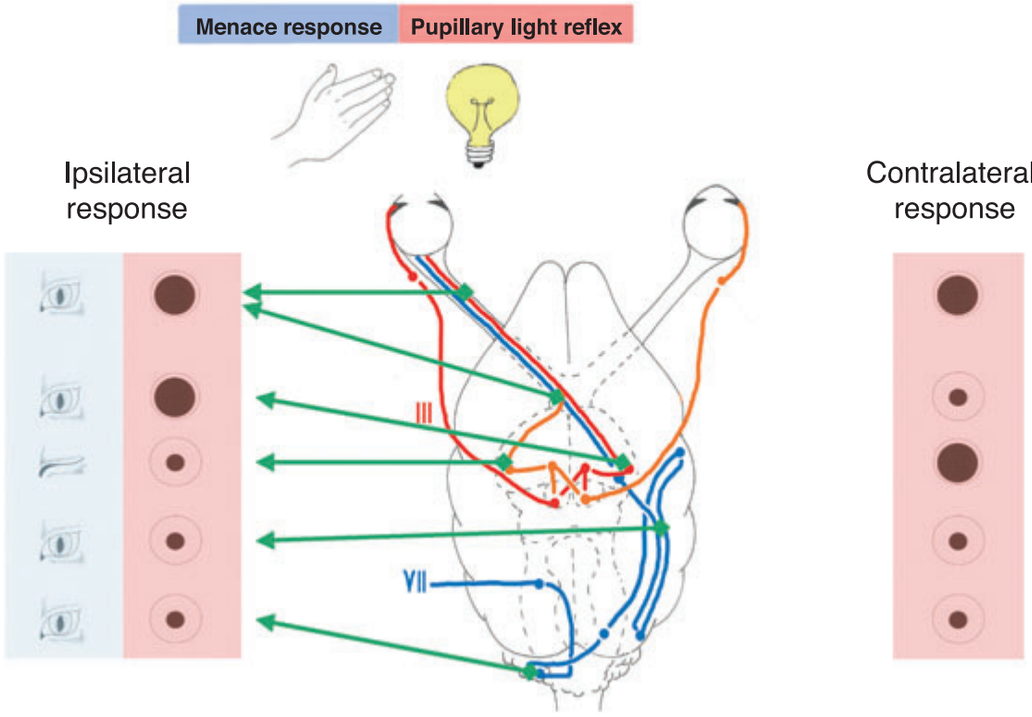


Fig. 1.19
Ipsilateral and contralateral reactions of the pupil to a light stimulus affecting the left eye. Ipsilateral response to a menace. The different lesions that are possible along the visual pathway are shown in green.

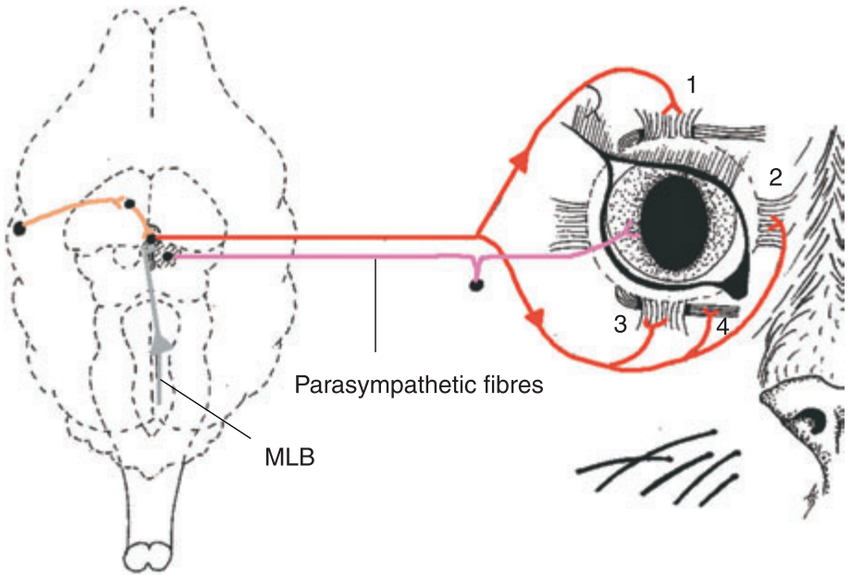


Fig. 1.20
Innervation of the eyeball via the motor nuclei of the oculomotor nerve (red) to the dorsal (1), medial (2) and ventral (3) rectus muscles as well as the ventral oblique muscle (4) and levator palpebrae muscle. The medial longitudinal fasciculus (MLB) connects these three cranial nerve nuclei with the vestibular system and the motor centres of the eye muscles. Movement of the head to the side activates the vestibular system (orange) and subsequently, the nerves to the eye muscles causing a physiological nystagmus.

The examination is undertaken by shining a strong source of light into the eye. The normal reaction is a constriction of the pupil both on the side of the light stimulation (direct reflex) and on the other side (indirect reflex).

An incomplete lesion of the optic nerve is usually only associated with a partial dysfunction. A lesion of the retina, optic nerve or optic tract causes a peripheral blindness with a greatly delayed pupillary light reflex (Fig. 1.19).

A lesion affecting only the parasympathetic nucleus of CN III on one side is rather rare as this nucleus lies only a few millimetres from the rest of CN III. Clinically, an unresponsive enlarged pupil is seen.

If there is a lesion in CN III, not only is an enlarged pupil evident but there is also a ventrolateral strabismus.

Cotton ball test
A ball of cotton wool is allowed to fall within one side of the visual field. The normal reaction is a movement of the head or eye in the direction of the ball. The left and right visual fields can be tested separately. A reaction to this test necessitates an intact optic nerve and also an intact optic cortex. A lesion in the optic region of the cerebral cortex is known as a "central" blindness.

Visual placement test
With this test, subtle abnormalities in sight can be differentiated. The neurological pathway consists of the optic nerve as well as the efferent motor pathways to the limbs.

Ophthalmological examination
If there are no obvious changes in the cornea, lens, aqueous humour or vitreous body, then the ocular fundus is also examined (see Chap. 1.5). Gross abnormalities such as inadequate filling of the blood vessels, bleeding, a loss in colour of the retina or a swelling of the optic disc can be recognised.

Using the abovementioned tests, a lesion in the visual pathways can be more accurately localised.

III.	**Oculomotor nerve**	**(eye position and movement)**
IV.	**Trochlear nerve**	
VI.	**Abducens nerve**	

The oculomotor nerve is primarily responsible for the normal movement of the eyeballs. The trochlear nerve is responsible for the movement of the eyeball upwards and the abducens nerve for its movements laterally.

Anatomy and physiology

The motor nuclei of the oculomotor nerve lie caudal to the parasympathetic nuclei of CN III, within the midbrain or mesencephalon, and innervate several specific eyeball muscles (Fig. 1.20).

The nuclei of the trochlear nerve are localised in the grey substance of the mesencephalon (Fig. 1.21).

The nuclei of the abducens nerve originate on the ventral floor of the fourth ventricle in the medulla and project their axons ventrolaterally to the pyramids (Fig. 1.22).

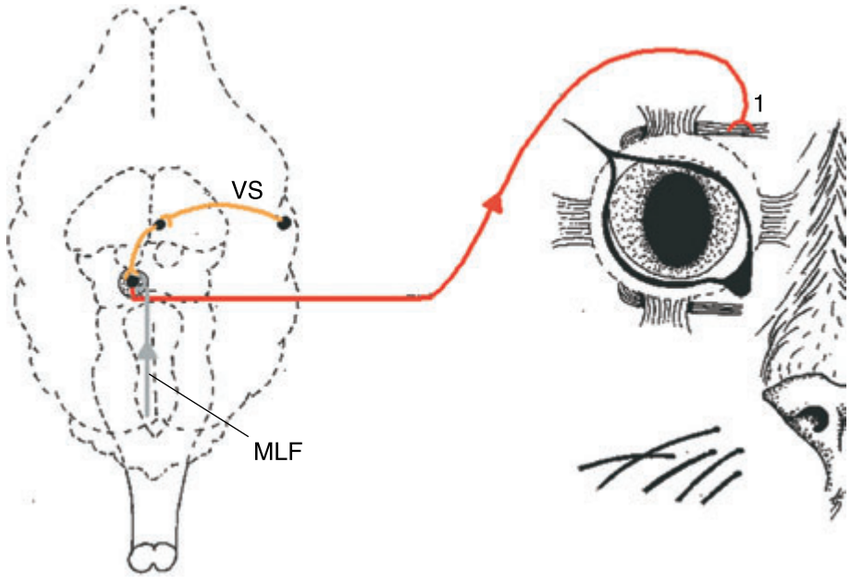


Fig. 1.21
Innervation of the dorsal oblique muscle (1) via the trochlear nerve (red), the medial longitudinal fasciculus (MLF) and vestibular system (VS).

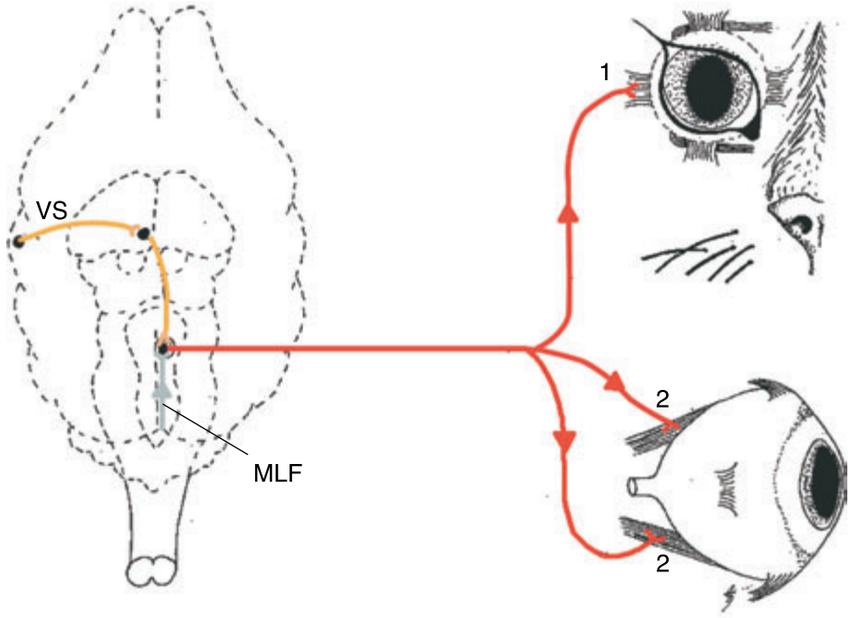


Fig. 1.22
Abducens nerve (red). Common innervation of the lateral rectus muscle (1) and the retractor bulbi muscle (2) via the abducens nerve and CN III and IV, the medial longitudinal fasciculus (MLF) and vestibular system (VS).

Examination and assessment

The eye movements are examined with the patient's head initially at rest and then during movement. The position of the eyes is observed and their ability to correct their position in a particular axis is tested. The passive movement of the head to the side, upwards or downwards simulates the active tracking of an object and so leads to a **physiological nystagmus**. This is composed of two components, a slow and a fast phase. The latter occurs in the direction of the head movement.

Strabismus is the name given to abnormal eye position. Knowledge of the anatomy and the innervation makes it possible to interpret the causative abnormalities (Fig. 1.23). As the three above-mentioned nerves are responsible for the innervation of the seven main muscles which dictate the position of the eye, lesions of one or more of these CN leads to a paralysis of one or more muscles resulting in an ipsilateral strabismus. A lesion of CN III causes a ventrolateral strabismus +/– ptosis (paralysis of the levator palpebrae muscle) and mydriasis (paralysis of the parasympathetic nerve).

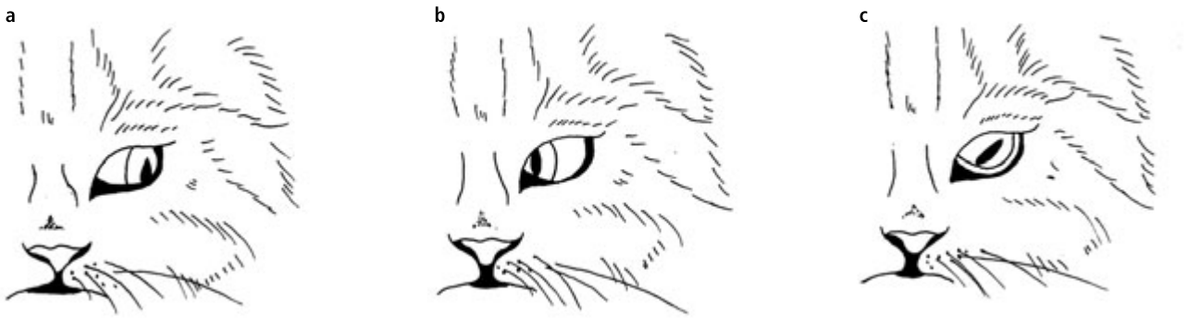


Fig. 1.23a–c
Abnormal positioning of the eyeball can occur due to lesions of the oculomotor nerve (ventrolateral, (a), the abducens nerve (medial, (b) or the trochlear nerve (rotation, c).

A rotational strabismus occurs with a lesion of CN IV. In this case, the dorsal part of the eyeball is pushed laterally. As cats have a slit-like pupil, this phenomenon is easier to detect than in the dog, where this condition can usually only be definitely diagnosed by using an ophthalmoscope and inspecting the retinal vascualture. The superior retinal vein is also shifted towards the temple. A lesion of CN VI leads to a medial strabismus with a loss in the animal's ability to retract the eyeball. Siamese cats can be affected by a congenital medial strabismus, which is not due to a paralysis of CN VI, but is related to an abnormal decussation of the optic tracts.

Incoordination of the eye movements, or more commonly a reduced to absent normal nystagmus, occur with a lesion in the vestibular system, cervical cord prorioception tracts or the medial longitudinal fasiculus in the brain stem,

V. Trigeminal nerve

Anatomy and physiology

The trigeminal is a mixed cranial nerve, composed of three branches (ophthalmic, mandibular, maxillary). It contains both sensory and motor fibres (Fig. 1.24). The sensory fibres are important as the afferent portion of the corneal and palpebral reflexes.

Examination and assessment

All three sensory branches should be examined.

1. The ophthalmic branch is stimulated by tapping on the medial canthus of the eye. The normal reaction is closure of the eyelids. It also innervates the nasal mucosa.
2. The maxillary branch innervates the lateral canthal area, the muzzle and the nasal mucosa. The animal should show a behavioural defence reaction such as pulling its head away or trying to bite in response to a pinching of the lips, tapping the lateral canthus of the eye or stimulating the nasal mucosa. Tapping the lateral canthus of the eye also results in a reflex closure of the palpebral fissure.
3. The mandibular branch innervates the skin of the lower jaw. Pinching this area induces a behavioural defence reaction. Obtunded animals may demonstrate reduced reactions when they are stimulated over the head region.

In addition to the examination of the **sensory innervation of the head region,** an examination of the motor part of the trigeminal nerve should be performed. Passive opening of the mouth assesses jaw tone and palpation of the masseter and temporalis muscles assesses their degree of tone or atrophy. Testing the sensitivity to pain by palpating the skull in the region between the orbit and the zygomatic arch may cause a behavioural response in some patients with "headache" due to elevated intracranial pressure.

A lesion of the motor nucleus of CN V causes only masticatory muscle paresis or paralysis without any loss in sensation. In a one-sided lesion, muscle atrophy can be seen after 7 to 10 days. Asymmetry of the palpable masticatory muscles and possible dysphagia can be observed.

A lesion of the sensory nuclei results in a decrease or loss of pain perception with normal motor functions. With a focal lesion affecting a unilateral sensory cerebral cortex, the contralateral facial skin may have reduced pain perception. The animal can still feel stimulation of the nasal mucosa, but has a reduced reaction to facial skin stimulation.

VII. Facial nerve (facial expression)

Anatomy and physiology

The reflex arc of the **palpebral reflex** is made up of an afferent part, (trigeminal nerve to brain stem), and an efferent part, the facial nerve (Fig. 1.25).

The neuroanatomical pathway of the **menace response** is made up of receptors in the retina, the optic nerve, optic chiasma, optic tract, lateral geniculate body, optic radiation,

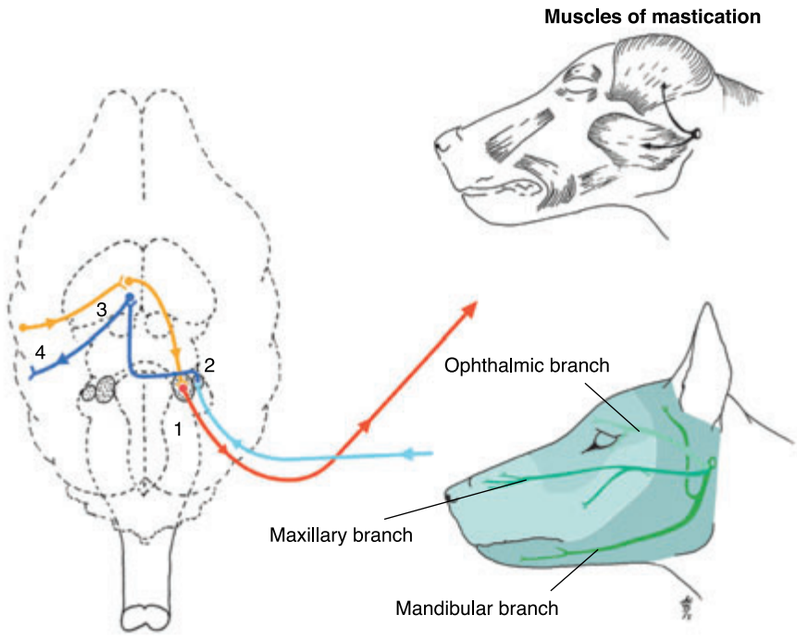


Fig. 1.24
Trigeminal nerve. The motor nucleus (1) lies within the pons at the level of the rostral cerebellar peduncle. The sensory part of CN V consists of the **pontine nuclei** (2), which receive information from mechanoreceptors and the **spinal nuclei**. The latter transmit information mainly from nocioceptors. Projections from these nuclei run rostrally with the contralateral fibres of the **lemniscus** via the thalamus (3) to the cerebrum (4). Axons from both the pontine and spinal nuclei project to the facial nuclei.

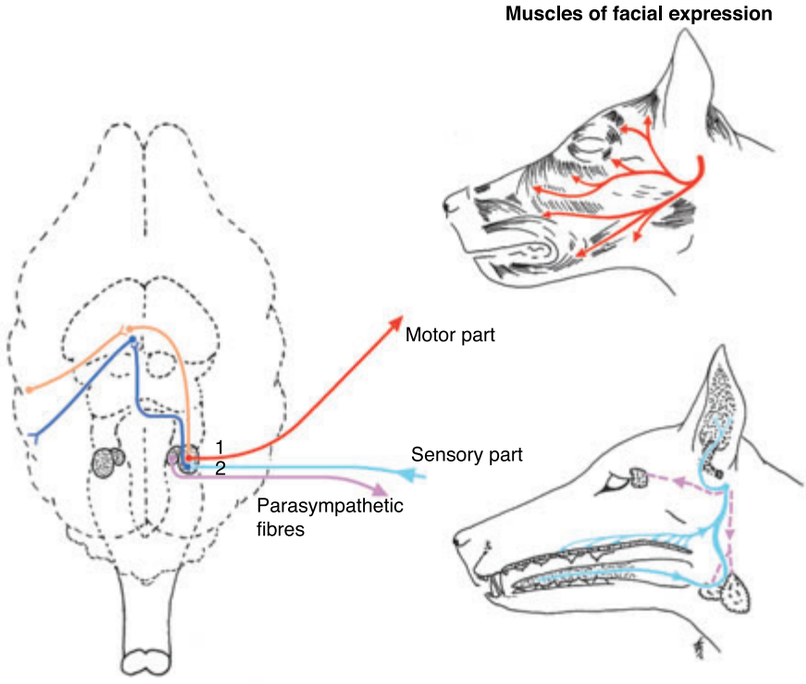


Fig. 1.25
Facial nerve. The nucleus (1) of the facial nerve lies in the rostral part of the medulla. The facial nerve enters the internal acoustic meatus with CN VIII and crosses over the facial canal. Its motor branches innervate the facial muscles and the caudal part of the diagastricus muscle. The cell bodies of its sensory neurons are found in the geniculate ganglion (2) in the petrous temporal bone. These innervate the caudal two-thirds of the tongue and the palate with the trigeminal nerve. The facial nerve innervates the stapedius muscle (inner ear) as the stapedius nerve. Its parasympathetic part innervates the nasal, mandibular, sublingual and lacrimal glands.

the visual cortex, descending crossing tracts to the brain stem, the cerebellum and the facial nerve.

Taste is conveyed via special taste receptors in the rostral two thirds of the tongue through the facial nerve. The receptors in the caudal third of the tongue are innervated by the glossopharyngeal nerve. The parasympathetic portion of the facial nerve is responsible for lacrimal gland stimulation.

Examination and assessment

A complete lesion of the facial nerve manifests itself clinically in many ways. Facial paralysis is characterised by a drooping of the ear and lip, a widening of the palpebral fissure, asymmetry of the nostrils and a loss in the sense of taste. As a secondary consequence, there can be damage to the cornea due to desiccation from a lack of tear production and the loss of the protective blink reflex. Inadequate closing of the lips can cause salivation or drooling on the affected side. A partial lesion of the facial nerve can be associated with one or more clinical signs.

The palpebral reflex is stimulated by touching the skin around the eyes and results in a closure of the eyelids. The corneal reflex is instigated by a gentle touching of the cornea. A closing of the eyes is again to be interpreted as a normal reaction to both of these tests, whereas lesions of the facial nerve result in the eyes not being closed in either test.

Additionally, the tear production test (Schirmer tear test) can help with the localisation of a lesion in this reflex arc. Intracranial-extramedullary lesions usually disturb all of the nerve's functions. A lesion in the petrous portion of the temporal bone, depending on its degree, affects one or more functions of the facial nerve. Additionally with such a lesion localisation, vestibular symptoms can be seen such as head tilt, nystagmus and positional strabismus.

The sense of taste can be tested with atropine. Healthy animals react quickly to atropine's bitter taste with immediate licking movements and salivation.

VIII. Vestibulocochlear nerve (balance and hearing)

Vestibular Nerve

Anatomy and physiology

The vestibular system is responsible for the static and dynamic position of the head. The CNS is informed about a change in the head's position by both labyrinths. The labyrinth of the inner ear contains the ampulla with its three semi-circular canals lying at right angles to each other, and the macula, another receptor system, that is found in the utricule and saccule. The organs of balance are stimulated by changes in position, acceleration or gravity. The resulting depolarisation is transferred via the vestibular neurons within CN VIII.

Examination and assessment

An examination of a patient in the acute phase of a vestibular syndrome is often difficult as the patient exhibits disorientation and is sometimes, obtunded. Head tilt, ataxia and nystagmus are the important cardinal signs of vestibular disease. It is important both for the therapy and the prognosis to determine between a central or peripheral cause of the vestibular dysfunction.

The postural and placement reactions and the direction of the nystagmus can help to localise the disease.

There are different types of nystagmus. Spontaneous nystagmus occurs without any movement of the head and is always pathological, comprised of rapid and slow phases, and defined according to the direction of its rapid phase. The three directions of nystagmus are vertical, rotary and horizontal, with the slow phase towards the diseased side. Pendular nystagmus does not have alternating rapid and slow phases, and can be elicited by a lesion in the cerebellum. Lesions of the peripheral vestibular apparatus mainly cause a horizontal or rotary nystagmus, where the direction of the nystagmus remains constant.

Normal nystagmus can be induced temporarily when the position of the animal's head is suddenly passively changed. Pathological nystagmus does not require head movement but may be elicited by certain head positions. In healthy animals the eye remains in the middle of the palpebral fissure when the head moves dorsally. In comparison, a vestibular (positional) strabismus occurs with vestibular lesions, whereby the eye becomes positioned downwards towards the damaged side when the head is moved dorsally.

As infections of the inner ear are frequently associated with a middle ear lesion, an otoscopic examination should always be undertaken in cases with vestibular symptoms.

Cochlear Nerve

Anatomy and physiology

Hearing is a complex process (Fig. 1.26).

Examination and assessment

A sudden acoustic stimulus (e.g. whistling, clapping) outside of its visual field causes a reaction in the healthy animal, whereby it turns its head in the direction of the sound. The localisation of a lesion within the auditory pathway is not possible with simple tests; only special and painstaking electrodiagnostic methods (auditory evoked potentials) are capable of achieving this.

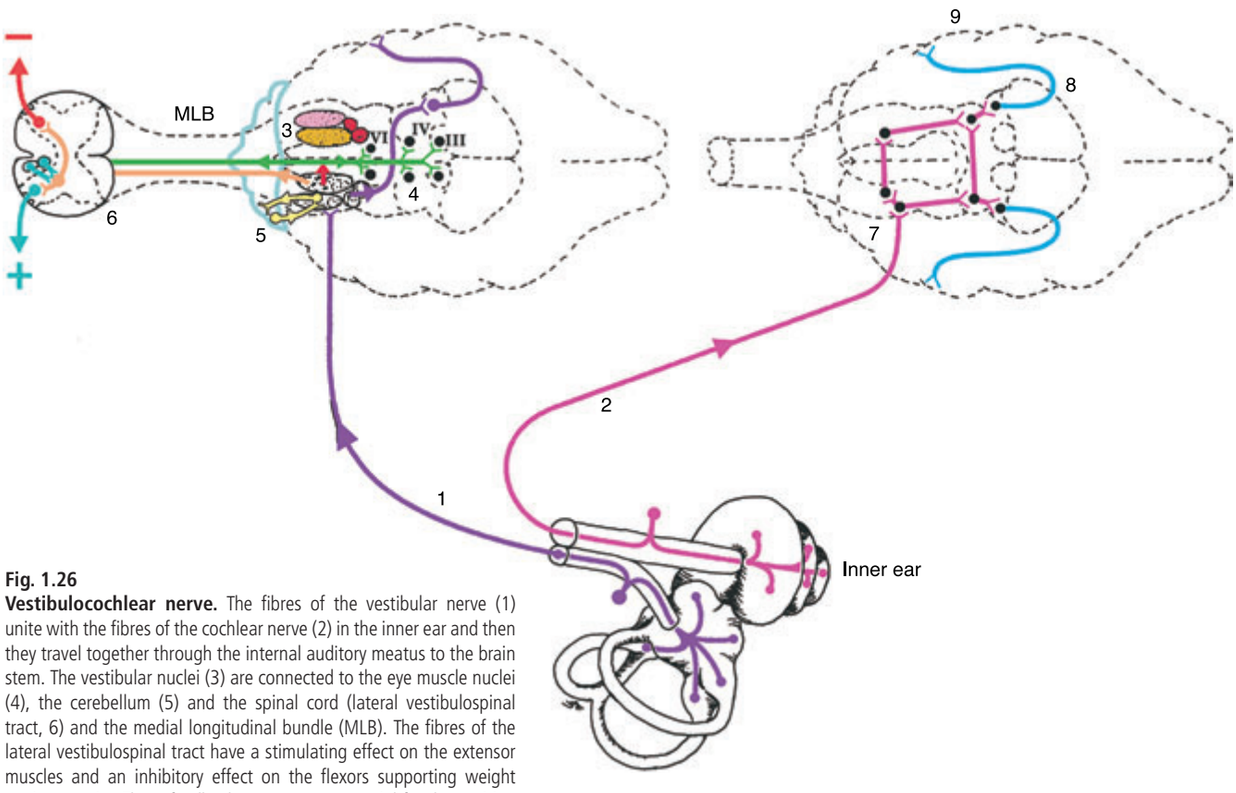


Fig. 1.26
Vestibulocochlear nerve. The fibres of the vestibular nerve (1) unite with the fibres of the cochlear nerve (2) in the inner ear and then they travel together through the internal auditory meatus to the brain stem. The vestibular nuclei (3) are connected to the eye muscle nuclei (4), the cerebellum (5) and the spinal cord (lateral vestibulospinal tract, 6) and the medial longitudinal bundle (MLB). The fibres of the lateral vestibulospinal tract have a stimulating effect on the extensor muscles and an inhibitory effect on the flexors supporting weight against gravity. These feedback systems are essential for the position and placement reflexes as well as for the movements of the eyes.
Hearing is a complex process. Acoustic stimuli reach the auditory cortex (9) via the external ear (outer ear canal and eardrum), the middle ear (malleus, incus and stapes), inner ear with its specialised receptor organs in the petrous temporal bone, the acoustic part of CN VIII (2), the cochlear nuclei (7), the superior olive, the midbrain and the thalamus (8).

IX. Glossopharyngeal nerve
X. Vagus nerve
} (swallowing)

Anatomy and physiology

The glossopharyngeal nerve is responsible for the sensory innervation of the caudal tongue, pharynx and larynx, and also for the motor innervation of the pharynx, the soft palate and the oesophagus. It conveys parasympathetic fibres to the parotid and zygomatic salivary glands (Fig. 1.27).

The vagus nerve innervates the pharynx, larynx and palate with motor and sensory fibres; additionally, it supplies parasympathetic fibres to all of the thoracic and abdominal viscera except for the urinary bladder and the pelvic canal, which are innervated by the sacral parasympathetic nerves. The recurrent laryngeal nerve leaves the vagus and goes to the upper third of the oesophageal musculature and the larynx.

Examination and assessment

The swallowing reflex helps in the examination of both nerves. It can be elicited internally (by touching the mucosa of the pharynx with the finger; wear gloves!) or externally (by a light stimulation of the pharyngeo-laryngeal area. A swallowing motion is the normal reaction.

The clinical signs of a lesion in CN IX and/or X are dysphagia, including problems with swallowing, asymmetry of the pharynx or larynx, as well as regurgitation.

Whilst a complete paralysis causes an inability to swallow, a one-sided lesion leads to one-sided problems in swallowing and regurgitation of food; with the latter even occurring through the nose.

Bradycardia, an increase in bronchial secretion, a reduction in peristalsis and the production of gastrointestinal juices all occur with vagotonia (hyperexcitability of the vagus nerve).

Always consider rabies in any patient presenting with swallowing problems.

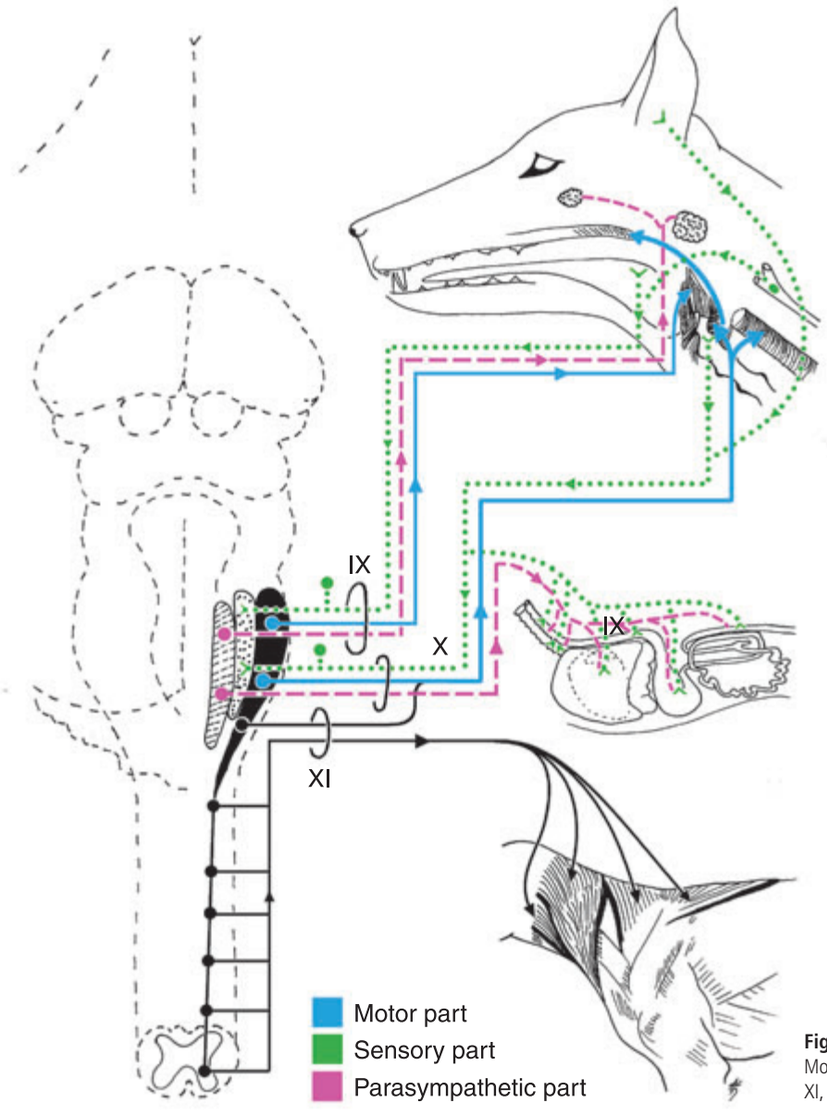


Fig. 1.27
Motor, sensory and parasympathetic parts of CN IX, X and XI, and their respective areas of innervation.

XI. Accessory nerve (neck muscles)

Anatomy and physiology

The motor nucleus of CN XI is found between the spinal segments C1 and C6. The nerve fibres run cranially to the trapezius muscle, parts of the brachiocephalicus muscle and the sternocephalicus muscle (Fig. 1.27).

Examination and assessment

Disturbances of the motor innervations of these muscles are difficult to diagnose. Neurogenic muscle atrophy can be ascertained by a careful palpation of the neck musculature. The function of CN XI can be ascertained from the posture of the head and neck, which may be deviated toward the affected side in chronic cases.

XII. Hypoglossal nerve (tongue movements)

Anatomy and physiology

The hypoglossal nerve is responsible for tongue function (Fig. 1.28).

Examination and assessment

Inspection of the symmetry of the tongue and its movements, observing intake of water and food as well as passive manipu-

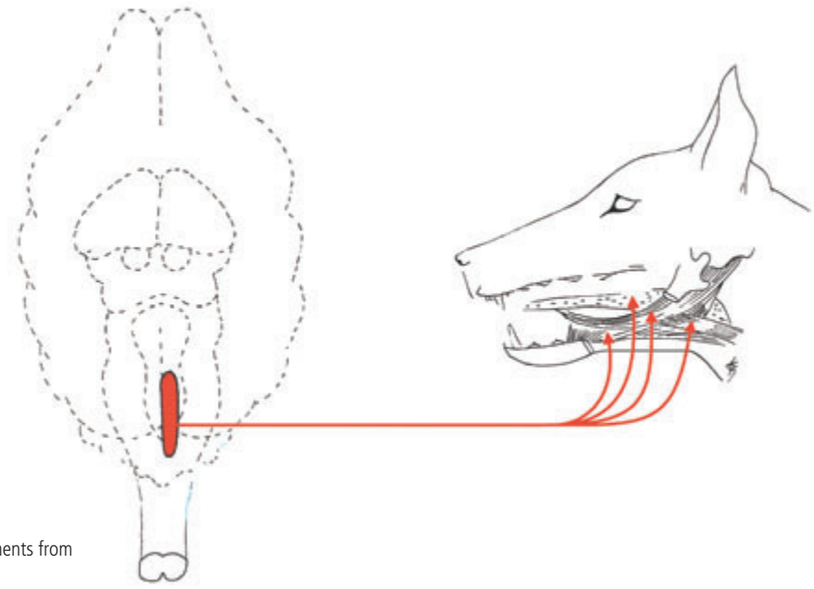


Fig. 1.28
Hypoglossal nerve. The innervation of tongue movements from the nucleus of CN XII in the caudal medulla.

lation of the tongue should be included in the examination. Difficulties in the intake of water and food as well as in chewing and swallowing are signs of a lesion of CN XII. If the tongue hangs out of the mouth, then it is usually drawn to the side of the lesion in chronic cases and away from the side of the lesion in acute cases.

Disturbances in the last four cranial nerves may be difficult to differentiate from one another.

1.4.6 Spinal reflexes

The spinal reflexes depend on intact motor and sensory nerve function, the effector muscles and the grey substance of the respective spinal segments. By testing the reflexes, it is possible to directly and simply examine the function of a specific segment of the grey substance in the spinal cord, and its associated nerve roots and nerves. There are two types of reflexes to be differentiated in the practical neurological examination: the **tendon reflexes** and the **withdrawal reflexes**.

In the tendon reflexes, the muscles, and so their neuromuscular spindles, are passively stretched by hitting them with a reflex hammer, whereby a signal is elicited that is transferred over the sensory nerves to the grey substance of the respective spinal segment (reflex centre) and then on via the motor nerves to instigate a reflex contraction of the muscle. More precisely, the dorsal horns of the spinal cord receive the sensory stimuli via the sensory nerve fibres, dorsal roots, and spinal ganglia. The stimuli are then transferred to the target muscle via the ventral horn cells, motor root and ventrally the spinal nerves. This reflex arc can be modulated by afferent and efferent pathways coming from the brain (UMN).

In the withdrawal reflex, stimulation of the skin leads to a contraction of the muscle. This flexor reflex is elicited by, for example, pressure on the foot pads or the skin between the toes.

The possible results of such reflex investigations are (i) normal reflex activity, (ii) areflexia (loss of reflex), (iii) hyporeflexia (reduction), or (iv) hyperreflexia (increase) and clonus (repetitive flexion and extension of the distal limb after a single stimulation). The spinal reflexes should be investigated with the animal in lateral recumbency. The animal is tested first on the uppermost side and then turned over and evaluated on the other side. It is important that the animal is **relaxed** during the reflex examination and its limbs are as loose as possible. A lesion in the LMN leads to a reduction or loss of a reflex. In contrast, a lesion in the UMN is associated with either a normal or increased reflex activity.

1.4.6.1 Pelvic limb reflexes

The reflexes of the pelvic limbs can be tested before those of the thoracic limbs and then the two sets of limbs are compared

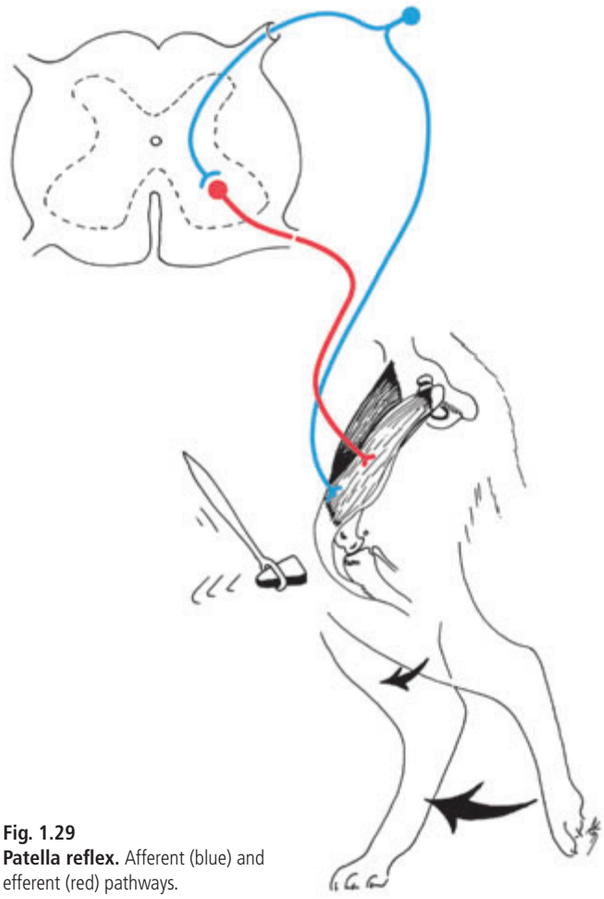


Fig. 1.29
Patella reflex. Afferent (blue) and efferent (red) pathways.

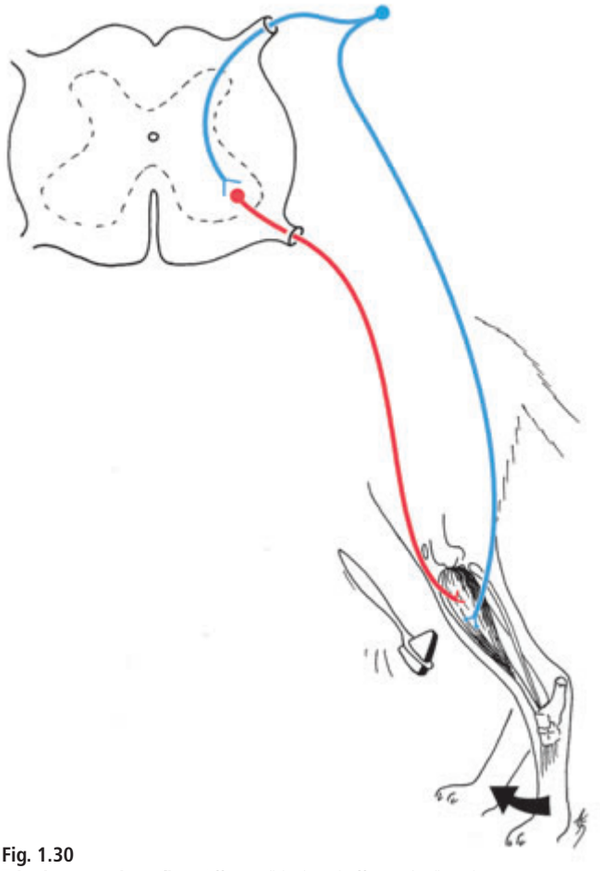


Fig. 1.30
Tibialis cranialis reflex. Afferent (blue) and efferent (red) pathways.

Patellar reflex (femoral nerve)

The reflex centre lies between L4–L6. The middle patella tendon is tapped whilst the animal is in lateral recumbency and the leg loosely supported. A reflex contraction of the quadriceps muscle and a forwards movement of the lower limb is to be expected (Fig. 1.29).

Cranial tibial reflex (peroneal nerve)

The reflex centre lies between L6–S1. Hitting the cranial tibial muscle (dorsolateral in the upper third of the lower leg) causes flexion of the tarsus (Fig. 1.30).

Flexor reflex (sciatic nerve)

The reflex centre lies between L4–S3. Pinching the toes, pads of the feet or the skin between the toes causes a sudden flexion of the whole limb; all joints must be seen to flex (Fig. 1.31).

1.4.6.2 Thoracic limb reflexes

Extensor carpi radialis reflex (radial nerve)

The reflex centre lies between C7–T1. The limb is supported under the elbow. A light tapping of the extensor carpi radialis muscle below the elbow causes a slight extension of the carpus (Fig. 1.32).

Triceps reflex (radial nerve)

The reflex centre lies between C6–T1. The thoracic limb is drawn slightly forwards and the shoulder is gently pushed outwards with the elbow flexed. A tap on the triceps tendon just above the olecranon causes an extension of the elbow and of the carpus (Fig. 1.33).

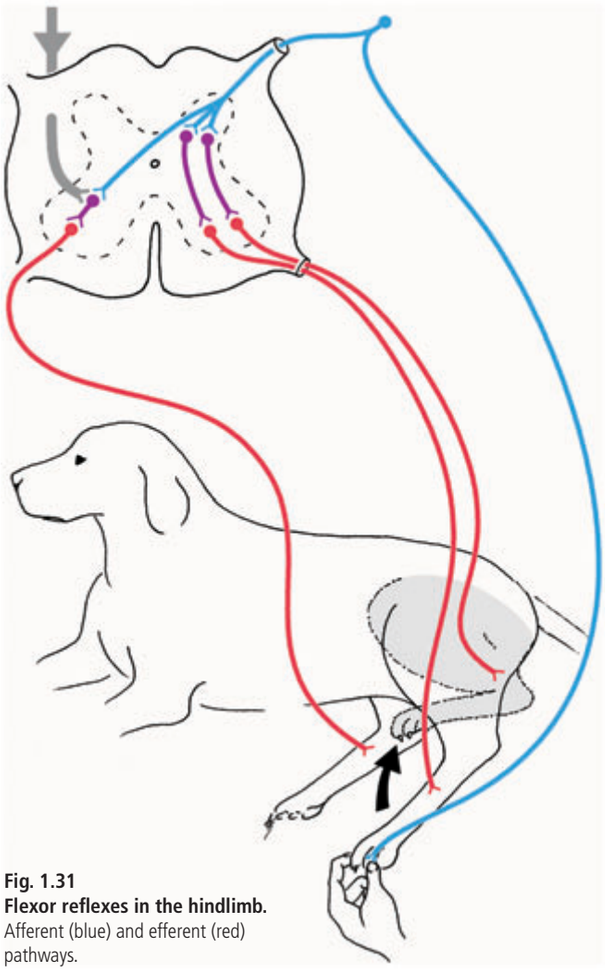


Fig. 1.31
Flexor reflexes in the hindlimb. Afferent (blue) and efferent (red) pathways.

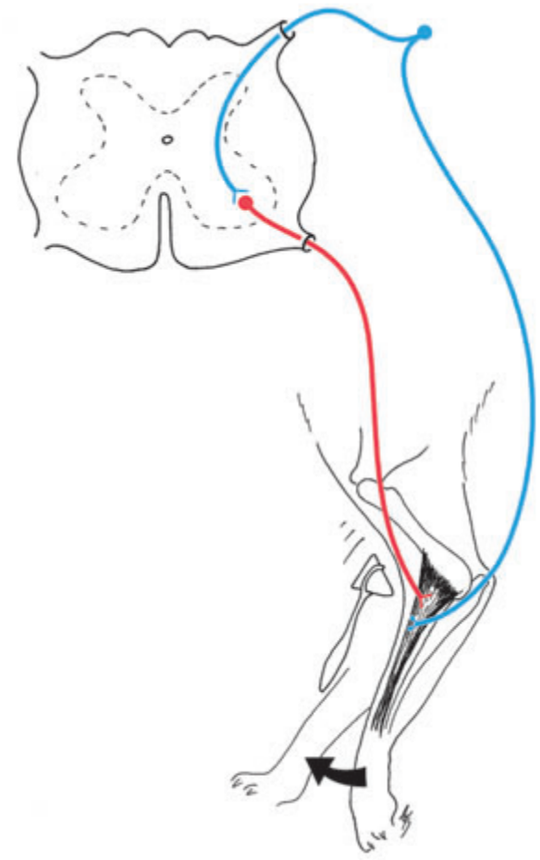


Fig. 1.32
Extensor carpi radialis reflex. Afferent (blue) and efferent (red) pathways.

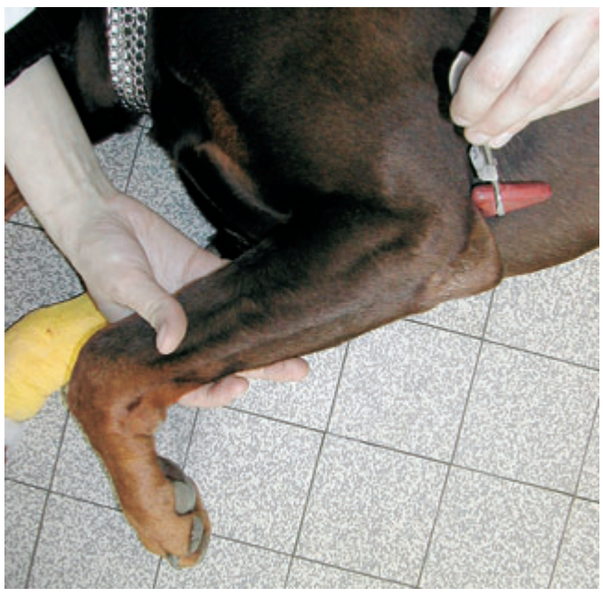


Fig. 1.33
Triceps reflex.

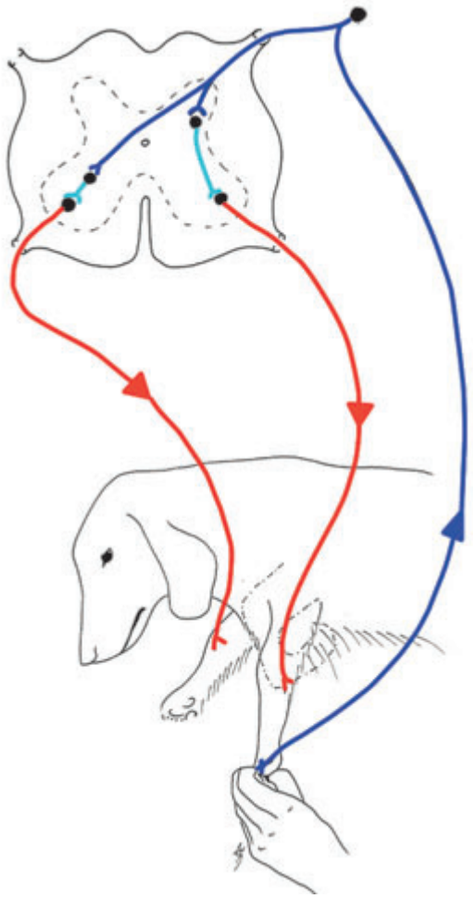


Fig. 1.34
Flexor reflexes in the forelimb. Afferent (dark blue), interneuron (light blue) and efferent (red) pathways.

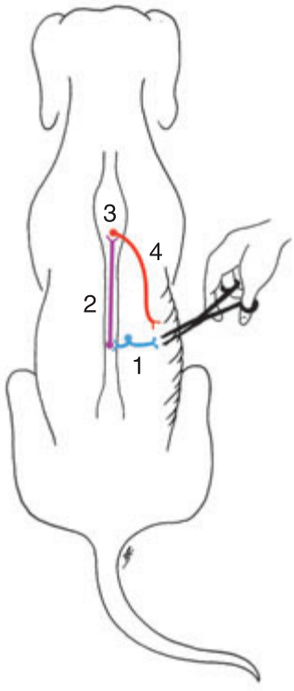


Fig. 1.35
Panniculus reflex. The stimuli are transmitted via afferent sensory fibres (1) to the spinal cord and along ascending pathways (2) to the motor reflex centre C8–T2 (3). The motor nerves (thoracic nerves, 4) arise from this centre and innervate the skin muscles.

Flexor reflex (musculocutaneous nerve, axillary nerve, median nerve, ulnar nerve and radial nerve)
The reflex centre lies between C6–T1. Pinching of the toes, pads or the skin between the toes causes a sudden flexion of the whole limb (Fig. 1.34).

1.4.6.3 Other reflexes

Cutaneous trunci (Panniculus) reflex
Pinching or touching the skin with a pointed object over the dorsal spinous processes from the caudal lumbar region to just below the shoulder blades causes a bilateral contraction of the skin muscles. The skin's nociceptors in specific dermatomes are stimulated. The function of this reflex not only depends on intact afferent and efferent nerves, but also on the ascending tracts between L4 and C8. These ascending tracts are situated on the grey and white matter border. As with all other reflexes, the left and right sides of the animal should be compared. The cutaneous trunci reflex may be absent caudal to a thoraco-lumbar spinal lesion (Fig. 1.35).

Perineal reflex
This reflex depends on the sensory and motor innervation occurring through the pudendal nerves and spinal cord segments S1–S3. Touching the anal or perineal area causes a contraction of the anal sphincter and flexion of the tail, with the latter mediated by the caudal nerves (Fig. 1.36).

Vulvourethral reflex
The neuroanatomy responsible for this reflex is as for the perineal reflex. Touching the vulva causes it to be slightly contracted and displaced dorsally (Fig. 1.37).

Bulbocavernosus reflex
The reflex centre lies between S1–S3. A slight pressure above the bulbocavernosus causes a contraction of the anus.

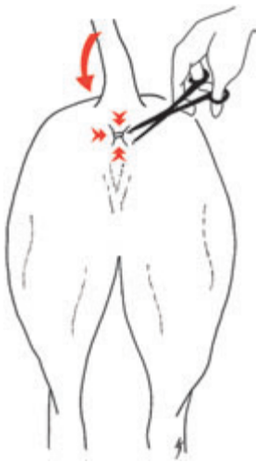


Fig. 1.36
Perineal reflex.

Fig. 1.37
Vulvourethral reflex.

1.4.6.4 Abnormal reflexes

Crossed extensor reflex
This reflex is part of the normal supportive mechanism of the animal; when one limb is flexed, the contralateral limb must extend to bear the weight. This should not occur when the animal is recumbent. The flexor reflex sensory fibres of L4–S3 (pelvic limbs) or C6–T1 (thoracic limbs), send collaterals to interneurons on the opposite side of the spinal cord, which excite extensor motor neurons. When the patient is in lateral recumbency, pinching the toes, footpads or between the toes stimulates a sudden flexion of the stimulated limb and an extension of the contralateral limb. This reflex occurs when there is a lesion of the spinal cord cranial to the reflex centre.

Mass reflex
Occasionally, severe spinal cord damage leads to a generalised dysinhibition of the reflex activity. The stimulation of a single reflex causes a struggling of all the limbs, a wagging of the tail and uncontrolled defensive movements.

1.4.7 Sensory system

Two types of sensory system are differentiated based on the location of the receptors: the superficial receptors (touch, temperature, pain, pressure) and the deep receptors (proprioception). The latter receptors inform the higher centres about the position of the different parts of the body with respect to each other and within space. The sensory system also forms the afferent component of the reflex arc (Fig. 1.38).

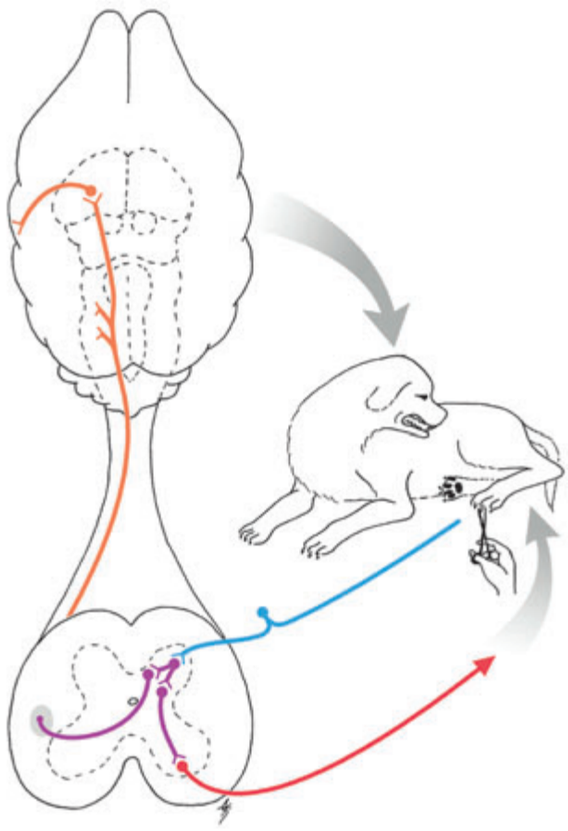


Fig. 1.38
Nociception.

As sensitivity to pressure and temperature are difficult to test and/or determine in small animals, pain sensitivity is the only practically useable component of the superficial sensory receptor system. Proprioception is examined using postural and placement reactions. Deep pain receptors are evaluated by noxious stimulation of deep digital structures such as periosteum.

Anatomy and physiology
The sensory fibres of the internal organs, joints, muscles and skin run via the dorsal nerves into the spinal cord. The fibres that are responsible for superficial pain sensation continue in the dorsal cord cranially and the majority are to be found on the contralateral side. The fibres responsible for deep pain sensation run somewhat differently. They have many intersegmental and suprasegmental connections, which is the reason why they are arranged in a bilateral and diffuse manner in the ascending pathways. Pain transmission ends either in the **thalamus** or some of the pain impulses are projected on to the **cortex**. From here, the respective behavioural reactions such as defence, avoidance and biting are initiated.

A dermatome is a piece of skin which is innervated by one or more nerves. Each dermatome is made up of large differently sized areas of skin (Figs. 1.39, 1.40).

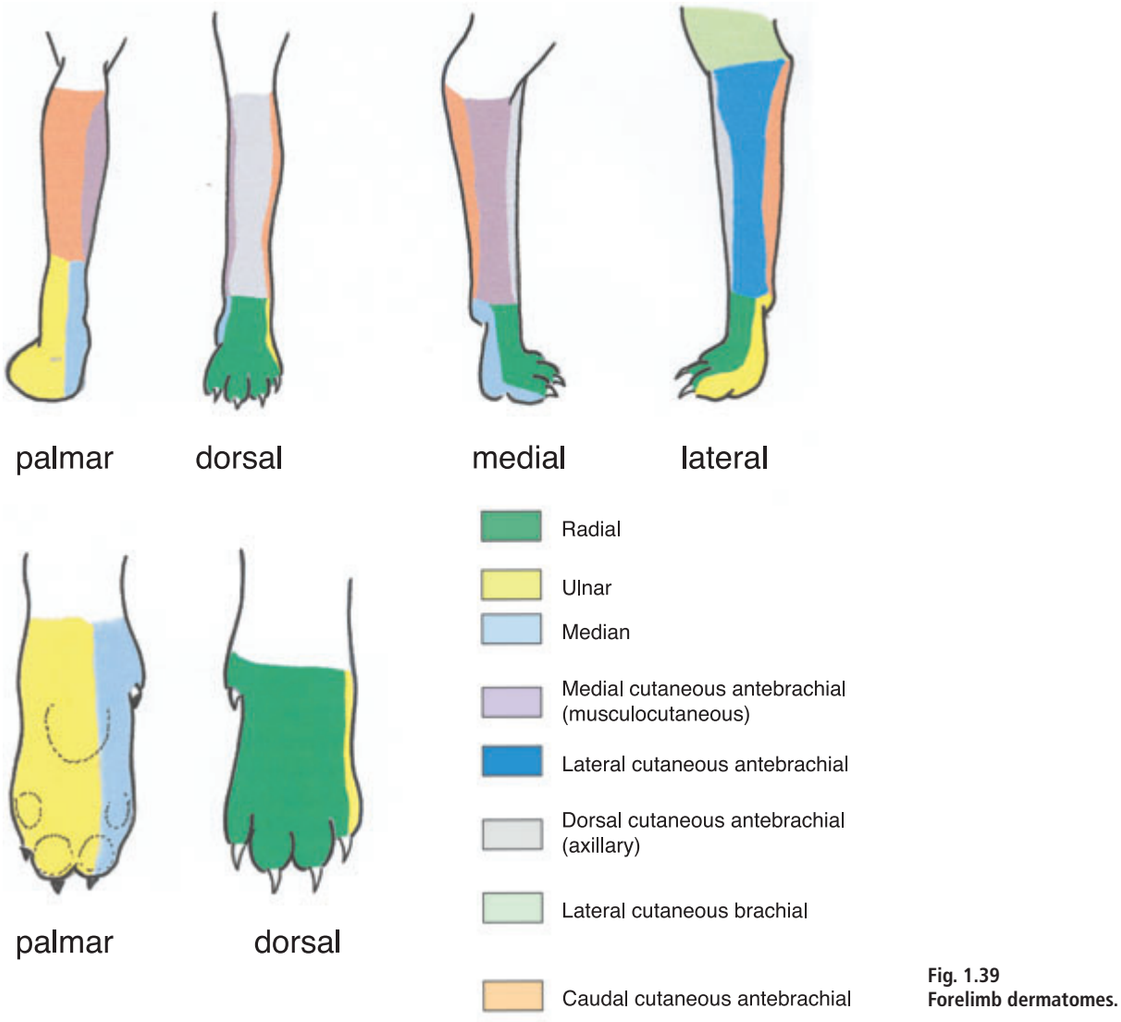


Fig. 1.39
Forelimb dermatomes.

Examination and assessment

In the assessment of sensation, the presence and localisation of pain, the presence of superficial pain sensitivity and the presence of deep pain sensitivity are all examined. By careful palpation of the area to be investigated and by targeted, careful pressure from dorsal and lateral areas with increased pain appreciation (hyperaesthesia) and areas with reduced pain sensation (hypalgesia) can be determined. As a lesion in the CNS results in hypalgesia caudal to the lesion, the limbs should be examined in a distal to proximal direction. The pelvic region and the rump are palpated in a caudiocranial direction.

The haemostat test gives the examiner a general impression of the pain appreciation on the rump and limbs. Stimulation of the skin with a haemostat at different places causes different reactions, either a change in behaviour in the form of defence reactions such as vocalizing, and biting (only reactions that show that the stimulus has been consciously appreciated should be considered as true pain) or a reflex reaction made up of a local skin or muscle contraction, which is not appreciated consciously. A systematic examination of the pain response is especially important when absolute motor dysfunction is present. When superficial pain appreciation is present then deep pain will be present. If the superficial pain reaction is missing, then the skin should be tested with a hemostat to stimulate the deep pain response. Breed specific and individual differences in temperament can make the interpretation of these tests difficult. Many animals overreact to the slightest touch while in others stimuli of a similar intensity are barely noticed.

It should be noted that the flexion of a limb away from a pain stimulus is a reflex reaction and not the expression of a conscious pain response. The deep pain response is the last one to disappear with spinal lesions and is therefore important for the animal's prognosis.

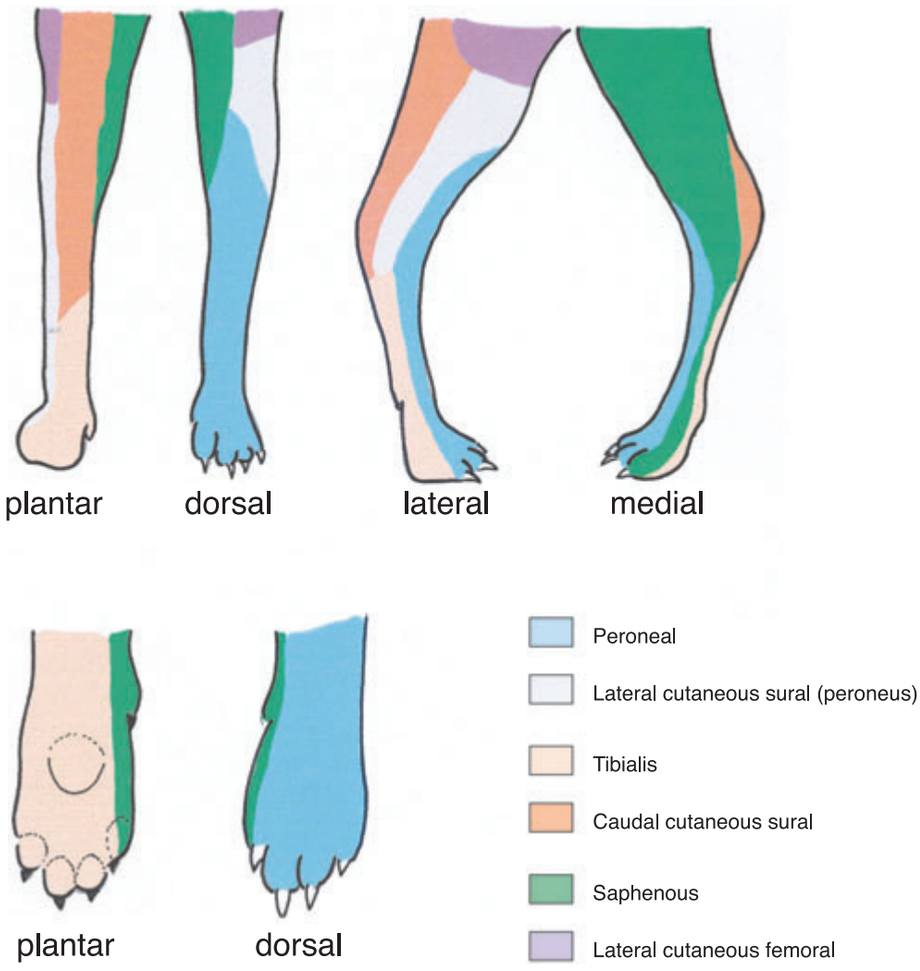


Fig. 1.40
Hindlimb dermatomes.

1.4.8 Localisation

To ensure a definite or suspected diagnosis, the examiner must delineate the pathological process using the neurological examination and its subsequent interpretation. Localisation is based on a neurophysiological interpretation of the clinical signs and the results of the neurological examination. Clinical neurological abnormalities may result from dysfunction of a specific anatomical area; i.e., facial paresis is seen with lesions of the facial nerve (CN VII). However, some clinical signs may result from dysfunction of more complex neuroanatomy or neuroanatomical interactions; i.e., ataxia may result from lesions of cerebellum, spinocerebellar tracts or the vestibular system. Clinical signs with a simple or complex physiology must be differentiated. The former are easy to localise and are the consequence of a lesion of the afferent or efferent nerves or their reflex centres (flaccid paralysis, loss of spinal reflexes and cranial nerve function, etc.). The picture found with clinical signs associated with complex physiological processes are indeed very characteristic of particular anatomical regions; for example, generalised ataxia and hypermetria with lesions in the cerebellum.

The nervous system is divided into seven regions for practical and clinical purposes. Lesions can be localised to one of these following regions:

- Peripheral nerves and nerve roots
- Muscles / neuromuscular junctions
- Spinal cord
- Brain stem
- Vestibular system
- Cerebellum
- Cerebral cortex (including thalamus, hypothalamus and the basal ganglia)

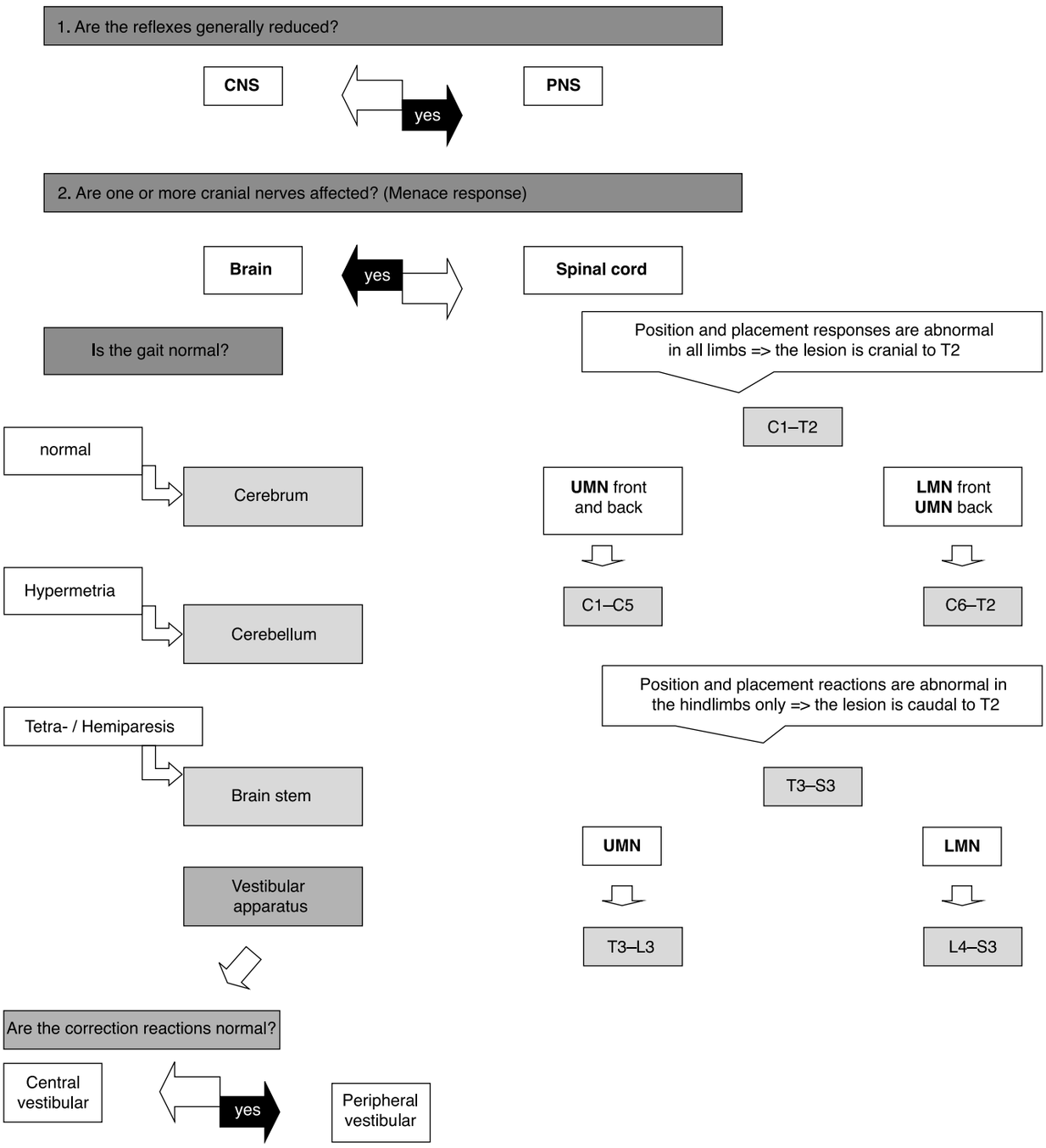


Fig. 1.41
Algorithm for the localisation of neurological disturbances.

The peripheral nerves can be subdivided into thoracic limbs, pelvic limbs, cranial nerves and the sacral nerves. The spinal cord can also be subdivided into the C1–C5 cervical cord, the cervical enlargement (C6–T2), the thoracolumbar cord (T3–L3), the lumbar enlargement (L4–S2) and the sacral cord (S1–S3). Within each region, the right or left side can be affected.

The first step in establishing localisation is the classification of the clinical signs based on whether they originate from a dysfunction of the peripheral nervous system (PNS), the central nervous system (CNS) or extraneural systems. The anamnesis and the results of the neurological examination can help with this classification (Fig. 1.41).

1.4.8.1 Peripheral nervous system

When spinal reflexes, i.e. flexors and extensors, in one or all four limbs are reduced, then the lesion affects the PNS. The majority of the diseases of the PNS affect the limbs. If all limbs are involved, this clinically represents a generalised lower motor neuron (LMN) disease. The LMNS consists of the α-motor neurons, the nerve roots, the peripheral nerves, the neuromuscular endplate and the muscles. The clinical signs depend on the extent of the lesion as well as its chronicity. Paresis, loss of proprioception, reduced muscle tone and muscle atrophy can all be observed. Disease of the LMN of a single limb or cranial nerve is relatively easy to localise.

1.4.8.2 Central nervous system

If the spinal reflexes are not reduced, then the lesion must be in the CNS. To further refine the CNS localisation, it can be divided into an intra- or extracranial lesion. **If one or more cranial nerve functions are affected and/or the menace response is abnormal, then the lesion is localised intracranially (brain)**. In addition, the postural and placement responses in all four limbs may be abnormal. These tend to be more one-sided with focal processes. **Abnormalities of the gait** can help to classify intracranial lesion(s) as affecting the cerebrum, cerebellum, brain stem or vestibular apparatus. Lesions of the cerebrum can induce a wandering, aimless gait, or compulsive pacing. Additionally, obtundation and/or changes in behaviour or seizures can be observed. Hypermetria and intention tremors usually indicate a lesion in the cerebellum. Other abnormalities are also to be expected with a localisation in this region.

A multifocal, disseminated or diffuse disturbance should be considered when numerous regions of the nervous system are affected at the same time.

In addition to observing the gait and testing of the spinal reflexes, postural and placement reflexes, palpitation of the limbs and the dorsum are very helpful in localising hyperaesthesic regions. During the manipulation, the examiner should observe whether signs of pain, e.g. resistance to movement or increased tension in the musculature occur. When the examiner places one hand on the abdomen of the animal while pressing each vertebral segment with the other, an increase in tension in the abdominal muscles is an indicator of a pain response. Pain in the cervical region can also be determined by palpation or manipulation of the neck, followed by lateral and dorsoventral flexion.

Cerebrum

Clinical signs of a lesion in the cerebral cortex include abnormal behaviour and levels of consciousness, seizure activity, head turn, circling and a wandering gait, abnormal postural and placement responses, loss of vision, with an intact pupillary light reflex and reduced nasal sensation. The abnormalities are mainly apparent on the **contralateral** side.

Brain stem

Impairments in consciousness such as obtundation, stupor or coma, disturbances in gait from hemi- to tetraparesis, ipsilateral abnormal postural and placement responses, and dysfunction of cranial nerves are all typical clinical signs of a lesion in the brain stem.

Cerebellum

The cerebellum is a regulatory organ and is involved in the modulation of movement, muscle tone and balance. This organ facilitates well-rounded and fluid movements.

The majority of diseases affecting the cerebellum are diffuse lesions which result in a characteristic collection of clinical signs, most notable of which is **cerebellar ataxia**. The other clinical signs include normal behaviour; a wide-legged stance; standing with the tendency to fall forwards, to the side or backwards; abnormal head movements (intention tremor), manifested as a nodding of the head and dysmetria (hypermetria).

A **delay in initiation** is obvious in postural reactions which are then exaggerated. The cranial nerve examination is normal apart from the menace response which may be absent on the ipsilateral side or there may rarely be nystagmus. The fact that the head is also affected by ataxia helps in the differentation between a cerebellar and a spinal lesion. Focal lesions are rarer and also more difficult to recognise. Abnormal movements with hypermetria on the side of the lesion can be seen. In the acute case of cerebellar disease, another clinical syndrome can be seen: there is increased extensor tone in the

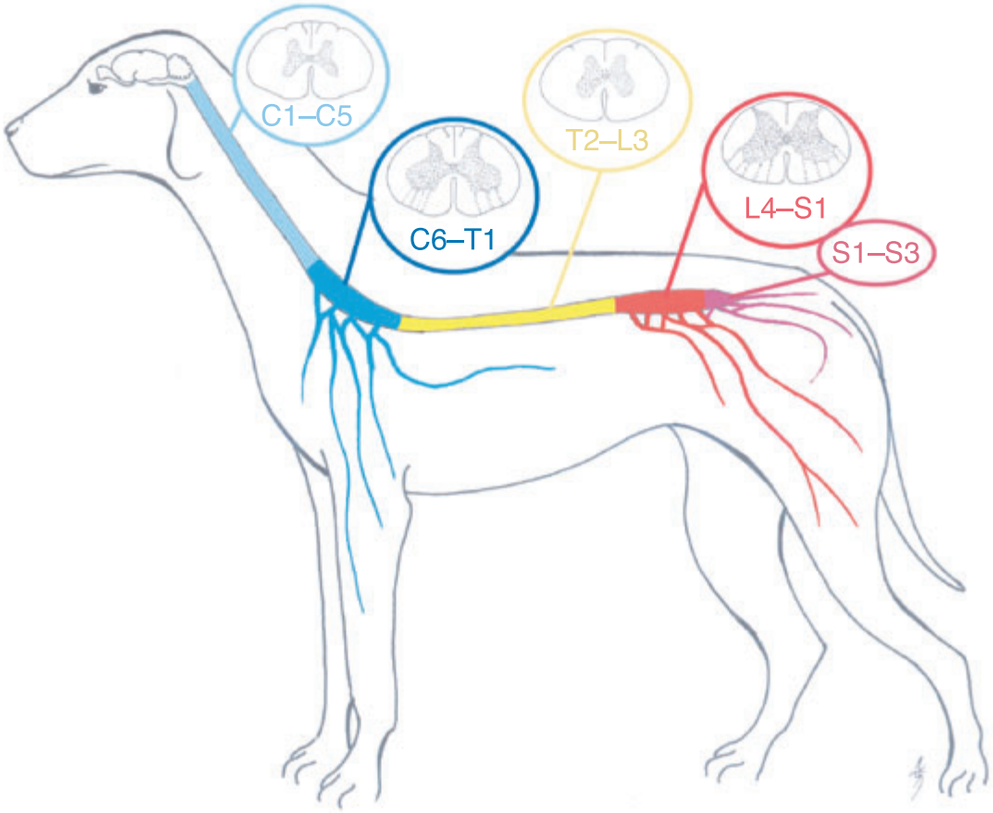


Fig. 1.42
Division of the spinal segments C1–S3 according to abnormalities in the positioning and placement reactions or the spinal reflexes.

thoracic limbs, flexion of the pelvic limbs, and opisthotonus (dorsal extension of the head and neck). Lesions in the flocculonodular lobes of the cerebellum and cerebellar peduncles cause similar clinical signs to those found in vestibular disease, but usually cause a contralateral head tilt (paradoxical vestibular disease)

1.4.8.3 Spinal cord

The spinal cord can be split into four sections: cranial cervical (C1–C5), caudal cervical (C6–T2), thoracolumbar (T3–L3) and the lumbosacral (L4–S3). In the lumbar region, the spinal cord is markedly displaced cranially with respect to the vertebral column (ascensus medullae). As a consequence, the lumbar enlargement lies over the L3–L4 vertebrae and the sacral grey matter over L5. Abnormalities in gait are noticeable with diseases of the spinal cord. Damage to the descending motor pathways (UMN) or of the α-motor neurons (LMN) leads to a paresis or a plegia depending on the severity of the lesion. With damage to the afferent pathways, an incoordination of the limbs occurs (proprioceptive ataxia).

The localisation of a lesion in one of the above sections of the spinal cord can be determined by an assessment of the **spinal reflexes** as well as the **postural and placement reactions**. Lesions that occur cranial to T3 change the postural and placement reactions in all four limbs. Lesions caudal to T2 cause abnormal postural and placement reactions only in the pelvic limbs. In order to allocate a lesion to one of the four spinal cord segments, one must differentiate between the UMN and LMN by assessing the **reflex activity** of all limbs. UMN lesions cause increased spinal reflex activity, while lesions in the LMNs result in reduced spinal reflexes.

If the postural and placement reactions are abnormal in all four limbs, then the lesion is cranial to T3 (Fig. 1.42). Whether or not it lies in the cranial or caudal cervical spinal cord is determined by the assessment of the spinal reflexes.

Normal to increased reflexes in the thoracic or pelvic limbs (UMN) when all four limbs are affected by a gait abnormality mean that the lesion is in the cranial cervical region (C1–C5). Reduced reflexes in the thoracic limbs (LMN) and normal to increased reflexes in the pelvic limbs (UMN) implies a lesion in the caudal cervical region (C6–T2).

If the postural and placement reactions are abnormal only in the pelvic limbs, then the lesion is caudal to T2. Normal to increased reflexes in the pelvic limbs implies a lesion in the thoracolumbar area (T3–L3) and reduced reflexes implies a lesion in the lumbosacral region (L4–S3).

The presence of Horner's syndrome, and an assessment of the panniculus reflex or the perineal reflex can further help in the localisation of a spinal lesion.

Localisation T3–L3

Typical examination results:

Pelvic limb signs: paraparesis, paraplegia, increased or normal reflexes, abnormalities in the postural and placement reactions, possible urinary incontinence, the cutaneous trunci reflex may be absent caudal to the lesion, and there is a loss of deep pain response in the most severe cases.

Localisation L4–L7

Typical examination results:

Pelvic limb signs: flaccid paraparesis to paraplegia, hyporeflexia, areflexia, abnormalities in the postural and placement reflexes, reduced muscle tone, and possible urinary incontinence.

Localisation S1–S3

Typical examination results:

Paresis or paralysis of the urinary bladder and urethral spincters, colon, anal sphincter and tail; analgesia of the perineal and tail regions is present; no disturbances in gait with focal processes.

1.4.8.4 Vestibular system

A differentiation must be made between the peripheral and the central vestibular system for localisation purposes. The peripheral vestibular system consists of inner ear receptors and the vestibulocochlear nerve. The receptor organ is made up of three semi-circular canals lying at right angles to each other. The receptors within these canals measure changes in acceleration and deceleration. The **classical clinical signs** of a lesion in the vestibular system are a head tilt to one side, an ataxia to one side with a tendency to fall, circle, drift, roll over, a pathological nystagmus, **positional strabismus** and abnormal righting reactions.

Table 1.2: Localisation of a vestibular lesion

	Peripheral	Paradoxical	Central
Consciousness	Normal-depression	Normal-depression	Depression-stupor
Behaviour	Normal	Normal	Abnormal
Posture			
Head tilt	Ipsilateral	Contralateral	Ipsilateral
Muscle tone	Abnormal	Abnormal	Abnormal
Gait			
Falling down	Possibly	Possibly	Possibly
Drifting	Possibly	Possibly	Possibly
Ataxia	Usually	Yes	Yes
Cranial nerves	VII (V) (ipsilateral)	Multiple (ipsilateral)	Multiple (ipsilateral)
Horner's syndrome	Yes	No	Very rare; possibly
Nystagmus	Yes	Yes	Yes
■ Horizontal	Yes	Yes	Yes
■ Rotary	Yes	Yes	Yes
■ Vertical	Very rare / no	Yes	Yes
■ Positional	Possible / rare	Yes	Yes
■ Pendulous (tremor)	No	Yes	No
Strabismus	Ventral (ipsilateral)	Ventral (contralateral)	Ventral (ipsilateral)
Postural and placement reactions			
Hopping	Abnormal	Abnormal	Abnormal
Righting	Abnormal	Abnormal	Abnormal
Wheelbarrow	Abnormal	Abnormal	Abnormal
Correction of hindlimbs	Normal to slightly delayed	Abnormal	Abnormal
Reflexes			
Normal to increased	Yes	Yes	Yes
Pain appreciation	Normal	Normal	Normal to reduced

The central vestibular system contains the vestibular nuclei in the brainstem (medulla oblongata), the caudal cerebellar peduncle, and the descending vestibulospinal tracts. The vestibulospinal tract modulates the muscle tone of the trunk and the limbs. This is important for balance, posture, tone and movement. The medial longitudinal fasciculus is responsible for the connection between the vestibular nuclei and the motor nuclei of the extrinsic eye muscles, (III, IV and VI). These tracts are integral for normal nystagmus. A lesion in the central vestibular system causes signs similar to those of a lesion in the peripheral vestibular system: however, differences include possible dysfunction of a number of cranial nerves, an absence of Horner's syndrome and deficits in ipsilateral proprioception. The most reliable point for differentiation between the two systems, is that a normal postural reaction is most indicative of a peripheral lesion and an abnormal one indicative of a central lesion. It is **essential** to differentiate a peripheral lesion from a central one for the purposes of the differential diagnosis and prognosis (Table. 1.2).

Lesions in the caudal cerebellar peduncle can lead to a paradoxical vestibular syndrome, in which the head tilt is to the contralateral side of the lesion (see Fig. 15.1)

Further reading

JAGGY, A., TIPOLD, A. (1999): Die neurologische Untersuchung beim Kleintier und beim Pferd. Opuscula Veterinaria.

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1.5 Ophthalmological examination

Bernhard Spiess

The experienced examiner can assess all twelve cranial nerves during an eye examination. The pupillary light reflex, in addition to the testing of vision, is important as a part of a general patient examination. In comparison to human beings, the examination of the visual field is not as important. The assessment of the position, mobility and movements of both eyes as well as the tear production are of neuro-opththalmic importance.

1.5.1 Examination of vision

The assessment of vision requires the patient's cooperation and is not always easy in companion animals.

1.5.1.1 Menace response test

The simplest and most commonly used test of vision is the menace response test or menace response. A "menacing" movement with the hand is made a short distance away from the eye to be tested. The other eye is covered so that each eye can be tested individually. It is essential that no draught is caused by the hand movement otherwise this could cause the patient to blink. Touching of the long vibrissae in the cat should also be avoided for the same reason.

The menacing movement is recognised in the healthy animal and it responds by blinking. Sometimes a moderate defence reaction can be expected (1).

The afferent arm of this response is the optic nerve (CN II), while the closing of the eyelids occurs due to the orbicularis oculi muscles, which are innervated by the facial nerve (CN III). Integration of the afferent and efferent impulses takes place in the contralateral cerebral cortex.

It should be remembered that the menace response is not a reflex as it is a learned response to a menacing action. Puppies up to the age of 12 weeks do not show a reaction to the menace test (2). Sight should be tested in another manner in such young animals.

1.5.1.2 Dazzle reflex

The dazzle reflex is a subcortical reflex elicited by a strong light. When strong light is shone into an eye, then a slight blinking will occur in both eyes, with the reaction on the contralateral side tending to be weaker (3). The afferent arm is the optic nerve, while the efferent arm of this reflex must have an intact facial nerve. This reflex can be used to determine whether the animal has an intact retina and optic nerve when there are opacities in the eye (cataract, etc.) or when there is no menace response. Although investigations in the decerebrate cat have shown that this reflex remains intact, the dazzle reflex can still be used to provide valuable information about a patient's potential ability to see.

1.5.1.3 Cotton ball test

A useful test for judging vision is the so-called "cotton ball" test. In this test, how the patient's eyes follow a moving object is observed. In young and playful dogs and cats, a small beam of light can be moved backwards and forwards on the floor in the animal's field of vision.

Also, small rolling balls quickly catch the attention of such animals. As a rule, one usually only uses the cotton ball test, whereby a small ball of cotton wool is thrown from behind the animal into its field of vision. A cotton ball is especially suitable for this test as it does not make a sound when falling. A patient with normal eyesight will follow the cotton ball with its eyes and possibly make a corresponding movement of its head. In placid animals, just a slight twitching of the ears is the only indication that the cotton ball has been seen. As with the other tests, the cotton ball test should be repeated a number of times to exclude the possibility of a chance reaction. Each eye should be tested separately using this test (2).

This test is positive in puppies before the menace response is functional.

1.5.1.4 Obstacle course

Under certain circumstances, the previous ophthalmological tests are not adequate for testing any slight impairment in sight. To test a dog's ability to see under different lighting conditions, the animal can be made to go around an obstacle course (3). In this test, a number of different obstacles (e.g. chairs, wastepaper baskets, plastic bags, etc.) are placed in a room unknown to the animal. The dog is held on one side of the room and then is called over to the other side by its owner. It is easy to observe whether the dog can move confidently around the obstacles or if it hesitatingly feels around the objects with its head bent to the floor. This test can be done first of all in strong light and then repeated with the light turned down. Dogs with retinal degeneration are affected, in the initial stages, by a night blindness. In the obstacle course test, such dogs have no difficulty when the room is well lit, but they are obviously slow and uncertain in their movements in semi-darkness, and may even bump into the obstacles. It is often desirable to do this test for each eye separately, but to do this the other eye must be well covered. Not every animal will allow this and they often refuse to move at all or will only move when the "irritating" eye cover is removed. The obstacle course test usually cannot be effectively carried out in the cat.

1.5.2 Examination of the pupillary light reflex and the size of the pupils

The pupillary light reflex (PLR) is a most important neuro-ophthalmological test.

First of all, the symmetry of both pupils is assessed. It is best to judge the dog from a distance of about 1 m through a direct ophthalmoscope. The light reflected from the tapetum lucidum makes the pupils shine brightly, enabling the size of each pupil to be compared exactly. This should be done at first in a bright room and then when the room is darkened. Certain anisocorias are better seen in a bright light and disappear in the dark, and vice versa.

The PLR is always determined in a darkened room. The direct PLR is tested in the eye into which the light is shone and the consensual, indirect PLR is evident in the opposite eye (1). A strong, focal light source is necessary for this test. The anatomy of the PLR is shown in Fig. 1.43.

The quickest and most reliable way of testing the PLR is the so-called pendulum or swinging light test. A focal light is shone in one eye for a few seconds, whereby the direct PLR can be judged. Then the light source is quickly changed over to the other eye. In the healthy animal, this pupil is already narrowed (positive indirect PLR) and remains so (direct PLR). Now when the light source is shone back into the first eye, the pupil in that eye is still narrowed (indirect PLR) and remains so (direct PLR) (2). The PLR allows a localisation of the lesion in unilateral visual deficits. In this (normal) case one talks of a **negative** pendulum light test. With a **positive** pendulum light test, the direct PLR on the unaffected side is physiological, meaning that the pupil narrows on direct illumination. When the light is then shone in the affected eye, its pupil is initially narrowed (positive indirect PLR), but it then dilates under the direct illumination (negative direct PLR). If the light is then changed back to the unaffected side, its pupil is initially dilated (negative indirect PLR) and then narrows when the light hits the eye. The positive pendulum test is pathognomonic for one-sided changes in the retina or unilateral prechiasmatic lesions of the optic nerve (4).

The lateral and medial sections of the retina can be illuminated separately with a good focal light source, and so their respective PLRs can be assessed. In this manner, the visual field can be approximately tested.

1.5.3 Palpebral reflex

The palpebral reflex tests the sensory innervation of the palpebrae by the trigeminal nerve and the motor innervation of the orbicularis oculi muscle via the facial nerve. This reflex is elicited by a light tapping of the medial and lateral canthi, testing the ophthalmic and maxillary branches of CN V respectively.