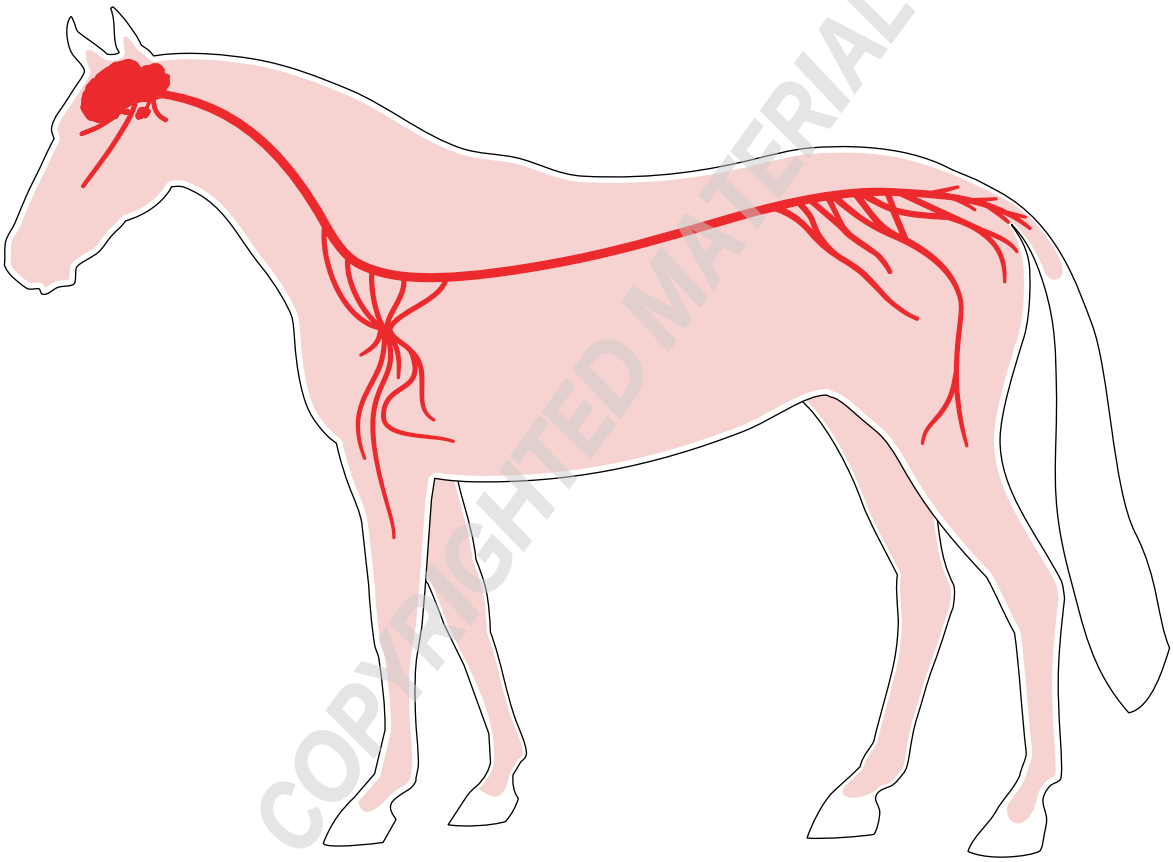
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Practical neuroanatomy



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Some of the most rewarding aspects of clinical neurology involve being able to associate observed signs and syndromes with neuroanatomic sites of the lesions. This may, for example, be experienced when recognizing a syndrome of ataxia in a purebred patient and associating it with a familial cerebellar disorder for a client. Alternatively, one may be able to explain to a producer how a certain lesion, detected by an astute neurologic

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examination of one representative animal and confirmed at postmortem examination is the cause of the clinical syndrome observed in the herd. The basic requirement for achieving this degree of diagnostic acumen is an understanding of applied neuroanatomy.

This chapter provides the basic information necessary to allow the clinician to appreciate the fundamentals of a neurologic examination and to interpret, accurately, the results of such an examination. As the clinician becomes adept at these tasks, further anatomic details may be sought. These can be found in the texts listed in the references.

At this point, a plea is made for a clear use of anatomic terms, based on *Nomina Anatomica Veterinaria, Nomina Embryologica Veterinaria* and *Nomina Histologica,*[1] along with the clinically applied terms used in functional neuroanatomy,[2–4] clinical neurology,[5–7] and veterinary neuropathology.[8–10]

Basic descriptive terminology

The following is a review of basic descriptive terminology; note that the derivation, abbreviation, combining form, synonym, or explanation is given parenthetically.

The central nervous system (CNS) consists of the brain (encephalo) (Figure 1.1) and spinal cord (myelo). It contains collections of neuronal cell bodies or somata in layers (laminae), nuclei, and columns of gray matter (polio). Tracts, sheets, and pathways of dendritic (afferent) and particularly axonal (efferent) processes of these cell bodies make up the white matter (leuko). These processes make up most of the CNS along with their fatty, myelin (myelino) coats. In tall, large animals, some of these neuronal fibers extend 2–3 m, and many exit and enter the CNS via the cranial and spinal nerves of the peripheral nervous system (PNS).

Structurally, the forebrain (prosencephalon) is composed of the cerebral hemispheres (most of the telencephalon), thalamus and hypothalamus (diencephalon), hippocampus, basal nuclei, and the limbic system, all situated in the rostral fossa of the neurocranium. The middle fossa contains the midbrain (mesencephalon). Caudally, the hindbrain (rhombencephalon) is composed of the cerebellum and pons (together with the metencephalon) and the medulla oblongata (myelencephalon) that all reside in the caudal fossa. Both the cerebrum (cerebro) and cerebellum (cerebello) have their own outer (cortical) and inner (medullary) portions, composed particularly of gray matter (neuronal cell bodies) and white matter (neuronal fibers), respectively. The remainder of the brain comprising the midbrain, pons, and medulla oblongata makes up the brainstem. This contains white matter pathways passing to and from brain and spinal cord regions, and gray matter, mostly contained in nuclei as relay centers and sensory and motor nuclear areas for the body, including the cranial nerve nuclei.

> To avoid some confusion, vertebral levels are labeled as C7, T3, L4, S5, Ca6 (caudalis as opposed to coccygeal), *etc.*, and spinal cord segments are labeled as C_8, T_3, L_4, S_5, Ca_6, *etc.*

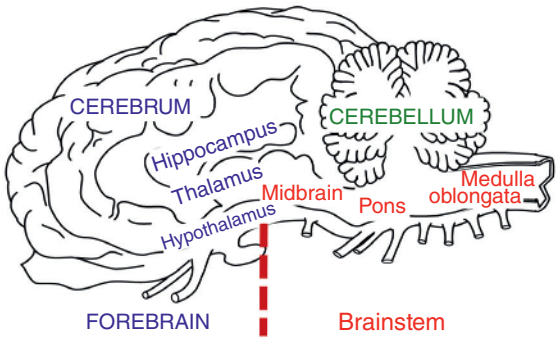


Figure 1.1 Basic areas of the brain can be readily recognized on this diagram of a median section of a horse brain. The terminology used in this book is shown. Wherever possible the authors use the terminology of *Nomona Anatomica Veterinaria*; however, some license is taken with respect to forebrain, which here refers to the prosencephalon plus diencephalon, as clinically, lesions in these collective regions generally present the same syndromes. It is important to recognize these various areas when sending brain sections for histopathologic examination.

The spinal cord is the conduit between the brain and the peripheral spinal (non-cranial) nerves and their innervated structures of the body. Thus, it contains all the afferent and efferent neuronal fibers connecting to and from the brain as superficial white matter tracts. It has a cervicothoracic (brachial) enlargement (intumescence) and a lumbosacral (pelvic) enlargement at the levels of the thoracic and the pelvic limbs, respectively. These swellings are the result of a higher density of neuronal cell bodies at these sites, collected in the butterfly-shaped, central gray matter and supplying the sensory and motor spinal nerves for the limbs.

Clinically, the autonomic nervous system functions independently and involuntarily from the rest of the nervous system, receiving afferent input from the environment via the basic and special senses to maintain the body's internal milieu. The parasympathetic component has a brainstem and sacral outflow to the

head and body, whereas the sympathetic component has a thoracolumbar outflow.

The entire CNS is protected within the bony neurocranium of the head and in the vertebral canal within the vertebral column. It is covered by meninges consisting of the thick (pachy) dura mater and thin (lepto) arachnoid and pia mater. Between the latter two membranes, is cerebrospinal fluid (CSF), produced by the choroid plexuses of the lateral and fourth ventricles. This fluid also fills the cavities within the brain (ventricles) and spinal cord (spinal canal), which are lined by ciliated ependymal cells.

Because we refer to vertebral structures very often, it is worth reviewing the recommended correct nomenclature as in *Nomina Anatomica Veterinaria* http://www.wava-amav.org/wava-documents.html

Columna Vertebralis

Corpus vertebrae
Extremitas cranialis (Caput vertebrae)
Extremitas caudalis (Fossa vertebrae)
Crista ventralis
Arcus vertebrae
Pediculus arcus vertebrae
Lamina arcus vertebrae
Foramen vertebrale
Canalis vertebralis
Spatium interarcuale
Foramen intervertebrale
Incisura vertebralis cranialis
Incisura vertebralis caudalis
Foramen vertebrale laterale
Sulcus n. spinalis
Processus spinosus
Processus transversus
Processus costalis
Processus articularis cranialis
Processus articularis caudalis
Processus accessorius

As well as meningeal cells, ependymal cells, neurons (somata and their axons and dendrites), and blood vessels, several other types of supporting, protective, and nutritive glial (glue) cells (neuroglia) make up a large part of the volume of the CNS. The largest of these glial cells are the astrocytes with their star-shaped processes. These cells basically act to support the CNS, which has little cytoskeletal framework. They also act as pseudofibroblasts and can lay down collagen in response to injury to the CNS. Originally, the processes of other small glial cell types were thought to be few (oligo); these cells being named oligodendrocytes. We now know that their processes are extensive and extend to and maintain all the myelin sheaths covering CNS axons. The small microglial cells appear to be the tissue macrophages of the mononuclear phagocytic system within the CNS, responding along with migrating inflammatory cells from the circulation when there are inflammatory processes occurring in CNS tissues.

The neuronal processes in the peripheral nervous system (PNS) with their myelin sheaths are called nerve fibers and make up the nerve roots, nerve plexuses, and peripheral nerves. Some neurons, particularly those in the autonomic nervous system and the sensory neurons, have their cell bodies in aggregations known as ganglia. Also, several networks of interwoven nerves (plexuses) occur in the PNS, the largest of which are the brachial plexus supplying the thoracic limbs and the lumbosacral plexus for the pelvic limbs.

The cells that ensheathe PNS nerve fibers are Schwann cells. These assist in maintaining a framework for nerves, as well as producing the abutting layers of myelin that surround all the larger fibers, allowing quite rapid saltatory (leaping, jumping) conduction of electrical impulses. The PNS has a fibrous connective tissue cytoskeleton that consists of the epineurium that wraps around a whole nerve, the perineurium that surrounds a bundle or fascicle of fibers, and the endoneurium that separates the individual nerve fibers.

Functional neuroanatomy

Probably the most important structural and functional unit of the nervous system is the simple reflex pathway (Figure 1.2). A neurologic examination basically involves testing simple and complex reflex pathways and interpreting the effected reflex activity and complex responses (see Chapter 2, Neurologic Evaluation).

A simple spinal reflex pathway, depicted as a patellar tendon reflex in Figure 1.2, is composed of two neurons. Stimulation of a sensory stretch receptor in a tendon with its sensory neuronal cell body in the dorsal root ganglion stimulates an efferent motor neuron. These motor neurons are usually alpha motor neurons and are collectively referred to as **final motor neurons**, having their cell bodies in the ventral gray matter of the spinal cord (and in cranial nerve nuclei in the brainstem). Reflex motor responses to sensory stimuli will occur without any

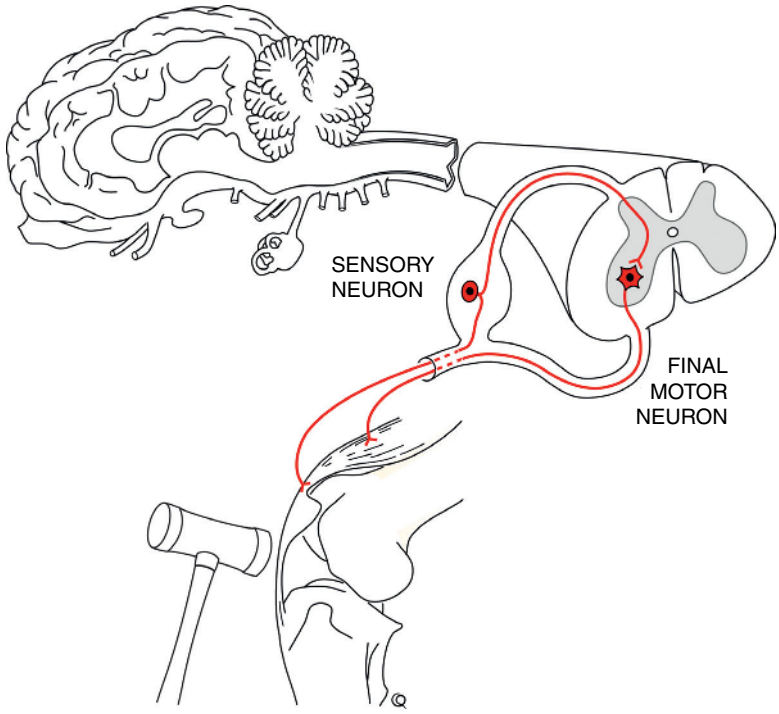


Figure 1.2 Basic monosynaptic spinal reflex pathway (patellar reflex) showing the sensory neuron synapsing on the final motor neuron. In many other reflex pathways, there is at least one internuncial neuron within the CNS allowing further modulation of the reflex.

other afferent or efferent connections within the CNS; so long as these two (or more) neurons comprising the reflex arc, as well as the sensory nerve ending, the neuromuscular junction, and the effector muscle, all are intact. In contrast, lesions, due to any cause, that damage parts of the reflex pathway will suppress those reflexes. Indeed, if the damage is to the alpha motor neurons in the ventral gray matter of the spinal cord or the cranial nerve motor nuclei, or to their peripheral axons, there will be loss of reflexes and severe weakness and ultimate muscle atrophy, *i.e.*, signs of a final motor neuron lesion.

The various reflex pathways with their respective final motor neurons throughout the brainstem and spinal cord are controlled for voluntary movement by neurons in motor centers in the brain which are collectively referred to as **central motor neurons**. Figure 1.3 depicts generic central motor pathways. Corticospinal motor pathways with neuronal cell bodies in the cerebral cortex (A) are very important in primates but do not appear to be very important in initiating voluntary limb and body movement in domestic animals. Quite massive lesions destroying the cerebrocortical motor centers that would be devastating in higher species do not cause permanent demonstrable abnormality in the gait of large animals. After the acute effects of such large lesions in our veterinary species have resolved, there can be subtle deficits in motor functions such as jumping fences and hopping on the thoracic limb opposite the side of the lesion. In contrast, quite small lesions in the midbrain and medulla oblongata usually result in hemiparesis or tetraparesis because of damage to central motor centers and tracts such as the rubrospinal (Figure 1.3B), reticulospinal (Figure 1.3C), and vestibulospinal (Figure 1.3D) pathways. These central motor neuronal pathways tend to have a calming effect on reflexes, particularly those involved with supporting the body against gravity such as the patella reflex. Because of this, central motor pathway lesions do not suppress reflexes and can indeed result in hyperactive reflexes and responses. The major role of these central motor pathways however is to direct the various final motor neurons in (voluntary) movement such that central motor pathway lesions will result in poor or absent voluntary effort (paresis or paralysis) in the face of very active spinal (and cranial) reflexes.

Just as brain motor centers help control the final motor neurons within the reflex arcs, the sensory inputs to these arcs are relayed to brain centers to give feedback on position sense (proprioception) touch, and pain perception (nociception) (Figure 1.4). Sensory pathways travel to the thalamus, and probably to the somatosensory cerebral cortex, for the perception of pain. These pathways are multisynaptic

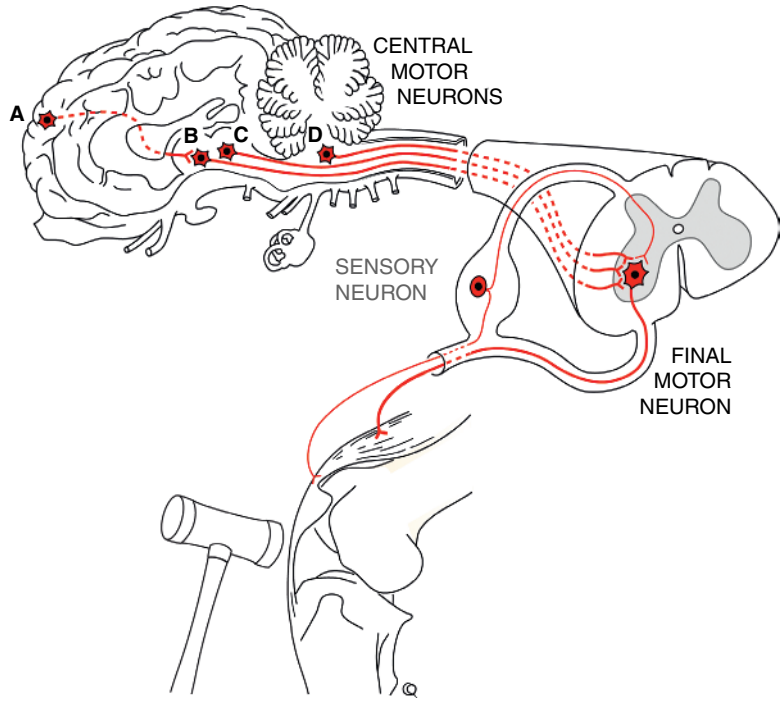


Figure 1.3 Central motor neuronal pathways predominantly originate in the brainstem and synapse on final motor neurons to effect motor activity. Depicted here are A the cortico (rubro) spinal, B the rubrospinal, C the reticulospinal, and D the vestibulospinal neurons and tracts.

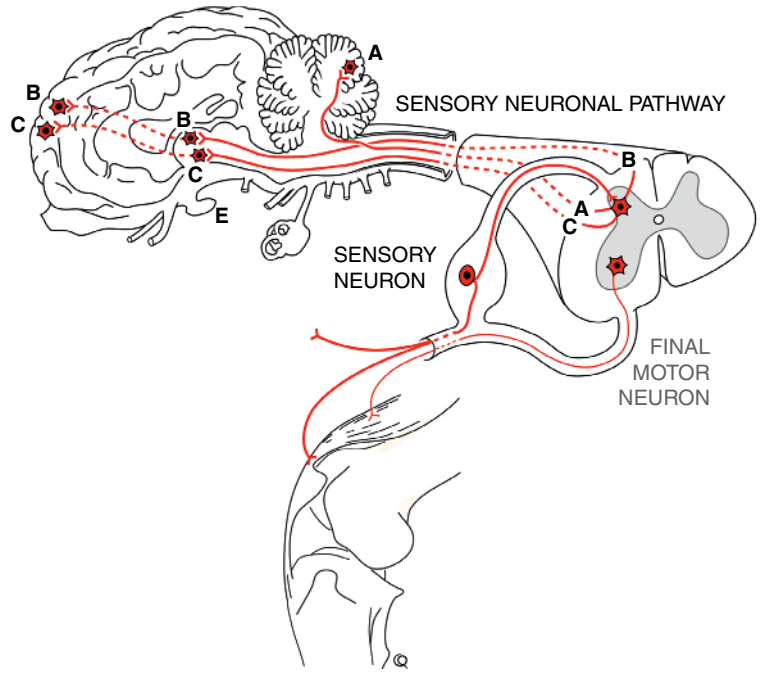


Figure 1.4 Sensory pathways convey somatic, proprioceptive, and visceral input to brain centers. Subconscious proprioceptive neurons A project to the cerebellum, whereas conscious proprioceptive neurons B and somatic and visceral afferent neurons C project to the thalamus and ultimately project to forebrain centers.

and contain small fibers, both characteristics making them resistant to interruption. Proprioceptive pathways that are presumed to be consciously perceived (position sense at rest) travel to thalamic and thence to cerebral conscious proprioceptive centers. Other subconscious proprioceptive information (movement sense) is contained in spinocerebellar tracts that pass directly to the cerebellum.

In summary, damage to the spinal cord at the level of a reflex arc at its final motor neuron will denervate the effector muscle, resulting in flaccid (loss of tone) paralysis with degrees of hypotonia and hyporeflexia

at the level of the lesion. In contrast, a similar severe lesion cranial or rostral to a reflex arc will result in hypertonic (spastic) paralysis. The latter is characterized by loss of voluntary motor function as with a final motor neuron lesion, but the reflex is not diminished. Indeed, the reflex may be released from the calming influence of its controlling central motor pathways and be hyperactive with concomitant hypertonia (spasticity—*sic*.) or stiffness in body parts such as limbs. When a central motor pathway lesion causes a degree of spastic paralysis, the involvement of adjacent proprioceptive and nociceptive sensory pathways also results in ataxia and possibly hypalgesia, respectively.

Certainly, the concept of considering motor activity on the basis of "upper" and "lower" motor neuron function and lesions, even for quadrupeds, is entrenched in veterinary neurology, although not without some comment.[3,5] Anatomically, upper and lower are not acceptable (with the exception of some cranial structures such as eyelids) as terms of direction in veterinary anatomy (*Nomina Anatomica Veterinaria,* 6th Edition),[1] making the use of these descriptors confusing to say the least. Also, in contrast to "upper" and "lower" motor function generators and pathways, the concept of central pattern generators has its merits when considering gait and posture in humans and animals.[11] In this model, there is a generator of highly developed skilled motor activity initiated, in primates, in the cerebrum. A further generator of motor function, most prominent in our large animals, resides in the midbrain and rostral medulla oblongata. Finally, there is a generator at the spinal level that can initiate reflex actions and indeed probably is responsible for primitive gait patterns (viz. "spinal walking"); the interested reader may find further reading on this concept quite intriguing.[12–15] For several reasons, therefore, there is a strong argument in veterinary anatomy and neurology to dispense with upper motor neuron (UMN) and lower motor neuron (LMN) terminologies: a strategy we have accepted for this text. The CNS pathways involved with initiating motor function and maintaining appropriate muscle tone and hence body posture are referred to as **central motor pathways** that directly influence the **final motor neurons** innervating muscle to effect all motor functions. Many final motor neuron somata reside in the brainstem and spinal cord of the CNS with axons in the PNS, the exceptions being the presynaptic final motor neurons of the autonomic nervous system (ANS) which are wholly in the periphery.

The cerebellum is the major coordinating center for voluntary movement (Figure 1.5). It synthesizes impulses received from the cerebral cortex, the brainstem motor centers, and spinocerebellar and vestibular (special) proprioceptive pathways. It then provides feedback impulses to the motor centers to coordinate all motor functions.

Cranial nerves (CNs), numbering I through XII, leave the forebrain and brainstem (Figure 1.6). Some are involved with specialized modalities such as smell (CN I), sight (CN II), and balance and hearing (CN

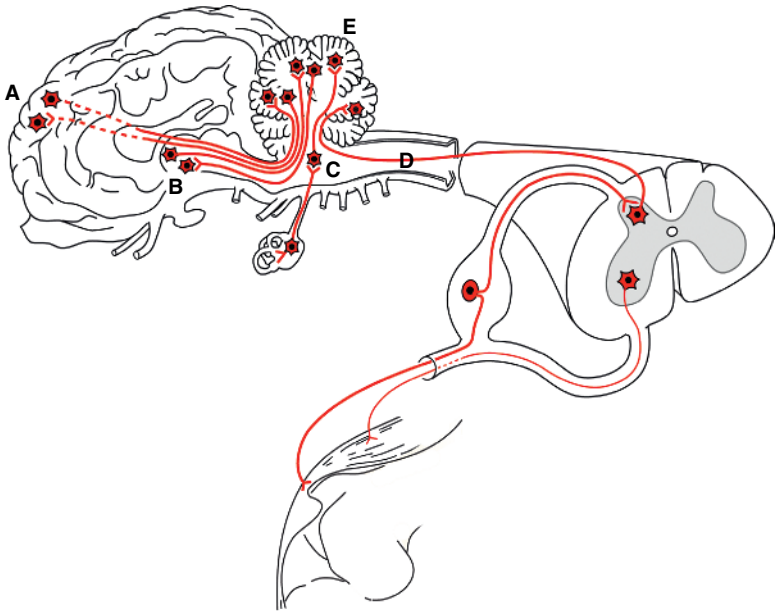


Figure 1.5 Important cerebellar connections include subconscious general proprioceptive input from the body such as via spinocerebellar pathways shown as D (same as A in Figure 1.4). Other input is received from cerebrocortical A and brainstem B motor areas and vestibular centers C. All cerebellar efferent output is from cerebellar cortical (Purkinje) neurons connecting to brainstem B and cerebrocortical A motor areas.

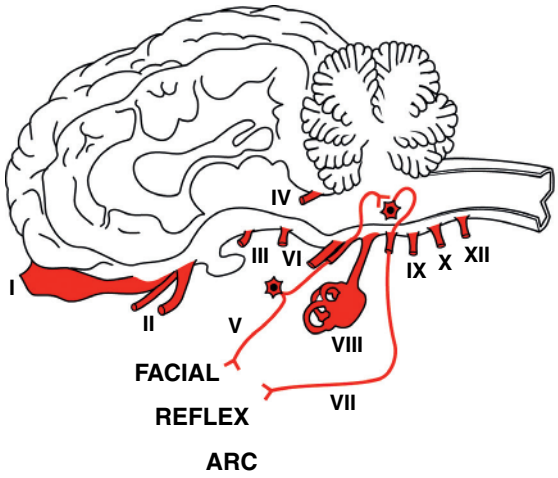


Figure 1.6 Some cranial nerves are involved with specialized modalities such as smell CN-I, vision CN-II, and balance and hearing CN-VIII. Others innervate extraocular muscles CNs-III, IV, and VI, pharynx and larynx CNs-IX and X, and tongue CN-XII, while some are involved with reflex function such as facial reflexes CNs V-VII, similar to spinal reflex arcs.

VIII). Some innervate eyeball muscles (CNs III, IV, and VI), muscles of facial expression (CN VII), pharynx and larynx (CNs IX and X), and tongue (CN XII). Others are involved with reflex functions such as facial reflexes (CNs V and VII) and the gag reflex (CNs IX and X). These cranial final motor neurons have central motor pathways controlling them. A final motor neuron lesion at the level of a CN or its nucleus can paralyze the motor function of the CN. Alternatively, a lesion affecting the efferent central motor pathways that influence the final motor neuron can curtail such CN motor function. As with spinal reflexes, in the latter case the structure (*e.g.*, face) shows variable hypertonic, spastic paralysis (*e.g.*, grimacing), with intact or hyperactive reflexes.

Consciousness or mental awareness must be maintained within the forebrain (Figure 1.7A) for neurologic functions to occur. Numerous sensory inputs, including light, sound, touch, gravity, and metabolic and endocrine factors, interact, usually on an area of the brainstem known as the reticular activating system (Figure 1.7B). This system, by relaying impulses to the forebrain and brainstem, maintains the state of awareness and activity.

The final concept of clinical neuroanatomy important in interpreting large animal neurologic examinations is that of the specialized areas of relatively well-developed functional centers that exist in the forebrain, but having brainstem connections, as introduced in Figure 1.8. The frontal and prefrontal cerebral

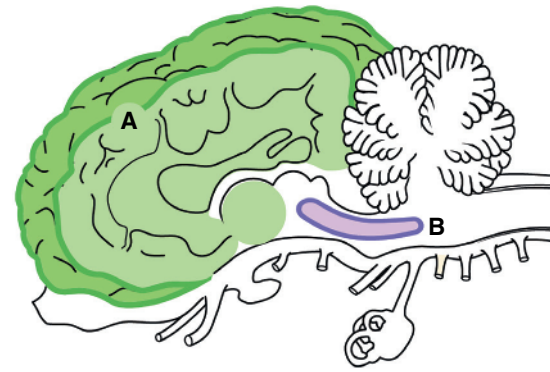


Figure 1.7 A state of alertness or consciousness is maintained by the forebrain A and particularly the reticular activating center B. These regions receive input from many other regions of the nervous system and are interconnected.

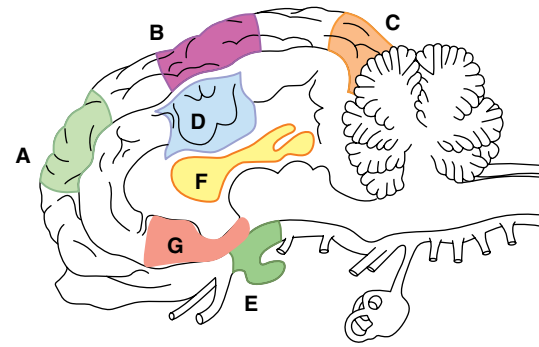


Figure 1.8 Functional regions of the forebrain include the frontal cortex A involved with mentation, behavior, and motor activity; the parietal cortex B involved with pain perception particularly from the head and conscious proprioception; the occipital cortex C involved with vision; the temporal cortex D involved with behavior and hearing; the hypothalamus and pituitary E involved with autonomic and endocrine functions; and the deep-seated limbic system F involved with innate behavior.

lobes (A) are involved with mentation and behavior, and with fine motor activity at least in primates. As in humans, the parietal lobes (B) integrate conscious proprioceptive and nociceptive input in the large animal species. Certainly, vision appears to require an intact occipital cortex (C). Control of normal learned behavior and perception of sound may be functions of the temporal lobes (D). The hypothalamus and its pituitary gland (E) help control autonomic and endocrine functions of the body, whereas the deeper, old part of the brain known as the limbic system (F) may be involved with innate behavioral patterns. Finally, the basal nuclei (G) do appear to have some motor control at least for cranial nerve function in horses. Irrespective of the

regions of the cerebral cortex, large lesions can occur which in their stable state do not result in permanent disability; so-called silent areas (e.g. Figure 2.5).

With this understanding of functional neuroanatomy and a little practice, a neurologic examination can be readily performed and interpreted. An outline of such a neurologic examination is given in the next chapter.

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