Ivan T. Draper

Lecture Notes on Neurology

Sixth edition

LECTURE NOTES ON

Neurology

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SIXTH EDITION

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Preface

Clinical neurology is founded upon the analysis of an accurate history and examination, interpreted in the light of a knowledge of anatomy and of the common neurological illnesses. These notes are intended to provide a basis for this exercise. They were designed for use in conjunction with a formal course of instruction and as a means of rapid revision. This format imposes limitations on the amount of detail which can be provided and it makes no allowance for controversial opinions. My approach to neurology owes a great deal to my own teachers, especially to Professor J.A. Simpson and the late Dr J.B. Stanton, and to my colleagues at the Institute and elsewhere.

I.T.D.

Part 1
The Structure and Function of the Nervous System

Contents

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Chapter 1
The Functional Organization of the Brain

The complexity of human behaviour, the range of man's imagination, the accuracy of his perception, his speech and the precision and power of his movements are products of a normally functioning nervous system. The basic unit of the nervous system is the nerve cell or neuron of which there are 10–50 000 000 000. The neuron is a relatively simple structure whose response is limited to the generation of a standard impulse. The variety and adaptability of neural function is achieved by the almost limitless number of connections which exist between the neurons.

Each neuron has a number of fibrous projections from the nerve cell body. There is one long fibre, the axon, which transmits the efferent nerve impulses. The shorter branching fibres are called dendrites: each of these has a number of stubby spines which form the receptor surfaces for the synapses between neurons. The dendritic spines can accumulate energy from a number of subthreshold stimuli from one axon or from different axons. Eventually the spine discharges and an impulse is transmitted to the cell body. This mechanism of cumulative responses adds spatial and temporal dimensions to neuronal activity. (Chains and networks of neurons are activated for various receptor (sensory) and effector (motor) functions.

Particular areas of the cortex of the brain are committed to specific functions (Fig. 1). The motor cortex is responsible for generating simple movements and the various primary sensory areas register the elemental sensory stimuli from the environment. The analysis and interpretation of such sensory stimuli take place in the tempero-parietal lobes, and is unique to that individual and his particular experiences. The generation of complex skilled movements is a function of the parietal lobes and associated motor areas. The left hemisphere is further specialized for language and abstract reasoning while the right hemisphere is the site for spatial analysis.

The fibre tracts connecting one area of cortex with another, and one hemisphere to the other, are of primary importance to this correlative and interpretive role of the brain and the generation of skilled movements.

Cortical lesions are associated with specific clinical deficits. Lesions involving the subcortical tissues are likely to disrupt interconnecting

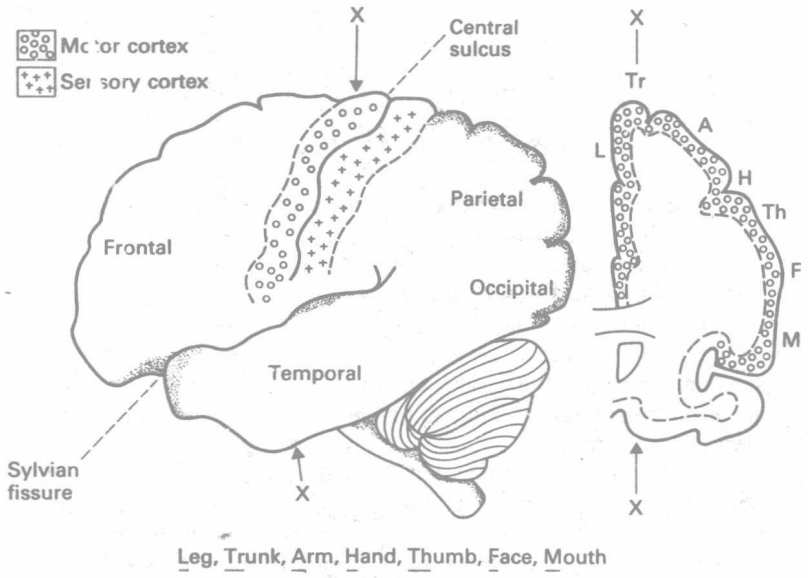

Fig. 1. The brain stem from the left side and the left hemisphere in coronal stem section showing the somatic representation over the motor cortex.

fibre tracts and may result in disorders of interpretation (agnosia), complex movements (apraxia), and language (dysphasia)—these are sometimes called disconnection syndromes.

Neurological diagnosis is based upon deductive analysis of symptoms and signs. It requires a working knowledge of the anatomy and function of the nervous system for the localization of lesions. In general, the right hemisphere receives sensation from, and controls the movement of, the left side of the body and vice versa. However, upper motor neuron fibres from both hemispheres innervate the muscles of the upper part of the face, jaw, neck and trunk, preserving these movements in the presence of hemiplegia.

Irritative lesions provoke positive symptoms, e.g. a scar on the motor cortex may induce motor seizures, a temporal lobe lesion—auditory hallucinations. Destructive lesions cause a loss of function, e.g. infarction of the occipital lobe causes hemianopia. Destructive lesions may also remove inhibitory influences and thus release reflex postural activity at a lower level in the nervous system.

The function of the *prefrontal cortex* in health is uncertain. There are, however, symptoms which point to disease in this area:
Mental changes—apathy, forgetfulness, indifference to his actions or personal condition. Inappropriate humour;
Ataxia—clumsiness and staggering which may mimic a cerebellar lesion;
Re-appearance of primitive reflexes, including grasp reflex and oral feeding responses.
Bilateral lesions may cause a profound akinetic state.

Motor cortex

Destructive lesions cause contralateral upper motor neuron weakness involving whole muscle groups and movements rather than individual muscles, and especially affecting fine manipulation. Lesions involving the lower part of the precentral gyrus affect movements of the face, mouth and tongue, and in the left hemisphere are associated with disorders of expressive language.

Irritative lesions cause focal motor or Jacksonian motor seizures. Jacksonian seizures are typified by spreading clonic jerks, e.g. from the thumb to the fingers, to the hand, to the forearm and arm. The irritative process spreads from a focus on the cortex along the precentral gyrus. The thumb, a corner of the mouth, or the great toe, are frequent points of origin. Lesions between the two hemispheres involve both legs and sometimes the bladder.

Accessory motor cortex

This lies anterior to the cortical motor respresentation for the lower limb, on the medial surface of the hemisphere. Irritative lesions in the accessory motor cortex give rise to tonic postural movements of the head and limbs.

Sensory cortex

The post-central gyrus is the site of tactile localization and discrimination. Irritative lesions of the sensory cortex give rise to contralateral paraesthesiae which may be localized or spreading in the same way as the similarly caused symptoms in the adjacent motor cortex. Destruction causes loss of sensation.

Parietal lobe

This extensive area of the brain cannot be related to specific parts of the body. Rather it is responsible for the interpretation and correlation

of sensation. There may be any form of sensory loss but astereognosis and diminished two point discrimination are outstanding.

Astereognosis (tactile agnosia). This is a difficulty in recognizing the size, weight, texture and shape of an object by touch alone.

Two point discrimination. This tests the patient's ability to recognize simultaneous but spatially separate stimuli. The normal individual can distinguish the two points of a pair of dividers only 2 or 3 mm apart when they are pressed on to the pulp of his finger. The accuracy varies from individual to individual and over different parts of the body. The most significant finding is a difference between the two sides of the body.

Sensory suppression may be found. If stimuli are applied simultaneously to both sides of the body, the sensation is neglected on the side contralateral to the affected parietal lobe.

The patient may experience temporal and geographic disorientation, so that he loses his way when in a normally familiar neighbourhood or even inside his own house.

Two well-delineated syndromes are associated with parietal lobe lesions, Gerstmann's syndrome and disorders of body image.

Gerstmann's syndrome is found only with lesions of the dominant hemisphere. It consists of right/left disorientation, inability to recognize the individual fingers, defective calculation and agraphia.

Disorders of the body image accompany lesions of the non-dominant hemisphere. The individual may neglect an otherwise useful limb, deny ownership of a limb, and fail to recognize the disability of a paralysed limb.

Lesions in the dominant parietal lobe are associated with interpretation of spoken and written language (p. 39).

Precise patterned movements are dependent upon a continuous analysis of muscle and joint and environmental sensory stimuli and parietal lesions cause disorders of consciously organized movements—apraxia (p. 20).

Occipital lobe—visual cortex

Irritative lesions may cause crude visual sensations—such as flashes of lights in the contralateral half field. Destructive lesions cause visual field defects (p. 50).

Temporal lobe

Those parts of the cerebral cortex directly concerned with taste, smell

and hearing are situated in the temporal lobe. In addition, it is responsible for the organization of memory, and it has close functional connections with the association areas of the visual cortex and somatic sensory cortex. It is intimately connected with the hypothalamus and so with the visceral motor system and the physical control of emotions.

Disease of the temporal lobe gives rise to characteristic disorders of memory, hallucinations of smell, taste, sight and sound; behavioural anomalies, and in the dominant hemisphere, dysphasia. These symptoms are particularly prominent as a result of irritative lesions—that is, in temporal lobe epilepsy (p. 88). Destructive lesions cause less easily recognizable symptoms. An expanding lesion will often invade the optic radiation, causing an upper quadrantic field cut (Fig. 16).

Brain-stem

Three nuclear masses lie embedded in the white matter of the cerebral cortex at the upper end of the brain-stem. These are the thalamus, the basal ganglia and the hypothalamus (Fig. 10). They form the organizing centres of the sensory, motor and autonomic systems respectively. From here the afferent and efferent conducting pathways extend down through the mid-brain, pons and medulla oblongata to the spinal cord. The brain-stem also contains the nuclei of cranial nerves III to XII, the cerebellar pathways and the reticular formation. The reticular formation is a network of nuclei and connecting fibres, responsible for the motor and sensory relays, the control of the vital centres, and the mechanism for cortical arousal.

Thalamic lesions raise the threshold of peripheral sensation. However, once this new threshold is exceeded, painful stimuli provoke a diffuse, burning reaction. This is the **thalamic syndrome**. Disease of the basal ganglia causes rigidity, bradykinesia and involuntary movements. Lesions involving the hypothalamus cause disorders of thirst, appetite, growth and temperature regulation.

Familiarity with a simplified anatomy of the brain-stem is required for the localization of lesions at this site. The sections (Figs 3–6) show the relative positions of the main fibre tracts and nuclei. It should be noted that the structure of all four sections is basically the same if the areas below the broken lines are discounted.

1. *The medial longitudinal fasciculus* is constant in its position close to the mid-line below the floor of the aqueduct and the fourth ventricle.
2. The *spinothalamic tract* maintains its lateral position but moves dorsally in the higher sections.

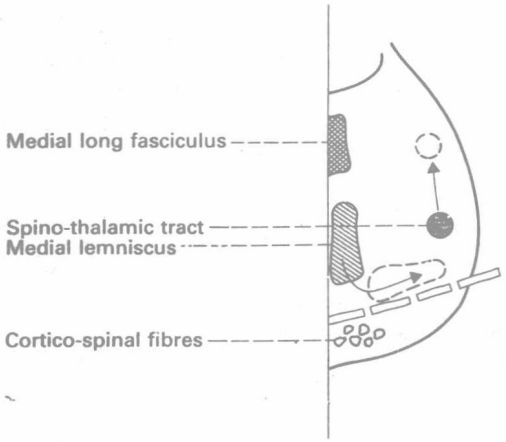

Fig. 2. Stylized section of the brain stem showing the location of important tracts and their relocation at higher levels.

3. As the *medial lemniscus* ascends through the brain-stem, it is displaced laterally from its original mid-line position in the lower medulla.

4. The *cranial nerve nuclei.* There are no simple rules which order the siting of the cranial nerve nuclei. The position of certain key nuclei is helpful in locating brain-stem lesions. The nuclei of the third and fourth nerves in the midbrain, the sixth nerve in the pons and the twelfth nerve in the medulla, lie in the grey matter beneath the floor of the aqueduct and the fourth ventricle.

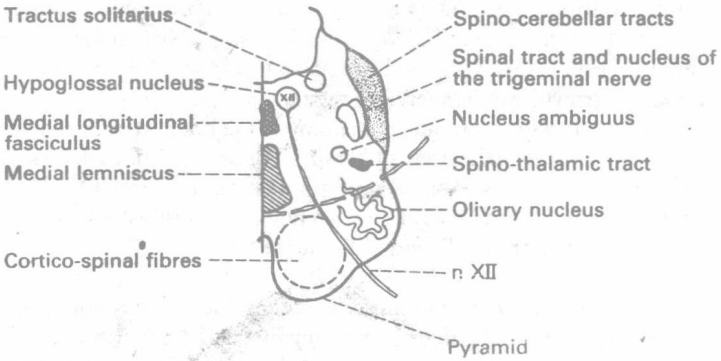

Fig. 3. Section through the medulla. After Buchanan.

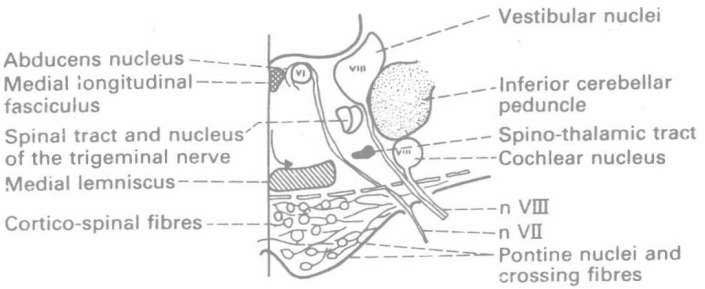

Fig. 4. Section through the lower pons. After Buchanan.

The nucleus ambiguus is an elongated motor nucleus serving the ninth, tenth and eleventh nerves. This, with the motor nuclei of the fifth and seventh nerves in the pons, form a broken column placed deeper and more laterally in the brain-stem.

The sensory nuclei of the fifth and eighth nerves occupy lateral positions in the pons. The spinal tract and nucleus of the fifth nerve extend caudally to the second cervical segment of the cord.

5. The areas below the broken lines contain the *descending cortico-spinal fibres.* In the midbrain they form large cohesive nerve trunks, the cerebral peduncles. These are the direct downward projections from the internal capsules. In the pons, some of these fibres synapse with the pontine nuclei and the second-order neurons stream across the mid-line to form the middle peduncles of the cerebellum. Thus the descending fibres are separated into small bundles by this transverse outflow. In the medulla, they regroup to form the pyramids and the

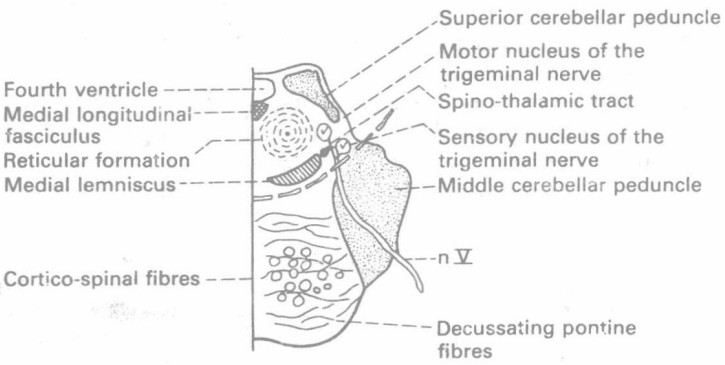

Fig. 5. Section through the mid-pons. After Buchanan.

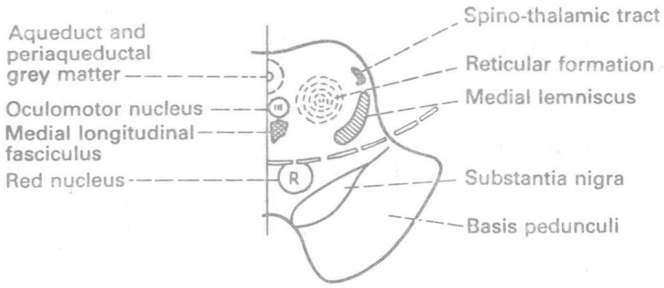

Fig. 6. Section through the mid brain. After Buchanan.

majority of fibres decussate where the brain-stem merges into the spinal cord.

Spinal Cord

The spinal cord consists of well-demarcated columns of motor and sensory cells (the grey matter) surrounded by the ascending and descending tracts (the white matter) (Fig. 7). It lies in the spinal canal and like the brain is surrounded by three fibrous membranes, the meninges. It is cushioned by the cerebrospinal fluid and held in place by the denticulate ligaments. Paired sensory and motor roots, corresponding to each segment of the cord, emerge from the canal by the intervertebral foramina. The cord segments are shorter than the corresponding vertebrae and the spinal cord terminates at the level of the first or second lumbar vertebra. The lowermost segments (sacral) are compressed into the last inch of the cord, known as the *conus medullaris*. The subarachnoid space extends beyond the end of the cord as far as the second sacral vertebra. This space is traversed by the remaining nerve roots, the *cauda equina*. A lumbar puncture needle may be introduced into the subarachnoid space, below the level of the second lumbar vertebra, without any danger of damaging the spinal cord.

Paraplegia

Paraplegia is a weakness or paralysis affecting both legs. Spastic paraplegia is most often due to disease of the spinal cord, but it may be caused by a tumour situated between the cerebral hemispheres, or thrombosis of the superior sagittal sinus.