THE DIAGNOSIS OF NERVOUS DISEASES

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NINTH EDITION

LONDON
EDWARD ARNOLD & CO.

First Edition	. . . . .		*1906*
Second	„		*1909*
Third	„		*1911*
Fourth	„		*1916*
Fifth	„		*1920*
Sixth	„		*1924*
Seventh	„		*1931*
Eighth	„		*1937*
Ninth	„ *1945*	*Reprinted*	*1947*

This book is produced
in complete conformity with the
authorized economy standards

Printed in Great Britain by
Butler & Tanner Ltd., Frome and London

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PREFACE TO THE NINTH EDITION

THE present book differs widely (I hope for the better) from its predecessors. Since the original version made its first appearance, nearly forty years ago in 1906, it has survived numerous revisions, mainly owing to repeated rejuvenations, notwithstanding the fact that with the passage of the years I have found myself transformed from a junior to a senior clinician and teacher. Progressive advances in neurology have been reflected in successive editions. These have tried to portray the current viewpoints of physicians in the modern scientific world and to help the physician and student to cope with the difficulties of clinical neurology. Some of its basic maxims have stood the test of time and experience; whilst other concepts, which a generation ago seemed established so securely as to be almost axiomatic, have been modified or superseded, not because they are old but because they are outworn and no longer helpful. New observations and new angles of approach have been added. Many problems, especially of etiology and treatment, still await solution. These, I doubt not, will in due time be solved at the hands of younger and better men, whom we elders recognize and salute with wholehearted admiration. Discriminating readers will readily detect numerous faults in the present work. At the same time they will appreciate some of the difficulties in the description of a constantly moving picture.

This work, besides serving as a clinical guide, although not a systematic text-book, describes some of the practical methods of approach employed by a clinician like myself in the examination of neurological patients. A study of each patient's personality and temperament should always be added, from which much information may be obtained, often of fundamental importance, particularly in estimating how much weight is to be attached to his subjective complaints.

Incidentally, this volume may have a personal interest, as forming a casual link between the great leaders of a former generation and their successors. I still recall, with reverence and affection, many of those great teachers, now passed on, at whose feet in the old days I was privileged to sit. They included such men as Hughlings Jackson, Gowers, Ferrier, Horsley, Bramwell, and Head in England; Weir Mitchell, Mills, Dercum, and Cushing in America; Dejerine, Babinski, and Marie in France; Mendel and Oppenheim in Germany; Pavlov in Russia; Bianchi in Italy; and many others, whom I knew personally, and of whom we still speak with bated breath. By neurologists of the present generation these are recognized as pioneers who inspired their disciples with enthusiasm and industry. I doubt not that their present-day successors, from whom I continue to absorb fresh knowledge, will carry on the torch of science with undiminished

enthusiasm and success. I refrain, at the moment, from saluting them by name.

Every thoughtful individual during his life's work makes his contribution, great or small, to the sum of human knowledge, and then passes on.

Sometimes, the personal opinions here expressed by me are at variance with those of other observers. This is almost inevitable, when discussing disputed points. The reader will exercise his own judgment in accepting or discarding my views.

This work has been re-written amid the hurricanes and emotional strains of a savage world-war, during a time of mortal national peril, in which the very survival of European civilization has trembled in the balance. At one time it looked as if it would crash to disaster. In some countries scientific activities were deliberately stamped out, and scientific workers were ruthlessly massacred, a danger which Britain has fortunately escaped.

In common with millions of my fellow-citizens I have shared the hazards of modern warfare and have witnessed at close range tragic but inspiring deeds of heroism. The dramatic events and emotional shocks of the war years have interrupted the continuity and academic calm of scientific investigations, even in those few countries which have withstood the ravages, attempted slavery and subjugation by enemy invasion. Catastrophes on land, at sea, and in the air, have compelled attention to problems of mere survival, which in pre-war days would not have arisen. Incidentally, the casualties of war, both amongst combatants and civilians, have revealed to us physicians a number of new and interesting neurological syndromes, *e.g.* the crush syndrome and the transient blindness of some aviators.

Even this modest book has had its war adventures. In 1939, and again in 1943, I travelled to the United States of America to study American neurological methods. There I had the privilege of visiting some of their most progressive medical schools and teachers. I also collected a number of fresh references and new illustrations. These latter I had planned to incorporate in this book. Alas! they were destined never to appear. During the voyage back to England all my manuscripts, together with my personal belongings, were sunk in mid-Atlantic by enemy action. I myself, happening to travel in another ship, had a fortunate escape. As a result, I had to begin all over again and to re-write the book *de novo*, utilizing such memoranda and references as I already possessed in England. I was also deprived of skilled help which at one time I had hoped to secure in America. This, however, gives it a more definite personal British accent, which may have both advantages and disadvantages which my readers will estimate for themselves.

Returning to my native land, I found my old Lighthouse home on the Sussex cliffs reduced by shell-fire to a heap of rubble. I then became

a war refugee for over two years in Kilmarnock, a Scottish country town, remote from personal scientific contacts and from free access to medical libraries.

At this stage I had the good fortune to secure the collaboration of my friend Sir Harold Scott, K.C.M.G., the distinguished physician and literary authority. His erudition and wide scientific knowledge have proved invaluable, both in correcting the proofs and in eliminating many errors. For his generous help and kindly encouragement I cannot adequately express my thanks.

Two years' war delay in publication has been inevitable. I hope, however, that the result, probably my swan-song, may meet with the acceptance of my readers, whose kindly approval has encouraged me in former years.

45, Edwardes Square, London, W.8.
1945.

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CHAPTER I

PHYSIOLOGICAL ANATOMY

THE living body is an organized family of living cells. This family or cell-assembly is integrated and controlled so as to possess a corporate unity. Moreover, each component cell in the family has an individual composite structure and an individual separate cell life, integrated within itself. It influences, and in its turn is influenced by, the other cells with which it is in contact.

The human body with its linked-up mechanisms—nervous, circulatory, and glandular—is fundamentally a neuro-sensitive apparatus in which the nervous system plays the dominant rôle. The function of the nervous system is to direct, control and integrate the activities of the individual and adapt him to the world in which he lives. Its activities occur in response to stimuli, external and internal. Some of these stimuli, and the responses which they evoke, give rise to conscious impressions; others elicit responses which do not rise above the threshold of our conscious attention.

The nervous system consists of two inter-communicating divisions:—

(1) The central, **cerebro-spinal** or **somatic** mechanism, comprising the brain and spinal cord, together with the cranial and spinal nerves.

(2) The **vegetative** or **splanchnic** mechanism, constituted by two chains of prevertebral ganglia, one on each side of the spine, together with other ganglia, situated peripherally. There are also central vegetative nuclei, situated within the cerebro-spinal axis.

The central nervous system is made up of three chief classes of cells. These differ not only in their function but also in their pathological reactions.

(1) **Neurones, neurocytes,** or nervous units; these constitute the master tissue. This is interspersed with interstitial cells of two kinds, *viz.*:—

(2) **Neuroglia** cells and fibres, ectodermal in origin, and

(3) **Microglia,** developed from the mesoderm.

Neuroglia cells are of two chief types, *viz.*—

(*a*) **Astrocytes,** with long stellate branches. One or more processes of the

astrocyte is attached to a blood-vessel, constituting a perivascular, pial, or " sucker " foot (Fig. 1). The network of astrocytes forms the normal supporting *vaso-astral framework*, binding together the blood-vessels in the brain. Proliferated astrocytes, with long fibrous processes, make up the secondary sclerotic

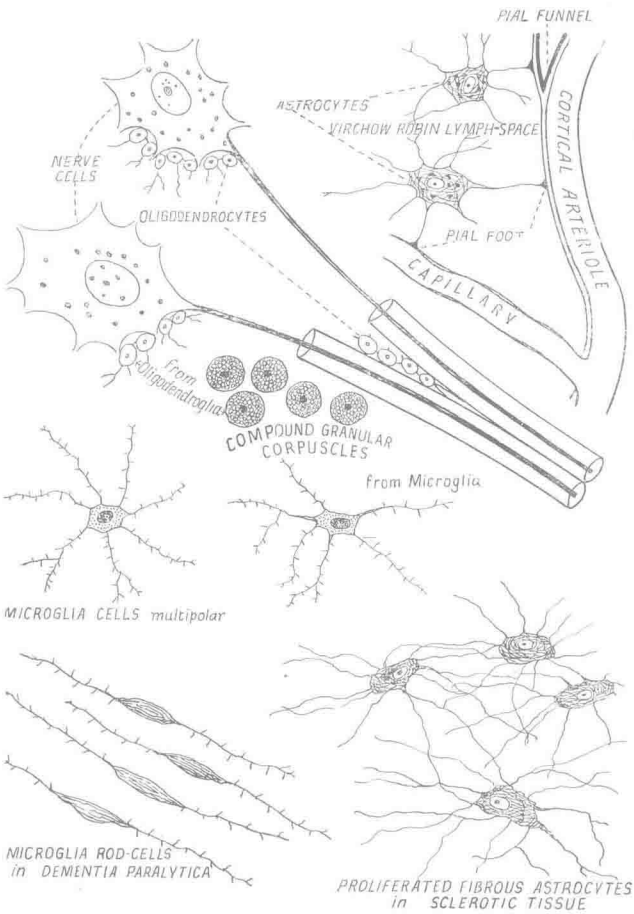

FIG. 1.—Diagram of Neuroglia and Microglia.

tissue which comes to replace degenerated nerve-fibres and nerve-tracts. They also constitute the scar-tissue during repair of a wound within the central nervous system.

(*b*) The **oligodendrocyte** is a much smaller cell, round or oval, without glia fibres, and, in particular, possessing no perivascular foot. The oligodendrocytes are normally arranged in rows, some between the myelinated nerve-fibres, others in satellite groups around the neurone bodies.

(*c*) **Microglia cells**, so-called Hortega cells,[1] are developed from the meso-

[1] Hortega, P. del Rio, *Lancet*, 1939, May 6, p. 1023.

derm. These multipolar cells resemble astrocytes in possessing complicated expansions, but differ from them in having no perivascular foot and in having terminal spines on their expansions, set at right angles to the main branches. These cells are commonest in the grey matter, where they constitute some of the perineural satellite cells. Their chief function, in conjunction with the oligodendrocytes, is the phagocytosis of waste products.

When a trauma is inflicted on the central nervous system, both the microglia cells and the oligodendrocytes rapidly transform themselves into "compound granular corpuscles," which undergo amœboid changes, and carry off fat globules and other products of degeneration towards the capillaries. They participate in all the inflammatory, degenerative and necrotic processes of the central nervous system.

In dementia paralytica the proliferated microglia cells are arranged in characteristic columns of rod-shaped cells perpendicular to the surface of the cortex and containing iron pigment.

In addition there are the **sheathing tissues** of mesodermal origin, comprising the **dura** and **pia-arachnoid.** From the latter the cerebral blood-vessels and lymph-spaces penetrate the brain and spinal cord.

Neurones.

The nervous system is built up of separate units or neurones. Each neurone is an embryological and trophic unit. It is constituted by a mother-cell, the *nerve-cell*, with its processes, the *axon* and the *dendrons*. All of these are excitable. It was formerly assumed that the nerve-cell or centron originated impulses, as does the cell of an electric battery, and that the nerve-fibres, axon and dendrons, served merely as conductors. But it is now recognized that an impulse within a nerve-cell arises as the result of a transmitted stimulus from another neurone.

To this rule the cardiac and respiratory nuclei within the medulla are notable exceptions. These vegetative nerve-centres are stimulated not only by transmitted stimuli from other neurones but also by chemical changes in the blood, *e.g.* by deficiency of oxygen and by excess of carbonic acid or of lactic acid.

Each nerve-fibre is made up of a bundle of fine neuro-fibrils. These traverse the nerve-cell, entering it through one process and leaving it through another, skirting past the nucleus. In this way the nerve-cell acts as a convenient shunt for stimuli, receiving them from one or more quarters and transmitting them to another. Nerve-impulses pass through the nerve-cell in one direction only, this direction being centripetal for the dendrons and centrifugal for the axon. The neurone has one end which is receptive in function and another end through which it discharges its impulse. This is the so-called "polarity" of the neurone.

The fibrils of one nerve-cell never *unite* with those of another. The axon and dendrons of each neurone terminate in end-tufts of various shapes. The free extremities of each tuft form a cluster of tiny bulbs which are in constant motion on their stems. They articulate with the corresponding terminals of an adjacent cell at a "*synapse*," across which a stimulus can

pass from one neurone to another, thereby slowing down the speed of conduction. Sometimes the end-tufts of the axon of one cell come into approximation with the dendron of another : sometimes the end-tuft of the axon terminates around the body of the second cell. But in neither case is there any fusion or continuity of structure between the two neurones, which remain independent morphological units.

The transmission of an impulse, whether from one neurone to another, or from a nerve-fibre to a muscle-fibre, or to a secretory cell, is achieved, not by the conduction of the impulse directly from neurone to neurone, or from nerve-fibre to muscle-fibre, as was formerly thought, but by the liberation at the synapse (whether inter-neuronal or neuro-muscular) of a specific chemical substance, acetyl-cholin, which in its turn is quickly hydrolysed and destroyed by the action of an enzyme, cholin-esterase. In the corresponding muscle-fibre there occurs only a single immediate twitch, in response to the acetyl-cholin liberated at the neuro-muscular junction.

The nerve-cell also exerts a trophic influence over the nerve-fibre and is intimately concerned with its nutrition, so that the nerve-fibre, if separated from its trophic nerve-cell, undergoes degeneration.

Although the neurone is a vital or trophic unit, it is not the functional unit of the nervous system. The real functional unit is the reflex or sensori-motor arc, through which a nervous impulse is conducted from sense-organ to muscle.

A **reflex** is the effective response of a living organism to an external or internal stimulus, independent of consciousness and volition. The term stimulus, in its medical sense, denotes any sudden change in the environment or in the physical conditions of the living organism which causes it to react.

A reflex motor act is the simplest manifestation of nervous energy. It is unlearned, being based on inborn neural mechanisms. Each reflex action serves some adaptive or protective purpose. For example in the plantar reflex the impulse or stimulus starts from a *sensory receptor* or end-organ, in this instance the skin of the sole. The impulse, which must be of adequate intensity and duration, is transmitted inwards by a conductor, the afferent or sensory nerve-fibre, through the corresponding posterior nerve-root into the spinal cord (Fig. 2). Here it traverses an intermediary nerve-fibre and cell in the grey matter of the cord and reaches an adjustor or correlation centre in the anterior cornu.

From the motor cell an efferent impulse starts, travels outwards along a second or efferent conductor, traversing, first, an anterior nerve-root and then a peripheral nerve, finally reaching the *effector apparatus*, which in this instance is the flexor muscle of the toes innervated by the corresponding segment of the spinal cord. The toe-flexors promptly give a single contraction.

The accompanying diagram (Fig. 2) will serve to recall the chief components of a simple spinal reflex, such as we have just described.

It must be remembered, however, that the skeletal muscles, as we shall presently see, have a double afferent innervation and control. Firstly, they are influenced by *exteroceptive impulses* from sense-organs on the surface of the body, conducted along medullated fibres in the sensory roots to the spinal cord; and secondly by *proprioceptive impulses* initiated in sense-organs embedded in the muscles, tendons, and joints, conducted along non-medullated nerve-fibres. This second group of sense-organs is stimulated by the contraction of the muscles themselves, by the stretching of muscles and tendons, and by alterations of posture in the joints. These proprioceptive stimuli co-operate with the exteroceptive in regulating the sequence and force of muscular contractions.

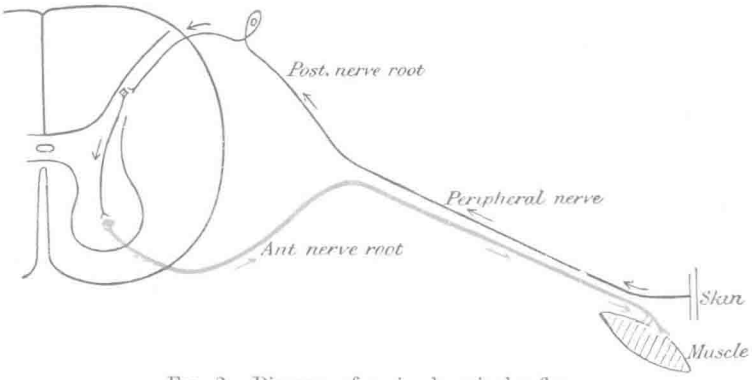

FIG. 2.—Diagram of a simple spinal reflex.

Some reflexes occur unconsciously, as for example the reflex contraction of the pupil when the retina is stimulated by light; or again, the normal movements of the stomach and intestines. In other cases the afferent impulse, besides exciting a reflex motor response, sends part of its impulse upwards to the higher centres of the contra-lateral cerebral hemisphere. It may either reach the thalamus, where it arouses a general sensation of awareness, or of heat or cold with their associated feeling-tone of comfort or discomfort; or the impulse may pass farther up, to the sensory cortex, where it produces a conscious discriminative sensation. This is accomplished by means of a sensory path passing upwards in the substance of the spinal cord through relays of neurones in the medulla, pons, thalamus, and so on, up to the perceiving area in the cortex (Fig. 18).

So long as the reflex arc (dominated in its turn by higher centres) is intact, the corresponding muscle-fibre, innervated by the anterior cornual cell, is maintained in a continuous slightly braced-up condition called **reflex tonus**. This muscular tonus is the basis of all postural reactions. It is also essential for the eliciting of the so-called tendon reflexes, *e.g.* the knee-jerk or ankle-jerk. The eliciting of tendon or deep reflexes is,

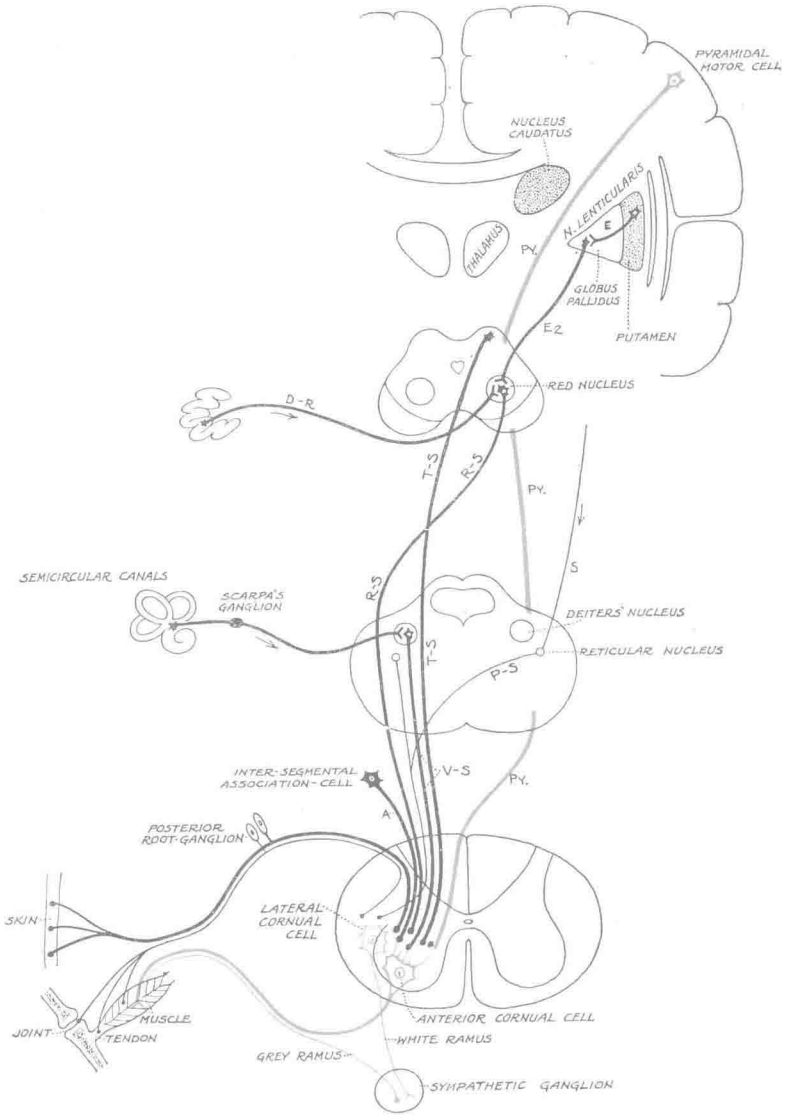

FIG. 3.—Diagram of some of the Impulses controlling the Anterior Cornual Cell.

A = Inter-segmental reflex control.
R–S = Rubro-spinal. Synergic control.
T–S = Tecto-spinal. Reflex visuo-motor control.
V–S = Vestibulo-spinal. Equilibratory control and muscle tonus.
P–S = Ponto-spinal. Muscle tonus.
D–R = Dentato-rubral path.
E = Automatic associated movements.
E_2 = Striato-rubral path.
S = Striato-reticular path, inhibiting muscle tonus.
Py = Pyramidal voluntary motor path.

therefore, as we find in clinical work, a useful index for determining the integrity of the corresponding reflex arc.

But discharges of motor energy from the motor cell in the anterior cornu can be produced not merely reflexly from the periphery, but also by the activity of a whole series of centres within the brain-stem (Fig. 3). And whilst the anterior cornual cell may receive impulses coming from any or many different receptors, it discharges its impulses into its motor unit through one "*final common path*," *viz.* the anterior-root fibre. The individual muscle responds inevitably to the nerve-impulses reaching it in this final common path, irrespective of whether these be part of a spinal, brain-stem, or cerebellar reflex, or of a voluntary impulse along the pyramidal path from the cerebral cortex.

Firstly, a motor act may require for its performance the activity of motor units, not in a single muscle, but in a whole group of simultaneously-acting muscles, innervated by neighbouring segments of the spinal cord. This **inter-segmental reflex activity** is made possible by means of inter-segmental association-cells, which may be situated either on the same side of the spinal cord, or, where bilateral muscular activity is required, by means of trans-segmental association-cells on the opposite side.

Further, the activity of the anterior cornual cell is controlled by impulses reaching it from more distant sources higher up. Impulses of muscular tonus and of equilibrium are transmitted from Deiters' nucleus, along the vestibulo-spinal tract (Fig. 3). Deiters' nucleus in turn receives impulses from the semicircular canals, the otoliths, and vestibular nerve of the inner ear. Therefore in lesions of the vestibular nerve or inner ear, or of the vestibulo-spinal tract, equilibrium is profoundly disturbed and the patient has difficulty in maintaining his balance when walking, standing or sitting. (The ganglion on the vestibular nerve, known as Scarpa's ganglion, situated at the outer end of the internal auditory meatus, is analogous to the posterior-root ganglion of a spinal nerve.) There is thus an **equilibratory control** of the anterior cornual cells.

For every activity of the skeletal muscles in moving a joint, the muscles are arranged into *agonists* (or prime-movers) and *antagonists*, the one set opposing and regulating the other. Thus, for example, when we flex our elbow by means of the biceps muscle, the antagonistic triceps relaxes proportionately at the same time, so as to determine the degree and the rate of the flexion movement. The biceps and triceps thus constitute a synergic unit. During voluntary contraction of a prime-moving muscle against resistance, the contraction of its antagonist becomes correspondingly inhibited, in accordance with Sherrington's "*law of reciprocal innervation*." Thus, for example, if an individual depresses his arm at the shoulder slowly, without resistance, the antagonistic deltoid muscle can be felt to be actively contracting all the time. If he now attempts to depress his arm from the horizontal position against an insuperable obstacle which is resisting upwards against the lower end of the humerus, thereby performing a maximal voluntary effort with his prime-moving depressor muscles, the antagonistic deltoid is at once felt to relax.

Synergia of the various motor units in the body is regulated by the activity of the red nucleus, which transmits impulses downwards to the anterior cornual cells *viâ* the *rubro-spinal tract*. Rademaker and Magnus[1] have shown that

[1] *Lancet*, Sept. 18, 1926, p. 587.

the red nucleus is the reflex centre for the execution of "*labyrinthine righting reflexes*" (p. 198) and of the *postural reflexes* excited by contact-pressure of the body with the ground (p. 199). The red nucleus in turn is influenced by impulses from the dentate nucleus of the cerebellum. If, therefore, the dentato-rubral mechanism (*i.e.* the cerebellum or the red nucleus with its descending rubro-spinal tract) be damaged, **synergic control** is lost and inco-ordination or ataxia of the corresponding synergic units is produced (Fig. 3).

There are also certain automatic actions, performed without conscious volition, such as the normal swing of the arms during walking. These are more complex than simple reflexes but more primitive than voluntary movements. They are initiated by the activity of the cells of the corpus striatum which send impulses down through the red nucleus to the anterior cornual cells and constitute a striato-rubro-spinal control over the anterior cornual cells for the performance of **automatic associated actions**.

Other reflex actions, with a long path, result from *visual* impressions, *e.g.* the protective blinking of the eyelids to a sudden flash of light, sometimes accompanied by a sudden movement of the head and upper limb so as to shield the eyes from the violent visual stimulus; or again the normal reflex turning of the face towards an object on which visual attention is directed. These **reflex visuo-motor movements** are carried out largely by the influence of the superior corpus quadrigeminum, which sends the *tecto-spinal tract* downwards to the anterior cornual cells of the limbs and head, and to their homologous cells in the oculo-motor nucleus.[1]

Lastly, and most important of all, we have the dominant path of the *pyramidal or cortico-spinal tract*. This carries purposive or **voluntary impulses** from the motor cortex to the anterior cornual cells. These are phasic, contrasting with the continuous, postural, or tonic impulses conveyed by the vestibulo-spinal and other infra-cortical systems. In addition to voluntary motor impulses, the pyramidal tract normally exercises an inhibitory effect upon reflex muscle-tonus, so that in pyramidal lesions the infra-cortical mechanisms become uncontrolled, and we have, not merely loss of voluntary motor power in the paralysed muscles, but also increased reflex tonus, accompanied by exaggeration of the corresponding tendon reflexes.

Lesions of the pyramidal tract are the only ones which produce exaggeration of deep reflexes. Corpus striatum lesions, as we shall see, may cause increased muscle-tonus, but they do not alter the deep reflexes.

Muscular tonus, or postural contraction, is a continuous involuntary braced-up condition of muscles for the maintenance of normal attitude. Clinically it is recognized as a slight but definite resistance to palpation and to passive stretching.

[1] The act of **blinking** is not a simple reflex and its movements are not dependent merely on the integrity of the optic, oculomotor, trigeminal, or facial nerve. The movements of blinking are centrally modified and dependent on intermittent impulses from the corpora striata. The rate of blinking is closely related to the emotional tension of the subject at the time, becoming more rapid when he is emotionally excited. Probably the blinking movements constitute a kind of relief mechanism (Ponder and Kennedy, *Quar. Jour. of Exp. Physiology*, 1927, vol. xviii., p. 89).

Blinking is a reflex mechanism for protecting the eyes from dazzle or against corneal irritants. It also shuts off macular vision temporarily and induces a change in the direction of fixation. It sometimes gives the individual a warning from extra-macular areas of the retina, helping him to recognize with the "tail of his eye" some approaching danger. (Hall, Sir A., *Brit. J. Ophth.*, 1945, p. 445).

Muscular tonus is a reflex reaction, dependent on the integrity of a whole series of proprioceptive reflexes, some of them muscular, some of them vestibular in origin. The sense-organs of these reflexes are situated mainly in the muscles, tendons, and joints. The effective stimulus is a stretching of the muscle, *e.g.* pressure by the sole of the foot on the ground in the standing posture. Other sense-organs are situated in the otolithic mechanism of the labyrinths.

The afferent impulses from the muscles ascend in the antero-lateral column (spino-cerebellar tract) to the pons, where the reflex centres are situated, in Deiters' nucleus and the reticular nucleus. From the labyrinth the afferent impulses reach Deiters' nucleus, *viâ* the vestibular nerve.

The main efferent paths for muscular tonus descend in the spinal cord along extra-pyramidal paths (Fig. 3) *viz.* the vestibulo-spinal tract and the ponto-spinal tract, to the anterior horn cells. There the extra-pyramidal tonic impulse is relayed through the anterior root to reach the peripheral motor nerve and the corresponding voluntary muscle. The function of these impulses is tonic and myostatic, maintaining any posture in which the limb is placed.

The postures thus established by tonic contraction are maintained, sometimes for long periods, with little or no fatigue, as in ordinary standing or sitting.

The tonus of a smooth muscle is intrinsic and persists even when the muscle is isolated from the nervous system. Striated muscle, on the other hand, shows no such intrinsic tonus, its tonus being regulated, as Sherrington [1] and others have shown, by the above-mentioned finely graded series of proprioceptive muscular or vestibular reflexes, conducted along extra-pyramidal paths.[2]

Every accurate movement starts from a definite posture and ends in a posture. More than this, reflex posture accompanies every movement "like a shadow." Muscle-tonus fuses with other varieties of muscle-contraction, whether voluntary or reflex, steadying them and maintaining their contraction both during the stimulus and after its cessation.

This **duality of pathways,** subserving the functions of active movement on the one hand, and of posture on the other, applies throughout the whole efferent nervous system. We have seen how there is a **kinetic** mechanism for active movements and a **static** mechanism for the maintenance of posture. In health these two mechanisms function together in harmony. In disorders of motor function both systems participate, although we can sometimes recognize one or the other as primarily at fault. Thus, for example, involuntary movements such as those of chorea, myoclonus, tremors, and convulsive seizures are hyperkinetic disorders; whereas myotonia and catalepsy on the one hand, with their excessive tonus, and cerebellar or tabetic hypotonia on the other, with their loss of tonus, are disorders of the static or posturing mechanism.

The same two efferent mechanisms, kinetic and static, are also present at a higher level in the psycho-motor sphere of the cerebral cortex, *i.e.* that part which has to do with the performance of purposive psycho-motor acts and postures. Loss of the power of performing purposive psycho-motor acts, notwith-

[1] Sherrington, Sir Charles, *Brain*, 1915, vol. xxxviii., p. 191.

[2] The theory of Hunter and Elliot Smith (*Brit. Med. Journ.*, 1931, Jan. 31 and Feb. 25), that the efferent fibres of the sympathetic chain exercise a controlling influence on muscle tonus, has now been abandoned by most physiologists.

Wilkinson (*J. of Compar. Neurol.*, 1934, vol. lix., p. 221) and others maintain that sympathetic nerve-fibres are distributed only to the blood-vessels and not to the striated muscle-fibres, which are innervated solely by somatic nerves.