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(1) the motor functions,
(2) the sensory functions, and
(3) the reflexes.

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NEUROLOGICAL EXAMINATION

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MOTOR FUNCTIONS IN LIMBS AND TRUNK

The first approach should always be by inspection. This will reveal any wasting of muscles, any involuntary movements, or any abnormal posture of the limbs, and will also enable any trophic changes to be observed. Wasting is a feature of lower motor neurone damage and may be accompanied by fasciculation, that is to say the twitching of groups of muscle fibres comprising an individual motor unit. Fasciculation may occur when there is incipient damage or degeneration of the anterior horn cell and the proximal part of the axone. Involuntary movements which, as the name suggests, are not under the control of the patient, may be of various types, the commonest being tremor. This may be the result of extra-pyramidal disease such as Parkinsonism or may be an exaggeration of physiological tremor as in anxiety neurosis, and thyrotoxicosis and other toxic states. The movements of chorea, which are semi-purposive, and the repetitive writhing movements of athetosis may also be seen, either combined (choreo-athetosis) or in pure culture. Abnormalities of posture of the limbs may reveal underlying disease of bone or joints or contractures of disused muscles, but they may also be characteristic of damage to various parts of the nervous system, as when the increased activity of the anti-gravity muscles produces the typical flexed posture of the upper limb in hemiplegia. After inspection the examination of the motor functions proceeds with the assessment of tone, power, co-ordination and maintenance of posture, and gait and these aspects will now be considered in turn.

ASSESSMENT OF TONE

This can only be achieved with considerable experience since it depends on two variables : (1) the patient's ability to relax, and (2) the observer's ability to assess objectively the degree of resistance offered to his movements of the patient's limbs. Usually tone is assessed in the limb muscles by passive movements of the wrist, elbow and shoulder, ankle, knee and hip. In addition in the lower limbs clonus may be tested for, at the knee by

extending the joint and pushing the patella sharply towards the anterior tibial tubercle, and at the ankle by forcibly dorsiflexing the foot. The repetitive contraction and relaxation of the muscles which constitutes clonus is usually the sign of an upper motor neurone lesion. Tone may be pathologically increased, as in spasticity due to an upper motor neurone lesion or in rigidity due to an extrapyramidal lesion. Spasticity is recognised by its "clasp-knife" character which describes the manner in which resistance to passive stretching of the muscle by moving a joint is maximum during the first part of the attempted movement and then breaks down suddenly, like the opening of a pen-knife blade. In contrast rigidity causes a uniform degree of resistance to muscle stretching throughout the range of movement of the joint. Tone may also be pathologically decreased, as in the hypotonia of a lower motor neurone lesion or of cerebellar hemisphere lesions.

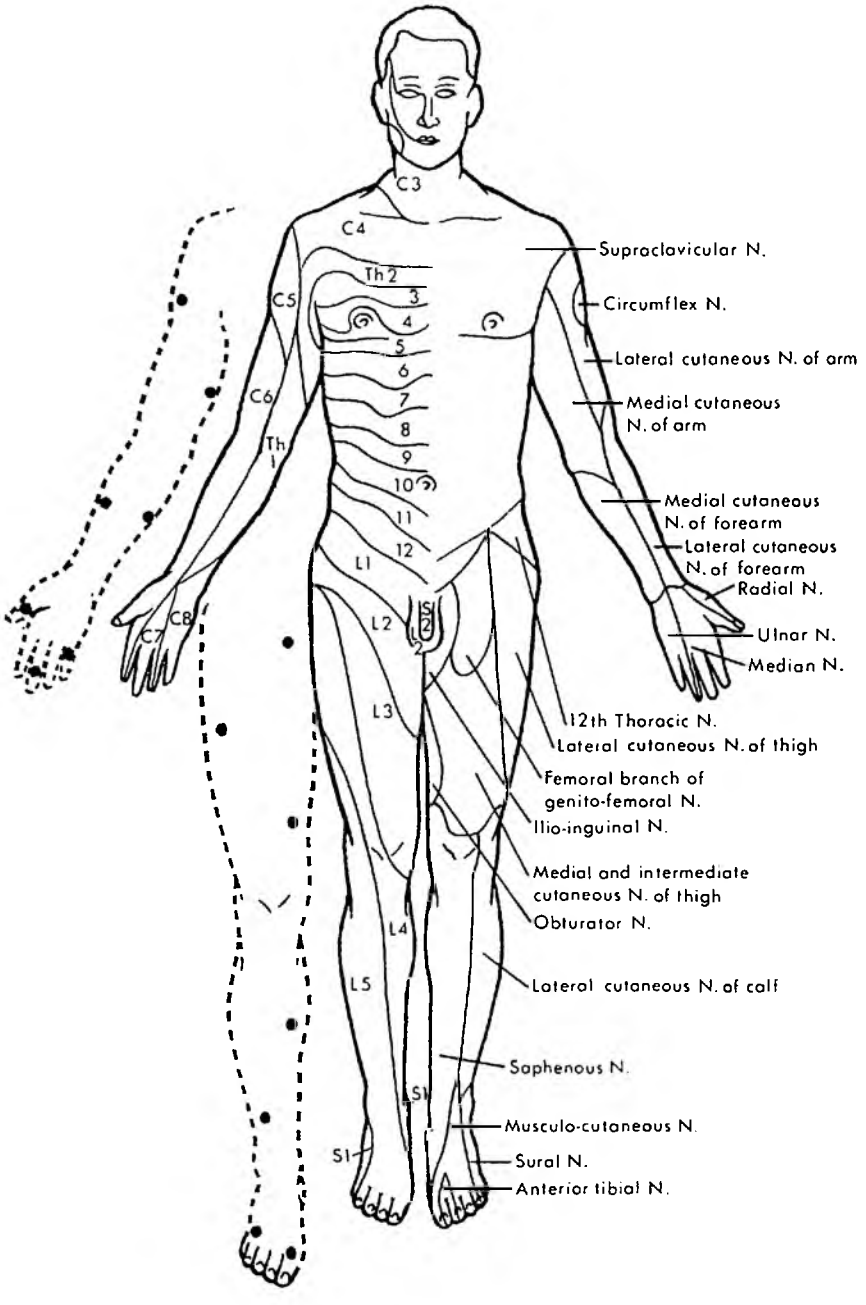
POWER

Muscular weakness is one of the cardinal signs of neurological disease and may result from disease of the muscle itself, as in myopathy, or from disease of the motor nerve, as in peripheral nerve and limb plexus lesions, or from disease of the anterior horn cell in the grey matter of the spinal cord. Weakness of voluntary movement will also result from interruption of the pathways (pyramidal tracts) between the motor cortex and the anterior horn cells or motor cranial nerve nuclei. Paralysis may also of course be a symptom of hysteria. When testing a patient's power it is usual to estimate the force which the patient can apply in performing a movement requested by the examiner. For instance the patient may be asked to flex his elbow while the examiner, grasping his wrist, tries to prevent the movement. Routinely, power in the upper limb is tested by assessing the strength of the hand grip, of dorsiflexion and plantar flexion of the wrist, of flexion and extension of the elbow, and of abduction and adduction at the shoulder. In the lower limb, dorsiflextion and plantar flexion of the foot, flexion and extension of the knee, and flexion, abduction and adduction of the hip are tested. If any weakness is found during the course of this routine survey then the power of individual muscles will have to be assessed in the light of the knowledge of their anatomical actions. When there is some doubt as to whether the patient is exerting full power in performing the movements asked of him, it is sometimes useful to change the procedure and ask the patient to keep the joint fixed in a certain position while the examiner attempts to move it. If in this situation the strength of the muscle appears to be good, whereas in voluntary movement it appears poor, then this may well be due to lack of co-operation by the patient or to hysteria rather than to true organic weakness.

CO-ORDINATION AND MAINTENANCE OF POSTURE

Co-ordination may be impaired by muscular weakness alone, but when it is disturbed in the absence of such weakness it is usually the result of damage to the cerebellar mechanisms (*i.e.* to the spino-cerebellar pathways, to the cerebellum itself, or to its connections with the motor cortex and basal ganglia), or to loss of proprioceptive sensation from muscles, joints and tendons. This latter form of incoordination is usually known as sensory ataxia and the former as cerebellar ataxia. Disturbance of co-ordination is often associated with an inability to hold the limbs in a steady posture. In the first place therefore the patient should be asked to stretch out his arms and to keep them steady in front of him. A similar test in the lower limbs is carried out on each side in turn by asking the patient to hold the leg at an angle of 45 degrees to the horizontal while lying on his back. In a patient with cerebellar disease, the limb on the side corresponding to the cerebellar lesion will often be found to waver whether the eyes are open or shut. On the other hand the patient with sensory ataxia will be able to maintain the posture of his limbs normally with the eyes open, but when he closes his eyes the posture of the affected limb will become unstable. This is because the visual sense can compensate to a considerable degree for loss of proprioceptive information from a limb. Co-ordination is also tested in the upper limb by the finger/nose test in which the patient is asked to place the tip of his index finger on the tip of his nose. This again should be performed first with the eyes open and then with the eyes closed to distinguish between cerebellar and sensory ataxia (see above). The corresponding test in the lower limb is the heel/knee test in which the patient, lying supine, is asked to place the heel of one foot on the knee of the other leg and to run it

Fig. 1



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steadily down the front of the shin bone. In either of these tests inco-ordination is revealed by oscillation of the moving limb in place of the normal smooth, steady movement. In particular an oscillation seen mainly at the end of the action, when the target (nose or knee) has almost been reached, is called intention tremor and is a sign of cerebellar ataxia. Rapid alternating movements are also impaired by inco-ordination and can be tested by asking the patient rapidly to pronate and supinate the forearms or, while lying on his back, to tap with the heel of one foot on the middle of the opposite shin.

GAIT

The examination of the gait and stance is an important part of testing the motor functions. The patient is asked to walk up and down and any abnormality is noted. Abnormalities may be the result of unilateral disability, as for instance in the gait of hemiplegia, or of bilateral disability, as in the slow stiff gait of a patient with spastic paraplegia. The disturbance may be less one of spasticity and weakness that of ataxia in which case the patient will walk with a broad wide-based gait, his feet held wide apart in an attempt better to maintain his balance. The short shuffling steps of Parkinsonism, and the loud slapping of the foot on the ground in a patient with paralytic footdrop, are characteristic disturbances which may be recognised. After his gait has been observed the patient should be asked to stand with his feet together and to maintain his balance which he should normally be able to do. He is then asked to close his eyes and should still be able to maintain his balance, unless he has a sensory ataxia of the lower limbs in which case he will sway or fall. This is Romberg's test.

THE REFLEXES

Routine examination of the nervour system includes the eliciting of the tendon reflexes of the jaw and limbs, and the abdominal and plantar responses.

The tendon reflexes are stretch reflexes and are elicited by a sudden stretch of the muscle usually brought about by striking the appropriate tendon with a sharp blow from a tendon hammer. The tendon reflexes are diminished or abolished by any lesion which interrupts the segmental reflex arc, and they are enhanced when damage to the cortico-spinal pathways releases the segmental reflex arc from inhibitory influences from higher levels of the nervous system.

The secret of eliciting the tendon reflexes efficiently is to place the relevant part of the body in the correct position. This should be such as to ensure that the length of the muscle involved is roughly midway between full shortening and full lengthening. The jaw jerk is elicited by grasping the patient's chin between the forefinger below and the thumb above. The patient is instructed to relax and to allow his jaw to hang half-open and the examiner's thumb is then struck a glancing blow with the tendon hammer, thus causing a sudden stretch of the masseter and temporalis muscles which gives rise to a reflex contraction. The jaw jerk is normally rather sluggish, but is clearly exaggerated in the presence of a bilateral upper motor neurone lesion above the level of the motor nuclei of the trigeminal nerves in the pons. In the upper limbs, the biceps jerk (segmental level C5, 6) and the brachioradialis or supinator jerk (C5, 6) are both tested with the patient lying supine and the arms slightly abducted at the shoulder with the hands resting on the abdomen while the elbows are half-flexed. The examiner's thumb or finger is placed firmly on the tendon of the biceps muscle immediately above the cubital fossa and is struck with the hammer. The supinator jerk is produced by striking the head of the radius at the wrist. To elicit the triceps jerk (C6, 7), the forearm of each side in turn is pulled across the patient's chest and the triceps tendon struck directly above the olecranon process. It is convenient to compare the reflexes of the two sides directly by eliciting each reflex first on one side and then on the other. In the lower limbs, the knee (L2, 3, 4) and ankle jerks (L5, S1) are tested. With the patient lying flat on his back the knees and hips are semi-flexed and the examiner passes his arm below the knees to support their weight and allow the patient to relax. The patella tendon is then struck on each side in turn. To elicit the ankle jerk, the hip is abducted and externally rotated with the knee flexed. The foot is dorsiflexed by the examiner's hand pushing against the ball of the foot, while he strikes the Achilles tendon with the hammer. The abdominal responses (D8-12) are tested by slowly and *lightly* stroking the abdomen with a pin. The stroke should be towards the mid-

line, parallel to the costal margin for the upper quadrants and parallel to the inguinal ligament for the lower quadrants. The abdominal responses disappear in the presence of an upper motor neurone lesion. The plantar response (L_5, S_1, 2) is elicited by scratching the sole of the foot with a pointed (but not sharpened) object, such as the end of a Yale key. The stimulus should be applied to the outer border of the sole, commencing near the heel and passing forwards to the base of the 5th toe and then turning medially across the ball of the foot towards the base of the great toe. This stimulus will produce reflex flexion of the great toe in normal persons, but in the presence of a lesion of the pyramidal tract the great toe will dorsiflex instead.

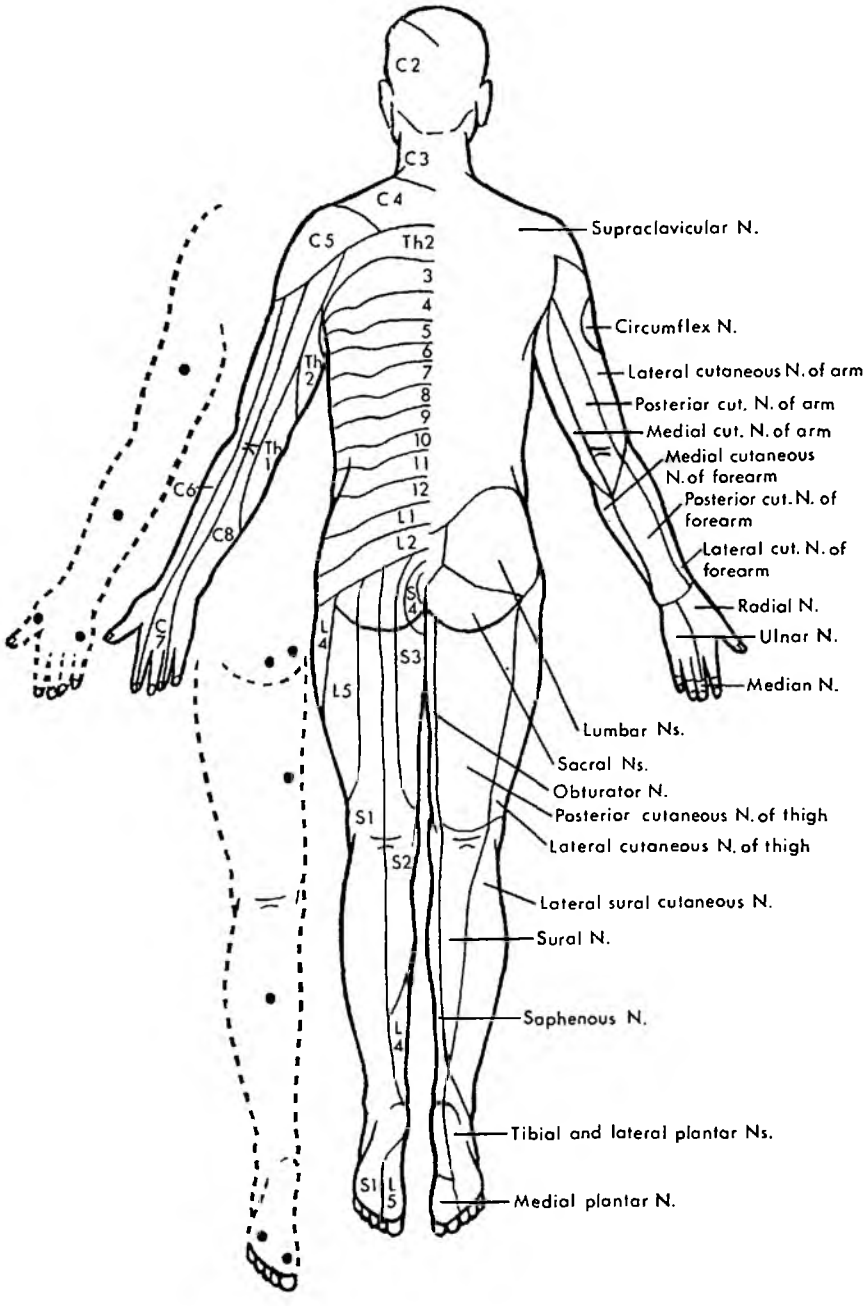
SENSORY FUNCTIONS

Sensory testing may be a fatiguing exercise both to the patient and for the examiner. If the patient is not in a clear mental state or if he becomes tired during the course of the examination then his answers will become inconsistent and worthless.

A number of different modalities or forms of sensation must be tested over the whole of the surface of the body during the examination. The different modalities of sensation include pain, temperature, light touch, vibration sense, and the sense of passive movement, and the student is less likely to omit testing one of these modalities if he adopts a systematic routine of his examination. It is helpful to remember that pain and temperature sensation travel together in the spino-thalmic tract of the spinal cord, and that light touch, vibration sense and proprioception travel in the dorsal columns. With this in mind it is logical to test the two spino-thalmic modalities, pain and temperature, one after another and then to continue with the modalities carried through the dorsal columns. Alternatively one may test superficial (cutaneous) sensation — pain, temperature and light touch — in one group, and deep sensation, including vibration sense and sense of passive movement, in another. Pain sensation is tested with a pinprick, temperature sensation with tubes filled with hot or cold water, and light touch with cotton wool. Vibration sense is tested by applying a tuning fork vibrating at 256 cycles per second to various bony prominences. Sense of passive movement is tested by moving the terminal phalanx of a digit in the hand or foot up or down and asking the patient to indicate the direction of movement while his eyes are closed. When there is no reason to suspect from the history that the patient has loss of cutaneous sensation, a fairly rapid survey of the limbs and trunk will serve to determine whether sensation is intact. Should an area of apparently altered sensation be found however, then it will be necessary to examine it more closely and to delimit its extent and outline. Cutaneous sensation may be impaired by lesions at any point between the sensory nerve endings in the skin and the sensory cortex, and the area and distribution of sensory loss on the skin will depend on the site of this lesion. Figures 1 and 2 indicate the peripheral nerve supply and segmental or dermatomal distributions on the skin of the trunk and limbs. By applying the stimuli for temperature, pain and touch to the points indicated by the black dots in the haloes to these figures, it will be seen that a fairly rapid assessment can be made of the integrity of sensation in both peripheral nerve and dermatomal territories of the limbs. Sensation on the front and back of the trunk should be tested from below upwards on each side of the midline. The most satisfactory procedure is to determine in the first place the integrity of cutaneous sensation all over the body. This should be done as quickly as possible so as not to fatigue the patient. If an area of apparent alteration of sensation is found the examiner should finish checking the sensation in other parts of the body before returning to a more detailed examination of the abnormal area. It may be wise to carry out this more detailed examination on another occasion, if the patient has become fatigued. When plotting the boundaries of such an area one should move the stimulus from the region of lessened sensitivity towards that of normal sensitivity, since the transition to normal sensation is most easily appreciated by the patient. It is noteworthy that when vibration sense and position sense are impaired they are almost invariably lost initially from the periphery, and for this reason if they are found to be intact at the distal phalanges then there is no need to test them more proximally.

Cortical sensory functions. Lesions of the sensory cortex in the post-central gyrus do not usually cause marked loss of sensation in the corresponding parts of the opposite side of the body, but impair the discriminative faculties in that part. For instance there may be inability to distinguish by palpation alone be-

Fig. 2



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tween the different shapes of objects held in the hand, or to discriminate between different textures, weights, etc. These discriminative functions are a property of the cortex, which extracts the necessary information from the basic sensory modalites and there are no separate 'discriminative' pathways. The integrity of the cortical sensory functions can only be tested profitably therefore if it has been shown that there is no impairment of sensation from interruption of the sensory pathways in the peripheral nerves, or in the spinal cord and brainstem. There are three simple tests of cortical sensory function : tactile localisation, two-point discrimination, and stereognosis. In testing tactile localisation the patient is asked to close his eyes and he is then lightly touched with a blunt object and asked to indicate the point on the body so touched. Two-point discrimination is the ability to distinguish between two separate points when applied close together. This can be tested by applying two pinheads simultaneously or with the aid of a specially designed pair of compasses. The threshold at which two points can be discriminated from one another on the pulp of the finger is less than 5 mm. in the normal. On the foot it is considerably greater and is about 3 cms. on the sole of the foot. Stereognosis implies the ability to recognise objects by palpation without looking at them. The patient is asked to close his eyes and various small objects, such as coins of different denominations, a safety pin, or a key, are placed in his hand and he is asked to identify them by palpation alone. Each hand is tested separately for accuracy in recognition.

Other higher cortical functions. In addition to cortical sensory functions, the functions of language, the ability to perform complicated voluntary movements, and the ability to recognise objects by sight or hearing may be tested. Impairment of these functions is called aphasia, apraxia or agnosia respectively. These and the intellectual functions in general are not tested routinely, but only in cases where cerebral damage is inferred and for the sake of brevity they are not considered in detail in this article. At this point however it may be emphasised again that the examination of the nervous system is only one part of the general medical examination and that a full medical examination must be carried out in each neurological patient.

INTERPRETATION OF THE PHYSICAL SIGNS

Reference has already been made in the first of these two articles to the importance of taking a full clinical history in neurological disorders, as in any other branch of medicine. The physical examination usually enables the physician to diagnose the site of the lesion in the nervous system — the "anatomical diagnosis". The final or "aetiological diagnosis" is arrived at by relating this anatomical diagnosis to the information contained in the history, which may indicate the nature of the pathological process which is at work, e.g. congenital, infective, traumatic, neoplastic, vascular, degenerative, etc. Often however it may be necessary to resort to further sources of information such as X-rays, examination of the cerebrospinal fluid, electroencephalography, cerebral angiography, etc., before a final diagnosis can be reached.

By eliciting the physical signs described in these two articles, the student will be able to reveal the defects in the patient's performance and, from his knowledge of the anatomy and physiology of the nervous system, he should then be able to determine at which point the nervous connections have been interrupted. For instance, the patient may have an impairment of his motor functions, whether in the motor cranial nerves, the limbs or the trunk. This may take the form of weakness, indicating either an upper or lower motor neurone lesion or primary muscle disorder. It may take the form of alteration of tone, such as the spasticity of an upper motor neurone lesion or the rigidity of basal ganglion disease, or it may take the form of ataxia and inco-ordination, indicating disease of the cerebellar or proprioceptive pathways. Various combinations of signs of motor dysfunction may indicate which pathways or groups of nerve cells are damaged. A combination of wasting, fasciculation, weakness and decreased tone in the muscles, together with loss of the corresponding tendon reflexes, are the signs of a *lower motor neurone lesion*, indicating damage somewhere between the anterior horn cell and the affected muscles. In contrast, weakness without wasting, but with increase of tone, exaggeration of tendon reflexes, absent abdominal repsonses, and an extensor plantar response on that side of the body, all indicate an *upper motor neurone lesion*. When the sensory functions are impaired, there may be

involvement of the special senses of smell, taste, vision and hearing, or of cutaneous and/or deep sensation. The impairment of both cutaneous and deep sensation in a relatively small area of the skin surface will suggest a peripheral sensory nerve lesion. The selective impairment of pain and temperature sensation with preservation of light touch, joint sense and vibration sense, indicates that the lesion exclusively involves the spino-thalamic tract. Conversely the dorsal columns may be involved alone, in which case light touch, joint position sense and vibration sense are impaired. Such selective disturbances can arise only from a lesion situated in a part of the nervous system where the spino-thalamic tracts and the dorsal column pathways are sufficiently separated in space for one to be involved without the other, i.e. in the spinal cord or lower brain stem. The finding of a "sensory level" on the trunk above which sensation is normal may reveal clearly in which segment of the spinal cord the lesion lies. Sometimes the anatomical diagnosis will not indicate a discrete lesion of a relatively small area of the nervous system, but will show that various widely separated cell groups and fibre pathways which nevertheless have close functional connections, are diffusely and selectively involved. For instance there may be evidence of combined upper motor neurone and lower motor neurone damage without any signs of sensory or other impairment. This situation occurs in amyotrophic lateral sclerosis, a form of motor neurone disease arising from widespread degeneration of the upper motor neurones (the Betz cells in the motor cortex), and of the lower motor neurones situated in the motor cranial nerve nuclei and in the anterior horns of the spinal cord.

From what has been said earlier it will be realised that the routine neurological examination which has been described in these two articles is the *minimum* which should be carried out and that the finding of abnormal signs will lead to more detailed attention being paid to the functions found to be impaired. With practice it is possible, while carrying out the examination to keep constantly in mind the various possible sites of damage in the nervous system that are suggested by the physical signs being elicited, and then to look for further signs which will confirm or refute these possibilities. In this way the clinical examination of the nervous system develops into a truly logical investigative procedure.