A Lecture

ON THE

EARLY SYMPTOMS AND DIAGNOSIS OF
DISEASES OF THE SPINAL CORD.DELIVERED BEFORE THE PLYMOUTH DIVISION OF THE BRITISH
MEDICAL ASSOCIATION, MAY 1ST, 1920,

BY

SIR F. W. MOTT, K.B.E., M.D., F.R.S.

WHEN the Plymouth Division of the British Medical Association did me the honour of asking me to give an address on the early symptoms and diagnosis of diseases of the spinal cord I readily accepted; but when I came to consider how I could best occupy the time permissible for such an address, I found considerable difficulty—not with what I *could* say on such a very important and extensive neurological subject, but what I could leave out. I will therefore be guided chiefly in the treatment of the subject by the difficulties I have experienced in differentiating early organic from functional disease, especially in the large number of cases which have passed through my hands during the last six years at the War Neurological Clearing Hospital in London.

The cases which presented the greatest difficulty were those in which the patients came under notice for subjective sensory phenomena, many of whom presented no objective signs.

SUBJECTIVE SENSORY SYMPTOMS.

The two subjective sensory symptoms which are the earliest evidence of disease of the spinal cord are pain and paraesthesia or dysaesthesia. They may be the sole symptoms, at first, of severe spinal cord disease, and it should be recognized that a patient who comes to a doctor complaining of localized pain or numbness, tingling, pins and needles, formication, or other abnormal sensations, presents the possibility of early signs of spinal cord disease.

Pain an Early Symptom of Spinal Cord Disease.

Pain is one of the most constant signs of all forms of disease; it is protective, and therefore constitutes an early symptom. When a patient comes complaining of pain in a part, the natural thing to do is to see whether there are any objective signs to account for the cause of the pain in the region where it appears. There may or there may not be signs. For example, a pain in the spine is a very frequent occurrence in the war psychoneuroses. Indeed, in my experience, only a very small percentage of cases complaining of pain in the spine suffered with disease of the spinal cord; and that is true in civil life as well as in military life.

A man may give a history of being blown up or of having fallen and sustained an injury of the back. He complains of pain and tenderness in the spine, which has caused him to lose the use of his legs or to walk with a bent back. Such cases came often under my notice. I will cite one in particular. A conscript had lost the use of his legs and suffered pain in the back following a fall; I examined his spine and told him to tell me exactly the spot where he felt the pain, percussing each spine as I went down. I marked with blue pencil the tender point; at the same time I remarked aloud that this was too low to affect the spinal cord, and he obliged me, when I percussed the spine again, by shifting it higher. Again I suggested that it was too low, and again he shifted it higher. I then tested his reflexes, and finding them normal, suggested that he should get up and walk, which he did.

Nevertheless, although the great majority of cases coming with pain in the spine and tenderness may be functional, occasionally cases occur in which there is organic disease. Thus a good soldier, a sergeant, who had been blown up and buried for three days, complained of pain on pressure over the fifth cervical vertebra on the left side. He had been regarded as a functional case of mild paraplegia. I found, however, a well marked extensor response of the big toe on the left side, absence of the abdominal reflex on that side; and an x-ray examination showed a fracture of the transverse process. I diagnosed

a resulting pachymeningitis. This has since spread, until now he has complete spastic paraplegia. Well, in this case, had his papers been filled up as "functional," he would not have received a gratuity nor an adequate pension.

Localized tenderness on pressure and pain of the spine which cannot be shifted by suggestion, should always be carefully investigated, even in the absence of any deformity and although a neuropathic tendency is obvious. Evidence of pain on movement of the spine in the various directions possible should not be neglected. The exact situation of the tenderness should be noted and the character and localization of the pains or abnormal sensations should be investigated—for this will give important information regarding the anatomical diagnosis. Pain and tenderness on pressure, combined with a localization of pain or paraesthesia in a skin area, corresponding to a segment or segments of the spinal cord, are of great diagnostic importance in cases and new growth of the spine, tumours, aneurysm eroding the spine, and extramedullary tumours affecting posterior roots. An x-ray examination will help to confirm the diagnosis, localize the disease, and indicate the treatment.

Pain in disease of the spinal cord is especially a sign of irritation of the spinal ganglia or sensory roots as they pass through the intervertebral foramina to the cord by a localized or general meningitis.

The Character of the Pain.

The pain due to early spinal cord disease is of a neuralgic character. It varies in severity and from time to time, according to the temperament and general health of the individual. It may be described by the patient as lancinating, boring, cutting, tearing, burning, and so severe may it be as to cause the patient to cry out. It may be continuous, dull, and contusive, with paroxysmal crises of acute pain. It is differentiated from true neuralgia and neuritis by its segmental topographical distribution. In neuralgia there are anatomical points, compression of which gives rise to pain. These neuralgic points are also the seat of pain in the nerve, and seem to be the point of departure of the paroxysms constituting the crises. Pains of a neuralgic type and especially severe, however, arise as a result of compression or irritation of the spinal roots or of the brachial, lumbar, or sacral plexuses. These pains are called *pseudo-neuralgic*, and are met with at the commencement of cases of compression of the spinal cord in cases of the spine, aneurysm of the descending aorta, in the paraplegia dolorosa of cancer, and in cases of irritation of the spinal roots from localized and general meningitis.

The pain of general or local meningitis is frequently associated with general or local muscular spasm and rigidity. This is very obvious when the cervical region is involved. In cervical cases there is local tenderness and rigidity, and if we see a patient supporting his head with both hands owing to pain in the neck, this is almost a sure indication that he is suffering from disease of the cervical vertebrae.

Sooner or later these root pains will be associated with objective sensory disturbances disposed in bands on the trunk and long strips on the limbs. The reason for this is seen in the distribution of the fibres of the posterior spinal ganglion.

Whereas pressure on nerve trunks in peripheral neuritis causes lively pains, this is not so obvious when a nerve is pressed in radiculitis (spinal root inflammation). But severe pain may be caused by sneezing, coughing, blowing the nose, or defaecation. The cause of root pain is a local or general meningitis affecting the posterior sensory roots in some part of their course from the intervertebral foramina to the spinal cord. Not infrequently, especially in syphilis, a diffuse meningitis is associated with the primary local condition, and that this exists is shown by a lymphocytosis of the cerebro-spinal fluid.

Syphilitic Meningitis a Frequent Cause of Root Irritation.

In the greater number of cases of spinal cord disease commencing with symptoms of local root irritation followed by a generalized spinal meningitis (often cerebro-spinal meningitis) syphilis is the cause, and in addition to an abundant lymphocytosis of the cerebro-spinal fluid, a

positive Wassermann reaction of the blood and fluid is obtainable.

The lumbo-sacral region is a frequent seat of root pain, but it may occur in the dorsal or cervical region in syphilis.

The initial lesion of tabes is marked by root pains of a lightning, stabbing, burning character, which are in all probability due to irritation of the rootlets of the posterior spinal ganglion at their entry into the cord. This irritation is probably set up by an inflammatory process in the membranes at the point of entry.

Segmental Pain and Herpes Zoster.

Severe neuralgic pain with a spinal segmental distribution does not always result in symptoms pointing to disease of the spinal cord, and the inflammatory process may affect only the posterior spinal ganglion. When this structure is acutely inflamed, "herpes zoster" appears in the area of distribution of the sensory nerve fibres issuing from the ganglion. The appearance of the eruption is usually followed by a cessation of pain. Intercostal neuralgia and pleurodynia are frequently diagnosed in early rachialgia due to organic disease affecting spinal roots.

Pain in Peripheral Neuritis.

Severe shooting pains in the limbs, especially the lower limbs, followed by objective signs of sensory disturbances, such as hyperaesthesia upon pressure of the calves, associated with anaesthesia to light touch and analgesia, occur in peripheral neuritis, but the history of the case and the distribution of the sensory disturbance will enable a differentiation between this and spinal cord disease to be made.

It must be remembered that in some cases the pains in peripheral neuritis may be of a lightning character and thereby simulate tabes, especially if there are ataxic symptoms and absence of the knee and Achilles jerks, but there is usually an associated motor affection in the form of paresis or paralysis, associated with wasting of the muscles. The pupils are normal, and the Wassermann reaction is negative.

Lightning Pains and Early Symptoms of Tabes.

It is noteworthy that in the pre-ataxic stage of tabes lightning pains occur, and yet Romberg's symptom may be absent and the knee jerks and Achilles jerks present. Hence these pains are often thought by the doctor to be due to rheumatism, muscular rheumatism, or neuritis. I have seen cases of tabes in the pre-ataxic stage where the deep reflexes have disappeared, first on one side and then on the other, after crises of pains. Lightning pains are rarely absent in ataxic patients; they constitute the earliest symptom. Often these pains are for a long time believed by the patient, and even sometimes by the doctor, to be rheumatic or neuritic in origin. It is rare that there is not some pupillary affection in this disease. Unequal pupils, irregular pupils inactive or sluggish to light and active to accommodation—the Argyll Robertson pupil—in conjunction with shooting pains in the limbs, are sufficient evidence to diagnose locomotor ataxy in the pre-ataxic stage. Cases of tabes occur where the knee jerks are present but the Achilles jerks are absent. These cases often show loss of bone sensibility to the vibrations of a tuning-fork.

Painful Visceral Disturbances an Early Symptom in Tabes.

Next to lightning pains bladder troubles are among the earliest and most constant symptoms of tabes; they are not severe, and, according to my experience, the patient does not usually seek the advice of a doctor for this reason. It is only as the result of inquiry, as a rule, that the patient in relating his symptoms mentions difficulties in starting micturition or holding his water.

Bladder crises have been described. They consist of violent pains which occur in the lower part of the belly, radiating to the inner side of the thigh. The patients have an urgent desire to micturate, but are unable; they experience the most severe burning and cutting pains in the urethra. These crises may last a few or many hours. Renal crises have also been described. These crises may simulate passage of a stone or gravel in a patient.

Gastric crises again are not infrequently the earliest symptom of the disease, and the attacks of pain and vomiting may be the cause for which the patient seeks advice. I have seen cases of visceral crises in which the surgeon has been called in with a view to operation for intestinal obstruction, and I have seen two patients, early cases of tabes with visceral crises, upon whom abdominal section had been performed. One patient had pus in the urine, probably owing to cystitis, but the surgeon found the kidneys healthy. Visceral crises may be the sole complaint in tabes of which the patient is conscious.

One patient of mine remained in the pre-ataxic stage twenty years, the sole symptom of the disease of which he was conscious being attacks of severe pain which extended both vertically and transversely between the shoulder blades and over the whole back; as soon as the pain became acute, vomiting would take place and relief would come. He had, however, Argyll Robertson pupils and thoracic anaesthesia to light touch.

Rectal and intestinal crises may also occur and are among the early symptoms of tabes. The patients complain of tenesmus and urgent desire to go to stool, of severe pain in the back passage, like the introduction of a hot iron; sometimes this is accompanied by tenesmus and straining. Laryngeal crises are a rare early symptom and the attacks are not so frequent as visceral crises.

The association of thoracic anaesthesia with visceral crises gives an anatomical explanation of their causation; the afferent visceral sympathetic nerves terminate in the posterior cornua of the spinal cord segments corresponding to the seat of the pain in the skin and the anaesthesia, the irritative inflammatory process that affects the posterior sensory roots innervating the skin irritates the afferent visceral fibres.

Again, painless spontaneous dislocation or fracture may be the first event to bring the patient under medical or surgical observation. Upon inquiry it will generally be found that these patients have had lightning pains which they attributed to rheumatism.

Impotence or satyriasis may be an early symptom of tabes.

The visceral as well as the somatic symptoms depend upon the anatomical seat of the irritative and destructive morbid process.

In all these early manifestations of tabes which bring the patient to the doctor, other signs or symptoms will be revealed by careful examination, the most frequent and important diagnostic clinical sign being the pupil phenomena. The Argyll Robertson pupil shows that a patient has tabes or is a candidate for it, and to me it is as sure a sign of syphilis as the chancre or the secondary eruption.

Girdle Pain an Early Symptom of Chronic Meningo-myelitis.

A girdle pain is common in tabes, but it is also an early symptom of a focal meningitis or meningo-myelitis of slow and progressive evolution, and marks the commencement of the affection.

This early symptom of root affection is very common in syphilitic disease of the spinal cord. The recognition of inflammatory root symptoms—namely, paraesthesia and girdle pains in syphilitic disease of the spinal cord—is a matter of great importance as regards early active treatment being successfully carried out before the inflammation has had time to spread to the spinal cord itself, producing thereby a transverse myelitis, which destroys the nervous structures and leads to ascending and descending degeneration of the tracts conveying sensory and motor impulses to and from the brain. Once this degeneration has occurred a permanent spastic paraplegia is installed, and no amount of specific treatment can restore the destroyed nerve fibres. I have recently seen an ex-service man who had all these warning symptoms, and yet no endeavour had been made to discover by a Wassermann reaction and examination of his fluid the cause of his symptoms and their progress until he became completely paralysed, when he came under my observation.

Syphilitic meningo-myelitis is the most frequent cause of paraplegia in young male adults, and root pains and dysaesthesia are the earliest warning symptoms. If treated in the early stage the results are surprisingly satisfactory. An idea unfortunately still lingers in the profession that affections of the nervous system are a late

tertiary manifestation of syphilis, in spite of the fact that Fournier long ago showed that the severest and largest number of cases of cerebro-spinal syphilis occurred within the first year after infection and diminished in severity and frequency with each successive year. Again, Jonathan Hutchinson pointed out that one-half of the cases of syphilitic paraplegia occurred within the first eighteen months after infection. These facts show how valuable is an examination of the cerebro-spinal fluid as an aid to diagnosis and subsequent treatment in all cases presenting possible symptoms of spinal cord disease. Thus a leucocytosis and discovery of the *Meningococcus intracellularis* is a positive proof of epidemic cerebro-spinal meningitis. A lymphocytosis points to either syphilis or tubercle, but a differential diagnosis is easily effected in a doubtful case by the results of a Wassermann reaction of the blood and fluid and attention to the history of the case, the clinical evidence, and the result of antisyphilitic treatment. I may mention that a lymphocytosis is present in practically every case of untreated syphilis or tabes, but the fluid does not always give a Wassermann reaction, although it may appear after a provocative dose of salvarsan.

DYSAESTHESIA.

Dysaesthesia is a very early symptom of spinal cord disease. Abnormal sensations, such as heaviness of the limbs, numbness, tingling, pins and needles, sensations of heat and cold in some part of the body, especially of the limbs, may bring the patient to the doctor. He may find him a neurasthenic, who has suffered with this symptom on and off for some time, or that it has recently commenced; in any case he should make a careful investigation to ascertain the cause.

The Conditions which Cause Dysaesthesia.

We are all familiar with the unpleasant effects of sitting for some time on a hard seat, thereby compressing the sciatic nerve: upon rising the leg feels heavy, there is a numb feeling associated with pins and needles. This is due to a temporary interruption, by the compression of the nerve, of the normal sensations which are continually contributing subconscious evidence of that part of our personality.

A number of morbid conditions besides compression of a nerve may cause dysaesthesia. They are:

1. Local transitory modifications of the circulation such as are met with in Raynaud's disease and erythromelalgia, due to vasomotor spasm. Also arterial circulatory disturbances in the limbs occasioned by atheroma, arterio-sclerosis, and endarteritis.

2. Reaction to cold.

3. Neuritis of traumatic, toxic, or infective origin.

4. Symptoms of dysaesthesia or, as these objective symptoms are also called, paraesthesia, especially of the extremities (acroparasthesia), are met with in neurasthenics. According to my experience subjective sensory symptoms of numbness and tingling of the extremities is a frequent symptom complained of by neurasthenics.

Having given the causes of dysaesthesia which may occur in other conditions than spinal cord disease, I will now pass on to the subject of dysaesthesia as an early symptom of spinal cord disease.

Dysaesthesia as an Early Symptom of Spinal Cord Disease.

Many of the conditions which give rise to neuralgic pains give rise to abnormal sensations of numbness, tingling, pins and needles—that is, in the prodromal periods of vertebral disease, intramedullary and extramedullary tumour, dysaesthesia may even precede the pain in these affections.

Again, dysaesthesia may be the first symptom in chronic spinal meningitis, acute or chronic myelitis, anterior poliomyelitis, myelomalacia, or thrombotic softening of the spinal cord, transverse syphilitic meningo-myelitis, and disseminated sclerosis. Therefore, when this symptom is complained of, even in the absence of objective signs of disease, it is imperative carefully to investigate the probable causation, look for objective sensory disturbances, examine the condition of the reflexes, and note if there is any evidence of motor weakness.

OBJECTIVE DISORDERS OF SENSIBILITY.

Total anaesthesia in disease of the spinal cord only occurs in cases where there is a complete transverse lesion, such as occurs in fracture dislocation, bullet wound, or severe meningo-myelitis caused by compression or disease; the prognosis is always grave. In meningo-myelitis the subjective sensory symptoms previously described and hyperaesthesia precede the anaesthesia in all cases arising from focal myelitis, or radiculitis, or diffuse myelitis.

Dissociated anaesthesia is more frequent and more important as a diagnostic sign than total anaesthesia in the localization of lesions. By dissociation I mean that all modes of sensation are not affected, or not affected to an equal degree. Superficial sensibility to a light touch with a wisp of cotton-wool, to pricking, and to heat and cold may coexist with unimpaired deep sensibility. An anaesthesia to light touch may be associated with the preservation of pain sensibility, and even so exaggerated that touch sensations are only recognizable as painful (anaesthesia dolorosa). This sensory dissociation may be met with in peripheral neuritis and in cases of compression and irritation of roots. In syringomyelia and haematomyelia, where the grey matter is disorganized, pain, heat and cold sensations are not felt but tactile sensations are.

Diseases of Spinal Cord in which Sensory Phenomena are Transitory or Absent.

There are certain diseases of the spinal cord in which sensory phenomena are transitory or absent.

Anterior poliomyelitis, an infective disease with an acute onset, is ushered in by fever, malaise (frequently vomiting), associated with numbness, tingling, and pins and needles in the limbs, followed in a few hours by a flaccid paralysis of the muscles, proceeding to wasting, with reaction of degeneration and permanent atrophy of some groups. The anatomical lesion is an inflammation affecting especially the anterior horns; the spinal motor neurones are damaged or destroyed, consequently the motor fibres degenerate and with them the muscles they innervate. The posterior spinal ganglia—the trophic centre of the sensory fibres—is not involved; consequently they do not degenerate, and the sensory symptoms are therefore only transitory.

In progressive muscular atrophy there is a progressive and insidious decay and destruction of the spinal motor neurones, and as they degenerate and die, the muscle fibres, which they innervate, degenerate and undergo atrophy. The characteristic fibrillary twitchings in the degenerating muscles is evidence of the increased irritability of the degenerating fibres. There is no sensory disturbance of function in this disease, as the sensory neurones are not affected. Amyotrophic lateral sclerosis is a degenerative disease, not only of the spinal motor neurones innervating the muscles but also of the cortico-spinal neurones which carry voluntary impulses from the brain by the pyramidal tracts to the spinal motor neurones which they control. In this disease also there is no sensory trouble. Both these diseases—progressive muscular atrophy and amyotrophic lateral sclerosis—tend to affect early the small muscles of the hand and shoulder girdle. In amyotrophic lateral sclerosis there is a wasting of muscles associated with exaggerated deep reflexes, patellar and ankle clonus and plantar extensor response; these form the early symptoms of the disease.

Another disease probably infective in origin but of extreme importance is disseminated sclerosis. This disease is easy of diagnosis when such cerebral symptoms are present as intentional tremors, staccato speech, nystagmus, and optic atrophy, but when only spinal cord symptoms are in evidence it is very apt to be mistaken for functional disease. Not infrequently dysaesthesia—namely, numbness, tingling, pins and needles in the limbs—marks the onset of the disease; this is followed in some cases by temporary anaesthesia; the patient then suffers with a spastic condition of the legs associated with a certain amount of paralysis or paresis, inco-ordination and difficulty or awkwardness in gait and station. When these symptoms are accompanied—as they often are in young women—by emotional symptoms, an erroneous diagnosis of hysteria may be made, especially if a systematic examination of the superficial and deep reflexes is not made. Absence of the epigastric and

abdominal reflexes on one or both sides, exaggeration of the knee-jerks, patellar clonus, ankle clonus, and—most important—plantar extensor response of the big toe, with fanning of the other toes, paresis, loss of sense of position, and diminution of bone sensibility show that there is a lesion involving the pyramidal tracts and the posterior column. The superficial sensibility is usually unaffected.

THE IMPORTANCE OF ANATOMICAL DIAGNOSIS IN PROGNOSIS AND IN CASES WHERE SURGICAL INTERFERENCE MAY BE CONTEMPLATED.

The diagnosis of the exact localization of disease of the spinal cord is of great importance from two points of view—namely, in prognosis and in the rare cases where surgical interference is contemplated.

The prognosis is always most grave where there is clinical evidence of extensive diffuse myelitis and where the lesion affects certain regions of the spinal cord owing to dangerous symptoms and complications arising. These lesions in the upper cervical region may affect the phrenic neurones. Again, a lesion in which the symptoms point to a complete transverse myelitis is evident by an absolute loss of sensibility below the lesion paraplegia and loss of control over the sphincters. In such cases cystitis and secondary infective nephritis are liable to occur; also large sacral bedsores, unless great care be taken by the doctor and nurse.

When the lesion affects the lumbo-sacral region and the lower motor neurones are destroyed there is, in addition, atrophy with reaction of degeneration of the muscles of the lower extremities; the sphincter troubles are more serious and bedsores are almost sure to occur in spite of careful treatment and nursing.

Anatomical Diagnosis in Relation to Surgical Interference.

A precise anatomical diagnosis in cases of extra-medullary tumour of the spinal cord and meningitis circumscripta is essential before operative interference can even be contemplated. Extradural tumour is really a more hopeful surgical operation than cerebral tumour. The difficulty of diagnosis lies, however, in the fact that we cannot always be sure that the tumour is outside the cord. It may be intramedullary. The existence of definite symptoms of root irritation prior to pressure symptoms serves as a means of localization of the situation of the tumour and affords evidence of its extradural situation.

The earliest symptom noticed by the patient in extradural tumour is pain or paraesthesia, generally on one side and only in the area of distribution of a root and associated with hyperaesthesia of the corresponding skin area. As the tumour increases in size it produces sooner or later a unilateral compression of the spinal cord of the same side, and this causes an interruption of sensory and motor tracts.

Now it frequently happens in these cases that a sensory dissociation known as the Brown-Séquard phenomenon is found—namely, there is loss of sensibility to pain, heat, and cold of the side opposite to the lesion and paralysis and loss of tactile kinaesthetic sense on the same side as the lesion. This can be explained on anatomical grounds. The fibres conveying sensations of heat and cold and pain decussate to the opposite side almost immediately in the grey matter, whereas the fibres conveying tactile kinaesthetic impressions do not decussate until they reach the medulla. The existence of compression in the pyramidal tract fibres is shown by weakness or loss of voluntary power in the limb, dragging of the foot, increase of deep reflexes, ankle clonus, and plantar extensor response. If unrelieved the pressure increases as the tumour grows and a complete paraplegia results.

These cases of slow-growing benign extradural tumour may be easily missed in the early stages and the case regarded as functional, unless a very careful and methodical examination is made, and that leads me to say a few words in conclusion regarding the differential diagnosis of spinal cord disease and hysteria in its multiform simulations.

Diagnosis of Functional Diseases.

The experiences of the great war have given us innumerable instances in support of Babinski's view that these

functional disabilities are the result of auto- or hetero-suggestion, and can be cured by suggestion or persuasion.

I do not, however, hold with those authorities who think it unnecessary or undesirable carefully to examine a case to enable them to exclude organic disease. Certainly there is a danger of suggesting to the patient symptoms, especially in respect of sensory disturbances, but after you have made a careful and methodical examination of the superficial and deep reflexes and found them all normal, you are justified in arriving at the conclusion that the contracture, paralysis, or inability to stand or walk (astasia abasia), which may have even existed for months or years, is the result of auto- or hetero-suggestion, and you can then with confidence assure your patient that he can be cured. In these functional cases, moreover, even after prolonged disuse of the limbs, there is little or no wasting, and the muscles all respond normally to electrical stimulation. I would remind you that in long-standing functional cases there is acrocyanosis and coldness of the feet, so that no response occurs to stimulus of the sole. If the foot be warmed the response can be obtained.

The recognition of functional sensory disabilities simulating spinal cord disease is easy: the superficial sensibility to pain, heat and cold, and touch is lost completely; there is no dissociation, neither the anaesthesia nor the pains complained of conform to the anatomical distribution of spinal roots or peripheral nerves. In the limbs the superficial anaesthesia takes the form of a stocking or gauntlet, and can easily be removed by suggestion, the restoration of sensibility being from above downwards. The secret of success in the treatment of these functional cases is faith; consequently, in the first treatment you must not leave the patient until you have established that by bettering or curing the disability. It may take minutes; it may take hours.

However, among the very numerous cases of functional disability there are many cases of organic disease of the spinal cord with a large halo of functional disturbance, which can be removed by various methods of suggestion, by re-education and various other forms of encouragement. But the fact must not therefore be overlooked that such a patient, in spite of the satisfactory results of such treatment, is suffering with a permanent disability, which may progress in spite of improvement by the treatment.

From what I have said it is clearly necessary to decide by examination: First, whether the case is functional or organic. Secondly, if organic, in order to form a correct judgement regarding prognosis and treatment, it is essential to diagnose the pathological nature of the lesion and its anatomical situation.

In conclusion, I would ask you to bear in mind that more mistakes are made from not looking than from not knowing.

THE LABYRINTHINE REACTIONS OF EXPERIENCED AVIATORS.

BY

DAVID RANKEN, M.S.LOND., F.R.C.S.ENG.,
SQUADRON LEADER R.A.F. MEDICAL SERVICE.

This investigation was undertaken in order to ascertain the difference, if any, between the labyrinthine reactions of experienced pilots and those of the average individual of the same age who has done no flying. In the American Air Force the Bárány rotation tests have played a much more important part in the acceptance or rejection of aviation candidates than in our own, and it is stated by Colonel Isaac Jones of the American Air Force, in his book entitled *Equilibrium and Vertigo*, that in addition to the majority of aviation candidates, all experienced pilots so far examined by them have without exception shown normal responses to their routine standards in the turning chair.

As the Aviation Candidates Medical Board, Hampstead, has during the last eighteen months examined several hundreds of experienced pilots for civilian aviation, the present seemed a suitable time for such an investigation. All tests employed have been performed as closely as possible in conformity with the rules laid down for the