A British Medical Association Lecture ON SOME CLINICAL MANIFESTATIONS OF TABES DORSALIS.

DELIVERED TO THE HARROGATE BRANCH, OCTOBER 7TH, 1922,

BY

GORDON HOLMES, M.D., F.R.C.P., C.M.G., C.B.E.,

PHYSICIAN TO OUT-PATIENTS, NATIONAL HOSPITAL; ASSISTANT
PHYSICIAN, CHARING CROSS HOSPITAL.

THERE can be little doubt that no nervous disease presents itself under so many and varied manifestations as tabes dorsalis; perhaps no other affection of the body assumes so multifarious forms, and the experience of my own practice is that in no other condition is my help in diagnosis more frequently asked by general practitioners and by other specialists. I have consequently selected as my subject some of the clinical manifestations of this disease, and will deal chiefly with those which offer difficulties in diagnosis. These are usually early or isolated symptoms. Time will, unfortunately, prevent any but a cursory reference to the pathology of the disease or to the pathogenesis of its symptoms, and even the question of treatment must be largely neglected, conscious though I am that to those engaged in the daily care of patients treatment must demand the foremost place in the discussion of any disease. My excuse is that in tabes diagnosis must precede efficient treatment.

Tabetic Pains.

Pains are notoriously among the earliest and most common symptoms, but they are by no means constant, for even the most careful questioning of some patients fails to elicit a history of any. There can be rarely any doubt of their origin when an accurate description of their nature is obtained, since the shooting, lancinating, or lightning pangs, or the sharp darting or burning stabs, often compared to red-hot needles, which shift from place to place, are almost typical. The difficulty in recognizing them occurs when their character or the description of them obtained is less typical, and especially when, being an early feature of the disease, it is for their relief only that the patient seeks our advice.

The proportion of early tabetics who first complain of "rheumatism," or, using the term made popular by the war, call their trouble "myalgia," is certainly considerable, and in many of these the knee-jerks are still present, the pupils react to light, and there is often no disturbance of gait. The features that should attract our attention are the reference of the pains to the muscle masses, or to the tissues between rather than around the joints, and the fact that they are usually neither excited, aggravated, nor otherwise influenced by movement or by pressure as rheumatic and myalgic pains are. Further, these early pains frequently occur in the trunk, where rheumatic pains are rare, or trunk pains appear simultaneously or alternately with limb pains.

In many cases where the pains are limited to a small region the diagnosis of "neuritis" is made, or they may be attributed to "sciatica," when they occur chiefly on the posterior aspect of the lower limbs. But if we have an intelligent patient, or can obtain an accurate history, it is usually easy to exclude a neuritis. In the first place the pains are rarely, if ever, limited to the distribution of the sciatic or any other nerve, and if they happen to be they spread over a small part only of its sensory area and do not follow its whole course; if they occupy the sciatic region they generally occur in other parts of the limb too, or in the opposite limb, or in the trunk, which is, of course, exceptional in sciatica. Further, in neuritis and sciatica the affected nerves are tender or at least sensitive to pressure and stretching, while in tabes they are usually completely or abnormally insensitive, as Biernacki pointed out.

It is, however, pains limited to certain regions that are most likely to confuse us or lead us into error. While the description of a typical girdle pain around the waist or thorax arouses our suspicions at once, we are more likely to overlook the origin of a pain which is more or less constant and fixed in the side of the abdomen or chest, seeking its explanation in some disease or disturbance of the underlying visceral organs. But such local pain is common in tabes, and is often an early symptom. Here again the nature of the pain and its distribution are valuable guides. As a rule the tabetic pain is not

limited to an area on any aspect of the trunk as visceral pain usually is, or though it may be greater at one point it tends to spread around the whole distribution of those sensory roots to the disease to which it is due. But this is not always a characteristic feature, since girdle or semi-girdle pain is often associated with visceral disease; we are all familiar, for instance, with the pain which encircles one side of the lower thorax and abdomen in basal pleuropneumonia, and the radicular hyperaesthesia of Head and McKenzie associated with disease of deeper organs is often described as pain. The absence of evidence of such disease is, of course, an important point in diagnosis; but still more is the fact that in tabes the pain is not accompanied by those manifestations which one would expect were it due to an affection of a visceral organ. Pain referred to the abdomen, which we might attribute to the appendix or gall bladder, is, for instance, not associated with tenderness or resistance over these organs, and flat in the thorax is not aggravated by deep breathing nor associated with limitation or alteration in the respiratory movements. Further, the patient's conduct and activity are not, as a rule, compatible with visceral disease of sufficient intensity to produce such pain, for the tabetic often continues to lead an active life, or shows at least little reluctance to move, though he complains bitterly of his sufferings.

The possibility of tabes must be therefore always considered when, on the one hand, movement or the functional activity of the organ suspected does not aggravate or increase in any noteworthy degree a severe and persistent pain, and on the other when pressure or handling of the parts to which it is referred has little or no influence on its severity. But caution is necessary in applying these tests, since the skin of an area where tabetic pains have persisted for some time often becomes unnaturally sensitive to certain forms of stimulation at least. It is most commonly excessively tender to moving stimuli, as to a hand or even a wisp of cotton-wool drawn over it, but then heavy pressure or a pin-prick may give no discomfort, or indeed less than it would on normal parts.

This observation leads us to the most important point in its differential diagnosis, which is that where tabetic pain has persisted for any considerable time there is a disturbance of sensation, most commonly a reduction of pain sensibility. The suffocating tightness around the thorax or the abdominal girdle pain occurs, as is well known, in areas of analgesia or hypalgesia, and the same general rule holds for pain of other distribution. It is, in fact, rare to find all forms of sensation intact where pain is severe, except in a small proportion of those cases in which it is recent.

These sensory disturbances are almost invariably the only objective or demonstrable changes associated with tabetic pains. Occasionally, though very rarely, the existence of pains in one region of the body is followed by changes in the skin; I have seen subepidermal haemorrhages in limbs that had been subject to prolonged local pains, and in one case repeated outbursts of herpetic vesicles. A local falling of hair and thinning and atrophy of the skin have been also described.

The frequency of local pains in the limbs, and particularly in the trunk, is generally recognized, but such local pains occurring in the head have attracted less attention. They are not prominent symptoms in a large proportion of the cases, but are yet sufficiently common to warrant notice. I have, for instance, seen more than once recurring tabetic pains in the cheek diagnosed and treated as trigeminal neuralgia, and one of my patients had even undergone alcohol injections for this condition, though careful questioning would have revealed the true state and obviated a therapeutic failure. But more common than facial pain is pain of either sharp and stabbing or dull and aching character, limited to or most pronounced in the occipital region of the scalp; it is therefore likely to be confused with occipital neuralgia. When an isolated complaint the origin of these pains may not be easily recognized, but a careful history generally discovers the existence at some time or other of more or less similar pains elsewhere, and examination brings to light other signs of the disease, for these facial and occipital pains are rarely early or isolated symptoms. When tabes is suspected their nature can be assured by even a cursory examination of sensation, for, as is the rule elsewhere, pain sensibility is absent or diminished where they occur, though tactile and probably other forms of sensation are intact or little disturbed.

Not infrequently it is not for the relief of pains that the patient consults us, but owing to the existence of an unnatural sensitiveness or tenderness of some part of his body to stimuli

which normally give little or no discomfort. Those accustomed to the routine examination of patients with nervous complaints are familiar with the excessive reaction and violent expostulations that even gentle scraping of the soles in eliciting the plantar reflex may evoke in a tabetic. Sometimes there is a similar hyperaesthesia to other stimuli, as the rubbing of the clothes on the trunk and limbs. Unnatural sensitiveness to thermal stimuli, and especially to cold, is more common. The touch of a cold hand may be resented, and a few drops of cold water sprinkled on the trunk, particularly on the back, may evoke intense burning or stabbing pains. This is occasionally one of the first symptoms of the disease. I lately saw a doctor suffering with tabes who discovered three years before any other symptoms appeared that he could no longer swim or take a cold bath, since immediately the water reached his waist he experienced what he described as "an awful feeling as if his body were being cut through by red-hot knives." Some of these patients come under observation complaining of rheumatism or neuralgic pains, which are excited or aggravated by a draught of cold air or by contact with any cold object.

Paraesthesiae.

Another important group of symptoms are the various paraesthesiae so common in tabes. Sometimes it is a complaint of numbness or loss of feeling; sometimes it is of a "uselessness" of the hands or feet, to employ a vague description often used by the patients; more rarely it is a distressing sensation of either heat or cold, or a feeling of dampness or wetness of some part. The difficulty in recognizing the nature of these symptoms is due mainly to the fact that objective changes corresponding to the loss or perversion of sensation of which the patient complains cannot usually be demonstrated by a casual examination. Though he may tell us that he does not feel the ground properly under his feet, that it seems to him as if he were walking on air or on soft cushions, we may find tactile and perhaps other forms of cutaneous sensibility on his feet unaltered, and we may consequently conclude that there is no organic basis for his complaint. But, as Hughlings Jackson pointed out, the sensation of resistance or the recognition of the consistence of objects, as the hardness of the floor, depends not on cutaneous sensation but on those afferent impressions which arise from the deeper structures of the limbs, especially from the muscles and tendons. The feeling of "uselessness" in the hands also is due mainly to disturbances of the sense of position, or of the power of recognizing the form and weight of objects handled, rather than to loss of cutaneous sensibility.

There is often, however, some disturbance of skin sensation too. In a patient under my care one of the first symptoms was inability to "feel" his billiard cue properly, which seemed at first inexplicable, as tactile, pain, and thermal sensibility on his fingers were intact. Later the "loss of feeling" increased so much that he had to cease dining out since he could not use his knife and fork properly, and since he could not "feel" the coins in his pocket he could take a taxi only when accompanied by a friend; but still there was little or no insensitiveness of the skin to the ordinary tests. A more complete examination revealed, however, great impairment of the sense of position in his fingers, and that he was unable to recognize accurately the shape, size, and weight of objects placed in his hands. Since it is to disturbances of these qualities of sensation that similar disorders are due in other spinal and cerebral diseases as well as in tabes, a careful investigation, particularly of the sense of position, is essential in attempting to elucidate their nature.

This numbness or subjective loss of feeling is occasionally found in unexpected parts; I have seen a few patients whose chief complaint was that their penis and scrotum felt numb or dead, and in one man at present under my care the most distressing symptom is that he has no feeling in his lips and cheeks.

Tabetic Crises.

Next in order after pain it is the tabetic crises that are most likely to offer difficulties, or to lead us into error in diagnosis. We are all familiar with the patient on whom perhaps several abdominal operations have been performed without giving the promised relief. I recently saw two patients on successive days, one of whom had undergone four and the other five abdominal explorations for attacks of acute abdominal pain and vomiting. The appendix or the gall bladder may be suspected, or the symptoms may suggest perforation. A neurologist may easily exaggerate the frequency with which this mistake is made, but that it is not

uncommon is shown by the experience of an American colleague lately working with me. He obtained permission to investigate patients on whom abdominal operations for acute symptoms had been recently performed in several general hospitals, and he discovered among them a proportion of tabetics subject to gastric crises much larger than I would have believed possible. Gastric crises probably occur in about 10 per cent. of all tabetics.

Here, as is the case in tabetic pains, the errors in diagnosis can be attributed largely to the fact that these crises are frequently early symptoms, and are often not accompanied by those signs that are commonly held to be characteristic of the disease. Many patients, in fact, state that apart from the abdominal attacks they enjoy perfect health.

We can as a rule gain little information of value from the patient, as the pain and the character of the vomiting resemble closely those due to intra-abdominal conditions. The pain, which generally precedes the vomiting, commences in the epigastrium, but later radiates into the back and spreads throughout the whole abdomen and into the loins; it is usually of an intense burning or violent cramp-like character, and is generally accompanied by sensations of constriction or distension. The vomiting which soon sets in may be more or less continuous for days, and may or may not be associated with nausea. The points which should arouse our suspicions that the pain and vomiting are of nervous origin are, in the first place, the history that similar attacks have occurred before, each of which ceased more or less abruptly and left the patient in apparently perfect health. But this is not a pathognomonic feature, since renal and biliary colic may also recur between periods of normal health, and it is whenever we are so unlucky as to meet with a patient in the first crisis that our difficulties are greatest. In the second place the most striking feature of a tabetic crisis is that, though the patient vomits incessantly and gives evidence of the most intense pain, the abdomen between the bouts of vomiting is lax, or at least not so resistant as it would be with an acute visceral lesion, and handling or palpation does not notably increase or excite the pain. But here too we must be on our guard, since the vomiting is often accompanied by intense hyperaesthesia of the skin; the first touch of the hand, and particularly gentle stroking, may evoke at once great discomfort and excite a reflex contraction of the muscles. But when the hand is laid firmly on the abdomen this tenderness disappears, and deep palpation can be often carried out without discomfort to the subject and without exciting muscular contractions; this is the most valuable diagnostic feature of tabetic vomiting.

Not infrequently patients complain not of the characteristic paroxysms of pain and vomiting, but of a constant nausea or aching in the region of the stomach. In these the symptoms are likely to be treated as merely manifestations of dyspepsia, but it is usually found that neither the nausea nor pain bears any relation to food, and that they do not yield even temporarily to change or restriction of diet, or to those drugs by which we treat dyspepsia or the conditions to which it is ordinarily due. Further, the appetite usually remains surprisingly good despite the discomfort, and there are no objective signs of gastric disturbance.

Laryngeal crises are probably the next most common variety. They, too, may give rise to errors in diagnosis. The most common form begins with tickling or a feeling of constriction in the throat, followed by a violent paroxysm of coughing, accompanied in severer attacks by dyspnoea and stridor, continuing till the patient becomes asphyxiated or even semi-conscious. An accurate description will probably indicate their nature at once, but unfortunately accurate descriptions, unless we elicit them by adequate questioning, are not common. The patient's complaints may consequently suggest either a local laryngeal disturbance or an attack of asthma; the latter is likely to be suspected, since laryngeal crises occur most frequently at night. Unless the signs of tabes are sufficient to attract attention or to indicate the diagnosis the conclusion must be based on negative rather than on positive evidence. Laryngoscopic examination reveals nothing to account for the attacks, or at the most an abductor palsy, while examination of the chest shows none of the signs usually associated with asthma.

Rectal crises, characterized by paroxysms of local pain, with distressing tenesmus, and often with the sensation of a foreign body in the rectum, are rarer, but I have seen them give rise to serious mistakes in diagnosis. Here, again, the absence of signs of local disease, the persistence of the symptoms over long periods, and the fact that the pain often

radiates more widely into the perineum and surrounding parts than it does in rectal affections, must put us on the search for a nervous origin of the complaint.

Bone and Joint Lesions.

The next group of symptoms that are perhaps most important to those engaged in the general practice of medicine are the bony and arthritic lesions which are so common in tabes. Fractures of the long bones, especially of the femur, may occur as a result of a trivial injury, or even of muscular action. The fact that there is often little or no pain may lead to failure to detect the true nature of the condition, and the difficulty in obtaining proper union may be, in the light of recent legal decisions, a serious matter for the surgeon who is so unlucky as to meet such a case. I obtained recently an autopsy on a woman whom I had followed for several years. About twenty years ago she slipped in the street, and, being unable to rise, was taken into hospital. There a fracture of the right femur was detected, and, according to her own story, the left femur was inadvertently fractured too, during the manipulations to set the broken limb. As a result she never rose from bed again. At the *post-mortem* examination I found both femora fractured, the broken ends widely separated and eroded, and enclosed in dense fibrous cysts. These fractures often occur under unsuspected conditions. Several years ago, while resident in a hospital, I was asked to see a charwoman employed there, who, while at her work immediately before, suddenly developed an enormous swelling of the right shoulder, with subcutaneous haemorrhage, which eventually tracked over that side of the thorax and down the arm. Examination revealed a fracture of the upper end of the humerus, which had apparently occurred without any pain; the haemorrhage was probably due to the jagged end of the broken bone having torn one of the large vessels in its neighbourhood. This woman's husband had died with tabes several years previously, but she had believed herself in good health, and even questioning failed to elicit any previous symptoms of the disease apart from a little "rheumatism."

The arthropathies are much more common, and are often the first symptom that brings the patient to notice. It is unnecessary to refer to the more or less characteristic features of these lesions, which were so accurately and vividly described by Charcot, but there are a few points in connexion with them which are of general interest. The first is their relation to injuries often so slight that they are disregarded by the practitioner who is called in, though emphasized by the patient, who is always on the look-out for an extraneous causal factor for his troubles. A slight strain at the knee or ankle is followed by a more or less painless swelling which persists longer than the nature of the injury appears to warrant, and is succeeded by disintegration of the joint, laxity of its ligaments, and the well-known deformities. It is often only when this stage is reached that the real nature of the condition is recognized. In fact the only feature which, as far as I am aware, may in the early stages excite suspicion is the painlessness of the swelling, but even this is not pathognomonic. Arthropathies in the limbs are generally easily recognized, but those which occur elsewhere may escape detection. A few months ago, for instance, a man was sent to me owing to a swelling in his lumbar region which obviously involved the spine. A vertebral tumour was suspected, the existence of which would have explained the absence of his knee-jerks, as the lumbar roots would have been certainly involved. It was only further examination of the patient which showed he was suffering with tabes, and the painless nature of the deformity then made diagnosis clear.

The arthropathies sometimes follow more serious injuries. Some time ago I saw a woman who, while in apparently good health, had twelve months previously sustained a Pott's fracture of her left ankle owing to a fall. The bones did not unite as would have been expected, and the swelling about the ankle-joint increased rather than diminished. When I saw her there was a typical Charcot lesion with considerable absorption of the lower fragment of the tibia and disintegration of the joint.

Among the so-called trophic disturbances the painless chronic and indolent perforating ulcers which occur on the soles need no comment; the only other condition in which they are commonly seen is diabetes, but they are in this disease, in my experience, usually associated with more pain or discomfort. It is, however, worth while bearing in mind that they are not limited to the sole; I have seen one on

the stump that bore an artificial leg, and one on the inner side of the cheek where a denture rubbed. Falling of the hair is less common, but lately a man under treatment for alopecia was referred to me as he complained of rheumatism; examination showed he was a typical tabetic who had almost suddenly and without apparent cause lost all his hair. Rapid loosening and falling out of the teeth is also sometimes seen.

Sphincter Disturbances.

The sphincter disturbances of tabes are another pitfall to the unwary. We all recognize the frequency of either retention or incontinence of urine in the advanced stages of the disease, but often forget that these may be among its earliest manifestations. Not infrequently I see patients who have been under treatment for local vesical troubles, or who have even undergone operations; it is consequently worth our while to consider shortly the more common forms that these bladder symptoms assume. The earliest change is generally slowness in starting the flow of urine; the patient has to wait an unnatural time before the urine comes and may have to use accessory means, as pressure over the bladder, to start it. Further, the bladder is often not completely emptied, so that a considerable amount of residual urine may be found immediately after micturition. In a man over middle age these symptoms may, of course, suggest an enlarged prostate or a stricture. Frequently occasional incontinence occurs at this stage too, the urine dribbling away in a few drops at a time, especially at night or on a sudden exertion, often unperceived by the patient, since the urethra and penis may be anaesthetic. In other cases there is a chronic distension of the bladder which may be unnoticed, since it is frequently insensitive to that degree of tension which normally produces pain or discomfort.

Rectal troubles are less common, but I have seen incontinence occur as an early symptom, occasionally the passage of faeces not being noticed by the patient owing to the anaesthesia of the anal region. As the sexual power is generally lost in males early in the disease, the possibility of tabes must be also considered in every case of impotence. Since the sexual appetite usually disappears at the same time it is not a matter of such distress and worry to the tabetic as impotence due to other causes usually is.

Ocular Symptoms.

One other group of symptoms to which I shall refer are the ocular troubles which occur so frequently in the course of tabes. Certainly the majority of patients afflicted with this disease have at some time or other during its course diplopia due to paresis of one or more of the ocular muscles. The external rectus is the muscle most commonly involved, especially in the early transient palsies, and next in frequency those supplied by the oculo-motor nerve.

There is nothing characteristic in either the form or course of these ocular palsies, and their origin can be consequently determined only when the disease is diagnosed. In many cases they are transient, disappearing within a few days or weeks; in others they are permanent and resistant to treatment of every form. Frequently the degree of dislocation of the visual axes is so slight that no squint can be observed, and the patient may complain of blurred or indistinct sight rather than of double vision. This is occasionally a source of confusion, since a disturbance of refraction may be suspected, or, the sight of each eye alone being good, the origin of the defect may be obscure.

Ptoxis, or drooping of the eyelids, may, of course, result from a lesion of the trunk of the oculo-motor nerve, or it may appear as an isolated symptom apart from palsy of any of the external ocular muscles. It is on the latter variety that I wish to lay emphasis, since it is an important diagnostic sign. There are, in fact, few cases who do not present it in some degree. The upper lids droop equally, or the one perhaps more than the other, but at first they rise to the full extent, or almost so, when the patient looks up. In order to compensate it the frontalis muscle is permanently contracted, and the forehead is therefore wrinkled. Largely to this is due the "tabetic facies" by which many cases can be recognized at the first glance; other factors in this typical appearance are the small pupils and the curious moist glistening eyes. So characteristic is this facies that one familiar with it can almost always pick out tabetics lying in a ward with, or sitting among, other patients.

It is also necessary to insist that though failure of the pupils to contract to light is regarded as one of the most characteristic signs of the disease it is not invariably present

in tabes, nor is it the only pupillary anomaly that occurs. The Argyll Robertson sign—that is, miotic pupils which fail to react to light but contract on accommodation—is probably seen in not more than 60 per cent. of the cases. In others the pupillary apertures may be normal in size, or those of the two eyes may be unequal, and not infrequently the pupils fail to contract on convergence too. In the latter case there is often a disturbance of accommodation; being a physician to a large ophthalmic hospital I often see patients complaining of defective vision due to palsy of accommodation, in whom examination reveals no cause other than tabes.

It must be borne in mind that though tabes is the condition in which the Argyll Robertson phenomenon is most commonly seen, it is not pathognomonic of this disease. There can be no doubt that it may result from local lesions of the mid-brain, and that it occurs occasionally in syphilitic subjects who never develop tabes; according to some authors it is found in uncomplicated cases of chronic alcoholism.

Finally, the frequency with which tabes is the cause of blindness or diminution of vision due to primary atrophy must not be forgotten. At the Royal London Ophthalmic Hospital a large number of cases with diminishing vision and the ophthalmoscopic appearance of primary optic atrophy are referred to me, and in a considerable proportion of these the neurological diagnosis is tabes. That the proportion is really larger than the physical signs at the moment of examination indicate I have no doubt, since the onset of blindness is frequently the first and the only sign of the disease. It is therefore worth bearing in mind that about 20 per cent. of all tabetics develop optic atrophy, and that tabes is a very common disease. Consequently, we can never afford to neglect the possibility of tabes in any patient who in adult years complains of failing sight.

DIAGNOSIS OF TABES.

As we have not considered some of those symptoms of tabes that may present difficulties in diagnosis, we must turn to the means by which diagnosis can be most accurately made.

The first question that arises is, What is tabes? Are we justified in making this diagnosis in patients with isolated symptoms, each of which may be due to other causes?—when, for instance, a man complains of pains in his legs and examination discovers only diminution of one knee-jerk; or when another has a primary optic atrophy and gives a history of transient diplopia, but presents none of the other so-called cardinal signs; or when Argyll Robertson pupils are the only demonstrable abnormality; or when we find a painless, swollen, and disintegrating joint, and loss or diminution of some of the reflexes. It may be easy to answer this question in any specific case, but it is certainly difficult to make a general rule. Even the pathological and serological tests are not conclusive, as they often are negative in patients with characteristic symptoms, and especially in slowly progressive cases in which the symptoms are not typical. Indeed, a positive Wassermann reaction is obtained in the blood in not more than 65 per cent. of all cases, and it may be negative even in the cerebro-spinal fluid, and the latter may show no other pathological changes in 20 per cent. If we therefore rely on the clinical pathologist we are liable to an error of 1 in 5 at least.

A short review of the pathology of the disease may help us. The most obvious anatomical change is a degeneration of the posterior columns of the cord. The older views that this is the primary lesion have been conclusively disproved, and all are now agreed that the spinal degeneration is secondary to disease of the posterior roots. But the nature of this primary affection is not yet conclusively settled. According to one hypothesis it is a primary degeneration of the fibres of the posterior roots due to the selective action of a syphilitic toxin upon them; according to another the main factor is injury of the roots by an overgrowth of connective tissue which surrounds and constricts them. All recent evidence points to the latter hypothesis as being the more probable. Some years ago Nageotte insisted that the essential lesion is to be found where the posterior root joins the anterior root before they reach the ganglion. This portion, which he calls "*le nerf radicaire*," is surrounded by a sheath of the arachnoid, and, according to him, in suitable cases it may be shown that the changes commence in the subarachnoid tissue around this portion of the root; they consist in the proliferation of granulation tissue, which, partly by interfering with the nutrition of the nerve fibres, partly by mechanical constriction of them,

leads to their degeneration. Nageotte's findings have been lately confirmed by Richter, who has in addition demonstrated the presence of spirochaetes in this arachnoid funnel.

Whether this theory of Nageotte and Richter ultimately proves to be true or not, there can be no doubt that the essential lesion of the disease is a degeneration of the posterior roots, and consequently the most constant and characteristic symptoms should be disturbances in the functions of the afferent systems. The knee-jerks and other reflexes depend on the integrity of this, and they are consequently usually absent in the advanced stages of the disease. But this is by no means so in the early stages, and in many cases they certainly persist for years after unequivocal signs have appeared. Further, Romberg's sign is by no means constant, especially in the early part of the disease when diagnosis is most important. Even the pupillary reflexes are often normal or the reactions are anomalous, and in quite a large proportion of all cases gait is never affected. We thus find that all the cardinal signs on which the ordinary textbooks of medicine lay so much stress may, like the clinical pathologist, leave us in the lurch.

When, however, we turn to the examination of sensation we find, as we might expect from the pathological site of the disease, that there are very few if any cases in any stage in which there are no sensory changes. But a complete examination of sensation is extremely difficult and time-consuming, and there are but few competent either by training or experience to carry it out. Further, the character and form of the sensory disturbances vary enormously from case to case. We must therefore for clinical purposes select tests that are simple, reliable, and practicable. And this is luckily possible, for the most constant and characteristic changes are in sensibility to cutaneous pain.

When sensation is tested with the point of a sharp steel pin or needle widespread disturbances are usually found even in the early stages of the disease. But even this simple test requires some care, as the insertion of a pin-point evokes, in addition to pain, an idea of sharpness by which the stimulus can be often recognized in analgesic areas, and unintelligent patients may consequently make replies that confuse the observer. This difficulty is obviated by placing the point of the pin against the tip of the observer's index or middle finger and pressing with the finger on the skin as the pin penetrates it; then the patient can be asked to reply whether each contact is only a touch, or if there is a prick as well.

The earliest loss of sensibility to pin-prick is usually found on the lower extremities; it is most marked on their distal segments, and diminishes gradually in intensity towards the groins. It does not as a rule correspond to root distribution; it may, for instance, be slight or absent on the thighs and buttocks though considerable in degree in the legs, which are innervated by the same sacral and lumbar roots. Sometimes, however, it follows the lines of the cutaneous root areas.

At first the loss is only partial—either the pain evoked by the stimulus is less than a prick produces elsewhere, or more commonly only a certain proportion of pricks of moderate intensity, but sufficient to cause pain in normal persons, are recognized. As the disease advances this hypalgesia becomes more intense. Not uncommonly patients appreciate no discomfort from a pin-prick on any portion of the lower limbs, or only in the upper thighs, but before this stage is reached certain anomalies of sensation are often present. There may be a delay in the perception of pain, or the prick excites abnormal sensations, as burning or tingling, or the pain persists unnaturally, so that the patient commonly attempts to "rub away the feeling."

Even more common than disturbance of pain sensibility on the legs is the zone of analgesia around the trunk, to which Laehr first drew attention. The upper margin of this zone generally lies at the level of the second ribs in front where the areas of the cervical and dorsal roots meet; it may extend downwards only a few inches or as far as the groins. As a rule the hypalgesia is most intense between the second ribs and the nipples, while the zone around the trunk immediately above the groins and buttocks is that which generally escapes longest. The loss of pain sensibility in this trunk zone is usually more profound than on the legs. When it is only partial it can be easily demonstrated by making a series of pricks from below upwards towards the clavicles or on to the neck.

Another common area of analgesia is the ulnar borders of the arms, but this usually appears later in the disease. It is variable in extent, and may be represented by a narrow

band in the inner border of the forearm, or may comprise the whole ulnar half of the limb, including the two or three ulnar fingers. Here too, as on the legs, the analgesia is rarely complete, and is often represented only by a relative insensibility as compared with the radial borders of the arms.

Duchenne observed many years ago that the face is occasionally analgesic in tabes, or rather that portion of it which is supplied by the trigeminal nerve. He spoke of this trigeminal loss as "*le masque tabétique*," but a complete mask is rare. Some diminution of sensibility to pin-prick is, however, extremely common on an irregular area in the centre of the face, especially on the nose and the neighbouring portion of the cheeks. It is an important symptom, as sensory loss of this distribution is not found in any other nervous disease, except perhaps leprosy. It is easily demonstrated, for probably on no part of the body is the prick of a sharp pin so painful as on the nose and adjoining portions of the cheeks, and it is exactly here that sensibility is often lost or diminished in tabes. The area affected may vary from a small patch on the nose to the greater part of the face, until indeed it forms a typical tabetic mask. If slight it may be apparent only when the sensibility is contrasted with that of a normal area, as on the neck.

The diminution, loss, or alteration of sensibility to pin-prick in some or all of these areas—that is, the distal portions of the lower limbs, the thoracic zone, the radial borders of the arms, and the central portion of the face—constitutes the most common, the most definite, and the most characteristic physical sign of tabes dorsalis. It is frequently present when the knee-jerks and the pupillary reactions are normal and before ataxia or other symptoms develop, and it is consequently a most important phenomenon in the diagnosis of the disease. The loss of sensation in these areas is a dissociated one—that is, its different modalities are affected in different degrees, or some forms only may be disturbed. Touch, for instance, is frequently intact on the chest and face at least, and the affection of thermal sensibility is inconstant and irregular. Its diagnostic value is enhanced by the fact that in no other disease is there dissociated analgesia of a similar distribution.

Though I lay so much weight on the presence of this cutaneous analgesia as a diagnostic sign owing to its constancy and the simplicity with which it can be demonstrated, I do not wish to underestimate the importance of other sensory disturbances. Tactile sensibility is rarely profoundly affected, but is often reduced, especially on the legs. Thermal sensibility is more irregular, and as the sensations evoked by the contact of hot and cold test objects may be perverted rather than reduced, its investigation is too difficult for ordinary clinical purposes. On the other hand, though changes in so-called deep sensation—that is, the sense of position and movement, the recognition of size, shape, and weight, and the appreciation of vibration—are certainly the most important in the pathogenesis of the symptoms of the disease, their investigation requires more care and skill, and disturbances in them are much less constant in the slighter cases and in the early stages.

Few diseases are so protean and irregular in their manifestations; few, if any, present so many symptoms that may lead us into error in diagnosis and prognosis, and few require in their early stages so much care in investigation and treatment. I have endeavoured to put before you some of the pitfalls that await us, for consciousness of danger is our surest safeguard, and to point out the simplest means by which, in my experience, we may avoid mistakes.

THE question of indirect advertising has been occupying the General Council of the Bar. The annual report for 1922 states that the attention of the Council was called to an article, which appeared with sensational headings in a Sunday newspaper, purporting to be written by a leading counsel. When asked for an explanation, this gentleman stated that the managers of the paper had pressed him to see a reporter and to give him his views on a legal subject. After several refusals the counsel ultimately acceded to the request, warning the reporter that the interview was not to be used in any way that might be regarded as advertising himself. The interviewer apparently worked up the conversation into an article, which appeared under the name of the counsel as if it had been actually written by him. Eventually the managers of the paper expressed regret for what had occurred. The General Council of the Bar, with a view to avoiding possible misunderstanding in the future, has passed the following resolution: "That it is contrary to the etiquette of the Bar for a practising barrister to give an interview to a representative of the press on any matter in which he has been or is engaged as counsel."

SOME APPLICATIONS OF PHYSIOLOGY TO MEDICINE.

I.—SENSORY PHENOMENA ASSOCIATED WITH DEFECTIVE BLOOD SUPPLY TO WORKING MUSCLES.

BY

J. A. MACWILLIAM, M.D., F.R.S.,

PROFESSOR OF PHYSIOLOGY,

AND

W. J. WEBSTER, M.B.,

ASSISTANT IN PHYSIOLOGY, IN THE UNIVERSITY OF ABERDEEN.

(From the Physiological Laboratory.)

ACCURATE knowledge of the effects of defective blood supply to the various tissues and organs is obviously of great importance in view of the innumerable conditions of stress, derangement, and disease in which this factor comes into play, with manifold results in the way of disturbed or impaired functions in the different systems of the body. "Defective supply" naturally covers different conditions—quantitative deficiency in normal constituents, or the presence of abnormal and injurious constituents, or inadequacy as regards the volume, pressure, and rapidity of flow of normal blood. This communication deals with the last-named—certain effects of deficiency in the supply of normal blood to normal muscles.

Many impairments of functional activity from more or less extensive interference with blood supply have long been known, such as the weakening of the heart muscle from deficient coronary supply and the common occurrence of fibrillation after sudden coronary obstruction; the effects on the brain in the form of giddiness, faintness, or loss of consciousness; and the primarily exciting and secondarily depressing influences exercised powerfully on the medulla (respiratory, vasomotor, and cardio-inhibitory centres, etc.) and on the spinal centres from sufficiently extensive or sudden acute lack of blood supply; also the derangement or stoppage of kidney function from similar interference.

Various observations are on record dealing with the functional behaviour of excised organs and muscles artificially perfused with blood or in the exsanguine condition, and also observations on the effects of artificial interference with the blood supply of organs and muscles *in situ* in animal experiments. Under such conditions there is of course no information obtainable as to sensory phenomena attendant on altered blood supply in the conditions of rest and activity.

The present inquiry deals with the behaviour of human muscles temporarily deprived of their blood supply while their normal innervation remains intact; the sensory phenomena recognizable in the states of rest and activity are examined and brought into relation with other functional conditions, such as changes in contractile power, etc.

Methods of Experiment.

The forearm was investigated (a) while the normal circulation was going on, and (b) when the blood supply was stopped, the limb either retaining its blood in a stationary condition or being rendered exsanguine before the circulation was arrested—that is, the "congested arm" and the "ischaemic" arm were examined with arrested circulation. The circulation was stopped by a blood pressure armlet applied to the upper arm, which was rapidly pumped up to a constricting pressure much above what was necessary to produce arterial obliteration in the particular individual examined—that is, an armlet pressure largely exceeding the systolic pressure. When this was done in the usual way, as for the measurement of systolic blood pressure, a "congested arm" was obtained containing a large amount of stationary blood shut off from the general circulation, the veins becoming prominent and tense. To obtain the bloodless or ischaemic arm an elastic bandage was first applied to the hand and arm, and removed after the armlet had been pumped up as described.

In the congested arm the sensory phenomena are naturally complex, being partly attributable to conditions attendant on the arrest of the circulation as influencing the muscles, etc., and partly to the discomfort caused by the venous turgescence. To avoid the latter complication the method of the ischaemic arm is employed; the sensations induced by muscular activity in presence of acute want of blood can then be examined.

Under these conditions muscular action was tested in various ways. Graphic records of the flexor muscle of the