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**Contributors**

Williams, Tom A. 1870-  
Royal College of Surgeons of England

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The Importance of Early Diagnosis of Tabes  
and Cerebro-Spinal Lues

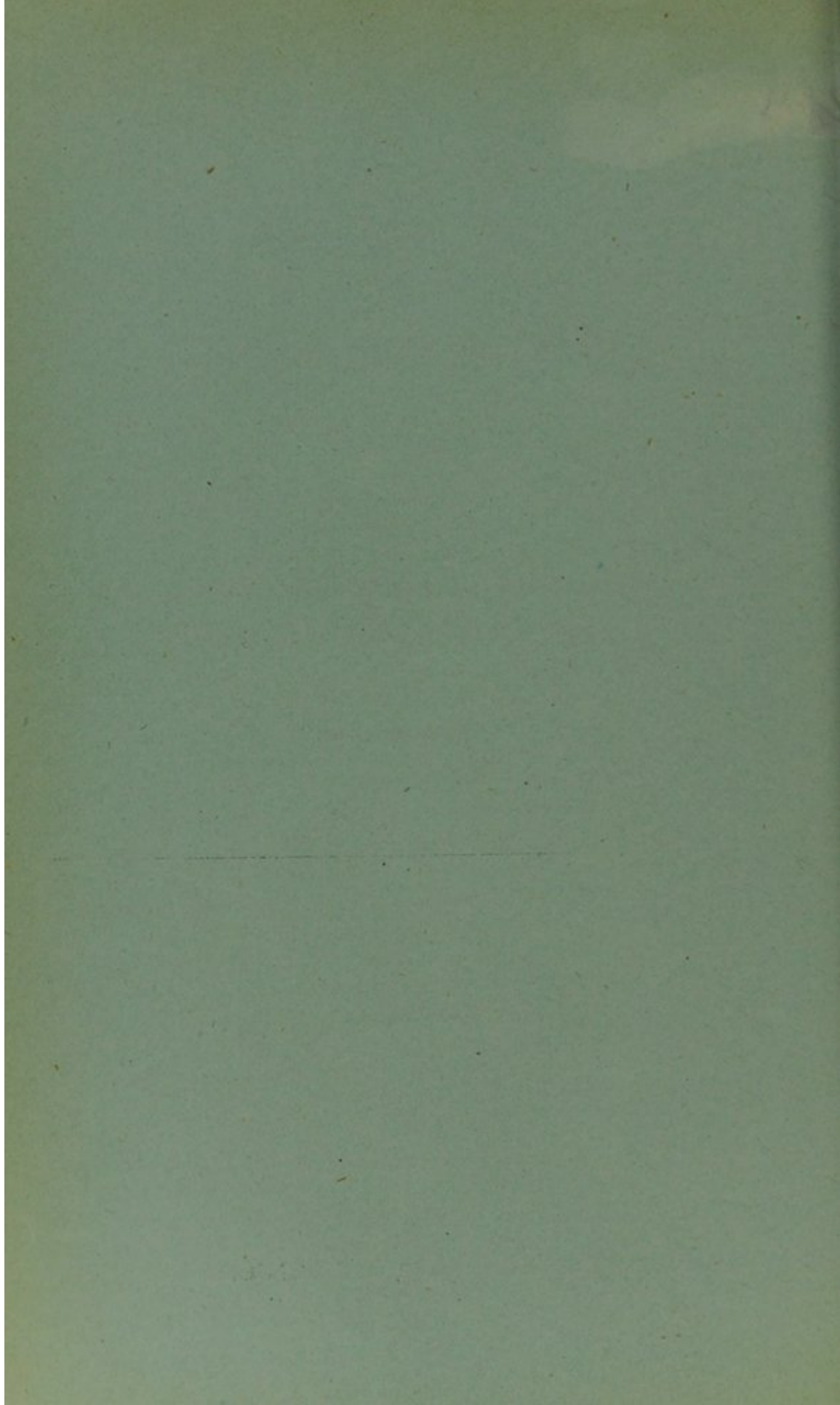
By

**TOM A. WILLIAMS**

Washington, D. C.

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## THE IMPORTANCE OF EARLY DIAGNOSIS OF TABES AND CEREBRO-SPINAL LUES

By TOM A. WILLIAMS

Washington, D. C.

Now that the troponema pallida and the Wassermann reaction are accepted facts, there is less excuse than ever for loss of time in beginning the treatment of organic disease of the central nervous system. Moreover, it is now unpardonable to experiment with mercurial treatment because of a remote chance that the patient's symptoms may arise from syphilis; for in the non-syphilitic diseases of the nervous system, early diagnosis and appropriate treatment are just as important, and often as successful as in the former.

Surgeons have been particularly guilty in this respect; but there is no longer the faintest justification for the common procedure of permitting a spinal or cerebral tumor to grow for ten weeks while mercury is tried; for not only can the clinical neurologist in most cases diagnose this condition without assistance, but the laboratory affords irrefragable proof whether or not the lesion is specific.

However, before applying to the patient so complicated and difficult a laboratory test as is even its simplification by Noguchi, it is as well to ascertain whether the clinical signs justify the essaying of the test.

I do not refer to such obvious changes as the loss of the knee jerk and locomotor ataxia and reflex iridoplegia; for it is surely the aim of modern medicine to anticipate, and in doing so to prevent supervention of such gross and irreparable defects. It thus becomes necessary to suspect, even if not to diagnose, tabes dorsalis long before the cardinal signs have shown themselves.

What then are the important prodromata? Of these the modifications of the sensibility usually precede others. These are generally at first subjective, although they ultimately manifest themselves also to physical examination. They are frequently labeled "rheumatic" for years when they are painful; but their tabetic nature should be suspected when they predominate in the lower limbs, when they are aggravated towards night, and when they intermit, apparently irrespective of external influences. Moreover, they are very frequently accompanied by paresthesiæ, which, although at first intermittent like the pains, become more and more constant. Prurigo, burning, dull numbness, tingling, formication may each and all in turn dominate.

But in these respects the picture to an extent resembles that caused by the early stages of irritation of the peripheral nerves so often seen in alcoholics. In this condition, however, it is unusual for the arms to escape very long, whereas in tabes they may remain free for many years or entirely. Moreover, tenderness on movement and pressure is a marked symptom in the peripheral affection; while when the inflammation affects the nerve roots, as in tabes, peripheral stimuli do not cause pain. Indeed, analgesia on deep pressure is one of the earliest sensory modifications of tabetics. It is due to the interruption of the functions of the fibers of deep sensibility. The end organs of these are the muscle spindles and corresponding organs in tendon and aponeurosis. They travel towards the spinal cord along with the motor nerves, comprising about a third of the bulk of these in some situations. As they approach the intervertebral ganglia, however, they leave the motor nerve, and in common with other sensory nerves, traverse the ganglion, their trophic center; and finally form part of the posterior root. It is here that they are attacked by the tabetic process, which is pathologically a specific spinal meningitis, generally ingravescens and exceedingly slow.

Convenient clinical signs to elicit this are pressure on the ulnar nerve, tendon Achilles or testicle, or faradization of the muscles of the calf. If these manœuvres do not cause tenderness, we can be sure that the afferent tract is affected.

Naturally, all sensory fibers may be affected in turn, and indeed generally are if the disease progresses. By appropriate treatment, however, and sometimes spontaneously, and perhaps by natural immunizing power, the process clears up before very many fibers are de-

stroyed even though their function may for a time be interfered with. In this case, function is resumed; but once destruction occurs, function cannot return; for though anatomical regeneration from the ganglion cells does occur, yet the fibers cannot grow beyond the reflection of the pia mater, where they enter the spinal cord; because at that point they lose the neurilemma. Hence, although post mortem the posterior root is full of regenerated fibers, which often end in small neuromata like those in amputation stumps, yet within the spinal cord one finds a relative absence of nerve fibers in the posterior columns. This, however, is not a sclerosis, as it has been called, but an atrophy with shrinkage.

As the process, for an unknown reason, usually begins in the lowest roots, the implication of the organic reflexes is an early symptom. The cause of urinary retention lies in the anesthesia of the bladder, which prevents its distension from being felt. The incontinence is an overflow symptom; but before this stage is reached, irritative symptoms usually show themselves, and these are frequently diagnosed as cystitis. The absence of bacteria or of an unusual number of bladder epithelia should, however, make the clinician strongly suspect a cause other than bladder inflammation to account for the hyperirritability these patients show; and a careful examination of the general sensibility should be undertaken.

The tuning fork is a most important means of investigating the sensibility in the early stages of tabes. The loss of its appreciation in the distribution of one or more posterior roots should always be looked for. Nerves which can convey stimuli at the speed needed to appreciate touches may not be able to do so quickly enough to appreciate intervals of vibration of  $1/12$  of a second; and in this case no tingling is felt.

The implication of the posterior roots again leads to the exaltation of sexual powers, followed by impotence; and this is often the earliest symptom of tabes.

A sudden giving way of the lower limbs may arise from either of two causes. One is the interruption in the posterior roots of the impulses which subserve the unconscious sense of attitudes. The messages as to their position being transmitted imperfectly, the motor impulses are not adapted to the real attitude of the limbs, and the patient may fall. The other cause for this symptom is the implica-

tion in the meningitic exudates of the anterior roots, from which cause the motor impulses themselves fail to reach their destination. This interruption is usually quite temporary; but if the inflammation is severe, muscular atrophy may supervene, although even this is not irremediable; for as resolution and absorption take place, the fibers regenerate from their trophic center in the anterior horn, possessing as they do a neurilemma. This is why the disability from muscular atrophy is much less serious in tabes than that due to interruptions in the afferent path, although at least twenty per cent. of tabetics show some muscular atrophy, and in some few cases it dominates the picture, and is probably the cause of some cases described as Aran-Duchenne palsy.

Any of the foregoing symptoms may present itself before the deep reflexes are modified; but on the other hand, the latter may show some impairment while the sensory symptoms are still exceedingly *fruste*. As the process usually begins low, it is to the most caudal reflexes that we must look for early confirmation of our suspicions. Loss of the knee jerks is a late sign compared to the loss of the Achilles and bulbo-cavernosus reflexes. The former is elicited by making the patient kneel far forward in a soft chair, seeing that the sural muscles are well relaxed, and when the feet hang quite loose, tapping the tendon Achilles gently with a percussion hammer. Normally, a brisk plantar extension of the foot occurs; but if the afferent path is interrupted on one side, the response is feeble and less extensive; and this finding may be the earliest sign of the tabetic process. Of course, if one or both reflexes are absent, organic disease may be postulated, though not necessarily tabes; for in multiple neuritis, etc., the same loss occurs.

The bulbo-cavernosus reflex is elicited by placing the left forefinger at the root of the penis behind the membranous urethra and stabbing the glans gently, though sharply, with a pin. Normally the left forefinger will feel as a brisk twitch the contraction of the bulbo-cavernosus muscle at the root of the penis. If this is absent, organic trouble may be postulated.

A diminution of the power of appreciating attitudes is often one of the earliest objective signs of posterior root implication. It is ascertained with the patient's eyes closed or bandaged while he is sitting or lying relaxed at ease. The limb to be tested is held in

both the observers' hands, which are separated as widely as possible from the joint to be tested, another joint intervening where practicable. Several manipulations are made and the limb finally held stationary and free from contact with clothing or surrounding objects. The patient is then asked to name the position of his joint. Those who cannot do this accurately, and few patients can, are asked to imitate with the corresponding limb the position in which they believe the observer has placed the limb he is testing. Many patients will try, by moving the muscles, to feel the position of the joint tested. This maneuver vitiates the test, which is one of passive position and not of sense of movement; and the greatest precaution must be observed to prevent these almost involuntary movements. In patients of low intelligence with poor control, the test is almost impracticable; but the incapacity to detect passive position of one's joints is a most important early sign when tabes is suspected.

Another important, though sometimes less early sign, is hypotonia. The most practical test for this is to lay the patient flat on a hard bed or the floor, and placing one hand on the knee, with the other gently lift the foot from the floor. Normally, this can hardly be done; but in proportion as the muscles are hypotonic, the interval to which one can lift the heel from the floor is perceptible.

But the foregoing signs are all more or less relative. A sign which is almost pathognomonic is the reflex iridoplegia, known as the Argyll Robertson pupil phenomenon. To detect this in its early stages, the patient should be taken into a dark room and a light of one candle power placed at his side. Between this and the eye to be tested is held a card at least large enough to completely obstruct the light. With the patient looking into the distance, towards, but not at, the light, the card is then lowered; as soon as the light impinges on the retina a normal pupil contracts briskly. In cases of asthenia, it may redilate to a certain degree after a few moments; but if the response is sluggish or absent, we can practically affirm that we are dealing with the results of a meningitis of specific character; provided, of course, that we eliminate cases of opacities of the media, functional incapacity of the optic nerve or its relay centers in the quadrigeminal region, and lastly, total or partial internal ophthalmoplegia. Another method of examining for this abnormality is



by suddenly shining an electric light into one eye while observing the pupil of the other while it is accommodated for far vision. Satisfactory examination cannot be made while the eyes are accommodated or converged to a near object; for this act itself causes the pupils to contract.

Long before the Argyll Robertson pupil appears, the edges of the pupil may be irregular. If any doubt is felt, a mydriatic may render this more clear, or examination with the lens may reveal atrophic areas at the edge of the iris. These pupillary reactions are almost pathognomonic. A rare early symptom is that of seeing better in the twilight than in full daylight; this is due to the contractibility of the pupil being interfered with in the more unusual position of dilatation, the more usual state in tabes being a myosis.

The final test, if the Wassermann reaction is unavailable, is the examination of the spinal fluid obtained by lumbar puncture. This is performed by inserting an aseptic long thin trocar into the fourth lumbar space (which is opposite the highest point of the iliac crests) slightly to one side of the midline, and slowly withdrawing not more than 10 c.c. of fluid. This is centrifugated for 10 minutes, and a scraping from the bottom of the tube is transferred to a slide by a pipette. Methylene blue renders the detection of lymphocytes easy; and if these occur in greater number than about three in each field of the microscope with a magnification of 300 or more, meningeal irritation must be suspected. This becomes a certainty in proportion as the number is increased; and there can be no doubt of it when the average reaches 8 and upwards.

This observation, in conjunction with any of the foregoing symptoms or with a diplopia due to paralytic strabismus, facial palsy, laryngeal cough and crises of vomiting, tenesmus, nervous diarrhea, or chronic aortitis, renders practically certain (even without access to the Wassermann reaction) a diagnosis of the pathological process which is at the bottom of tabes dorsalis.

This pathological process is a syphilitic one, contrary to a common belief that it is a dystrophy of the sensory neurone. It should accordingly be susceptible to specific treatment; and with certain reserve this is indeed the case, and many examples are being reported of arrest of tabetic processes by frequent and intensive mercurial treatment, especially after intramuscular injection.