I wish to thank the Council of the Newcastle-upon-Tyne and Northern Counties Medical Society,[2] Professor Rutherford Morison, and Dr. W. K. Russell, for permission to include Cases V and VI.

REFERENCES.

[1] BRITISH MEDICAL JOURNAL, April 2nd, 1921. [2] *Newcastle and Northern Counties Medical Journal*, April, 1921.

A CASE OF SLIPPING PERONEAL TENDONS TREATED BY KELLY'S OPERATION.

BY

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W. B. F., aged 22, was admitted to the Ministry of Pensions Hospital, Streatham Hall, on July 10th, 1921, complaining of pain and weakness in the right ankle, and inability to walk any distance.

History.

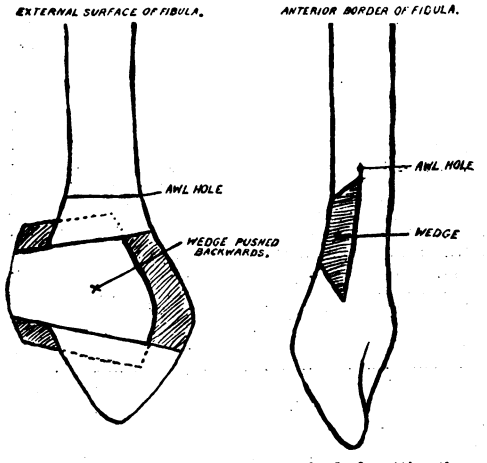
He stated that while at physical training in June, 1918, he fell and twisted his ankle rather badly. The injury was diagnosed as a sprain, and he was sent to hospital. Nothing definite was discovered on *x*-ray examination, and his leg was put on a back splint for five weeks. After this he was given massage for six weeks, followed by a stay of two months in a convalescent camp. He was demobilized in January, 1919. For some time after this his ankle swelled at night, and his leg and foot were put in plaster for seven weeks, but, after the plaster was removed and he was treated by massage and exercises, there was still some swelling of the ankle at night, and, on extreme flexion or extension of the foot at the ankle-joint, something slipped on the outer side of the joint, giving rise to much pain locally and a jarring sensation up his leg and body.

Condition on Admission.

On admission the movements at the ankle-joint were good, and there were no signs of any disease of the joint, but on dorsiflexion a distinct slipping of the peroneal tendons could be felt. A skiagram did not show any damage to the ankle-joint. An operation for retaining the peroneal tendons in their normal position was necessary, and I decided to perform that described by Mr. R. E. Kelly in the *British Journal of Surgery* for April, 1920.

Operation.

On January 26th, 1921, I made a hockey-stick-shaped incision immediately behind the fibula, commencing at a point about 2½ inches above the lower extremity of the bone, and following the course of the tendons downwards. The incision was deepened and the periosteum covering the outer surface of the fibula was incised and stripped, so as to expose the outer surface of the bone and its anterior and posterior borders. The peroneal tendons with their synovial sheath were retracted with the periosteum stripped from the posterior border. At a point about 1½ inches from the lower end of the fibula on its anterior border, and midway between its external and internal surfaces, a hole was bored horizontally backwards through the bone with a small bone awl. This hole was enlarged downwards and parallel to the external surface with a Gigli saw, and when the cut was large enough to admit the point of a Hunter-Mackenzie saw, this instrument was used, until the cut reached a point about half an inch from the extremity of the bone. Two cuts were now made in a horizontal direction but approaching one another posteriorly to join the vertical cut, so as to free a wedge-shaped piece of bone with its narrow end at the posterior border of the fibula. The saw was sloped while making these cuts, so that the external surface of the wedge was narrower than the internal surface. The wedge thus formed could only be removed from the bone by sliding it forwards, and when it was pushed backwards and tapped gently it became fixed in the groove made by the saw cuts, with about a quarter of an inch projecting from the posterior border of the bone, thus making an external lip to the peroneal groove. It was found, however, on attempting to bring the superficial tissues together over the bone, that there was a tendency for the wedge to slide forward in its groove, but this was easily prevented by raising the floor of the groove slightly in front of the wedge with a chisel.



Diagrams illustrating the method of cutting the wedge. The wedge is shown wider than is necessary. The dotted lines indicate parts of the wedge in the slot in the bone.

After the wound had been closed, the foot was put in plaster, with a window over the incision, in a position of plantar flexion.

Result.

The plaster was removed one month after the operation, and recovery was uneventful. The movements at the ankle-joint were perfect, and no slipping of the tendons could be obtained. The patient states that he is cured, and that he has none of his previous difficulties in walking.

A skiagram taken six months after the operation showed the wedge firmly fixed in the position in which it was placed.

The main difficulty in the operation is, as pointed out by Kelly, the thickness of the saw. Allowance has to be made for this in the inclination of the horizontal cuts to one another, so as to avoid too great projection of the posterior part of the wedge when driven into position. A small fretsaw, such as is used by watchmakers, would be an excellent tool for this operation, and I propose to use it in future cases. It is also rather difficult to avoid injuring the synovial sheath of the peroneal tendons, and I accidentally made a small opening into it in this case, but no harm resulted.

THE INTERPRETATION OF SYMPTOMS IN DISEASE OF THE CENTRAL NERVOUS SYSTEM.

ABSTRACT OF THE GOULSTONIAN LECTURES, DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS,

BY

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LECTURE III.

IN this final lecture Dr. Feiling said that he proposed to deal with some of the more general rather than the particular aspects of disease of the central nervous system. Any discussion on the interpretation of symptoms could not fail to take account of the principle laid down by that master mind of modern neurological theory and practice, Hughlings Jackson, who distinguished between negative and positive symptoms, a negative symptom implying some definite loss of function, and a positive some accelerated function. The actual symptom-complex produced by disease provided, more frequently than not, both positive and negative symptoms, destructive lesions producing the latter, and irritative lesions the former. The term "irritative," as applied to a pathological lesion, must not be interpreted too narrowly, in the sense of a mere mechanical irritation. Brief or temporary circulatory disturbances might be included in such a category, and the convulsions or deliriums occurring in various states of toxaemia were to be regarded as positive symptoms produced through the medium of the circulation. It must be understood also that when a destructive lesion occurred in the nervous system it did not follow that the ensuing disorder of function was necessarily due to that destructive lesion itself; it might be due, wholly or in part, to the unbalanced action of other parts of the nervous system.

Negative and Positive Symptoms.

The same morbid process might produce at one and the same time negative and positive symptoms. The early stages of syphilis of the central nervous system, for example, might be marked by pain—a positive symptom—while later on negative symptoms in the shape of palsies made their appearance. A more difficult question to decide was whether pain and other subjective sensory disturbances could occur as the result of purely destructive lesions, for this would seem to imply that positive symptoms could be produced by such lesions.

The lecturer did not wish to suggest that in investigating any disease of the nervous system it was necessary to set out by classifying symptoms as negative and positive. It was only necessary to realize that there were these two kinds of symptoms, and from such realization one was led to consider the nervous system as a whole. The consideration of these principles had its value when one was confronted with the task of localizing a lesion in the central nervous system. It was notorious how misleading in such cases the signs might

be. A palsy of the sixth cranial nerve, occurring as a late event in intracranial tumour, furnished an illustration of a late sign which frequently had little or no localizing value.

Disorders of the Cerebral Circulation.

In the two previous lectures he had been concerned principally with symptoms arising from disease of the central nervous system itself; but it was a matter of everyday experience that many of the symptoms of which patients complained were to be interpreted as disorders of function produced by temporary or recurring disturbances in circulation. Such disturbances might be quantitative, as anaemia or hyperaemia, or qualitative, in which there was some chemical or biochemical alteration in the blood itself. Symptoms referable to the nervous system formed a not inconsiderable part of nearly all forms of cardio-vascular disease, but there were many other conditions in which disorders of the cerebral circulation played a leading part. One of the previous Goulstonian lecturers (Dr. Risien Russell, in 1909) had dealt fully with disorders of the cerebral circulation, so that it was not necessary for him to cover the same ground. It was well known how epileptiform convulsions, quite indistinguishable from those of ordinary epilepsy, might occur in many toxic states—eclampsia, lead poisoning, and so forth—and typical epileptiform convulsions might be seen in cases of heart-block constituting the Stokes-Adams syndrome. There seemed to be some common ground between all these different conditions where the immediate or proximate cause of the convulsion could be fixed. Such cause, to his mind, lay in some disorder of the cerebral circulation, and on this point he was in absolute agreement with Russell as well as with many others.

It became a principle of some importance, therefore, to regard a fit as a symptom only, and not as a disease *sui generis*. Mr. Percy Sargent, in his presidential address to the Neurological Section of the Royal Society of Medicine, had related that in a series of 270 cases of brain tumour operated on by him a fit was the first symptom complained of in 40 per cent. This was probably an unusually high proportion, for there were many cases of cerebral tumour in which fits never occurred at all, and, in addition, many cases of cerebral tumour which never found their way into the hands of a surgeon. The mere presence of cerebral tumour was not of itself sufficient to cause a fit. It was the same with injuries; one might hazard the assumption that there were probably a great many people nowadays with foreign bodies embedded in their skulls who did not suffer at all from epilepsy.

The Cause of Epilepsy.

The textbook descriptions of the fits of epilepsy and of hysteria led the reader to suppose that there was a great distinction between them. In practice such distinction was not always evident, and increasing experience had taught the lecturer the great difficulty of making a diagnosis in some of the periodic attacks of apparent loss of consciousness which he was called upon to treat. It was, of course, well recognized that many epileptic attacks might be precipitated by various psychical conditions or emotional stresses. Recently an attempt had been made to include epilepsy among the group of diseases or symptoms which might be said to have a psychogenic origin. Such an explanation might seem at first sight fantastic, but there were certainly cases where attacks similar to those of idiopathic epilepsy had yielded to psychological treatment. There was another school which would include epilepsy among the group of so-called toxic idiopathies, thus bringing it into line with such conditions as asthma, hay fever, and urticaria; and here also there were cases which seemed to support this view. Whatever might be the exact physical production of epilepsy, the final cause must, to his mind, lie in a disturbance of the cerebral circulation.

It was only in comparatively recent years that the involuntary nervous system had received much attention at the hands of clinicians, who were just beginning to realize the wide part which it played in the production of symptoms. It would be wise to recognize that multiple causes might be at work. Perhaps observers were too much occupied with symptoms in attempting to correlate disturbances of function with actual structural disease. Even in cases where actual structural disease of the central nervous system was present, *post-mortem* examination would not always succeed in connecting symptoms with the actual morbid anatomy.

"Organic" and "Functional."

In conclusion the lecturer spoke on the use and abuse of the terms "organic" and "functional" as commonly employed in connexion with disease of the nervous system. In investigating cases of disease of the central nervous system it was in reality disturbances of function which were studied rather than alterations in structure produced by disease. Hence the continued use of the terms "organic" and "functional" might be misleading. The term "functional" had gradually become identified with the broad meaning of "hysterical," and on looking more closely into the results produced by this abuse of the term it would be found that it was diseases in which permanent changes in the reflexes occurred which were called organic, and that unless such changes could be demonstrated there was a great risk that the case would be called functional. No doubt many of these cases were really functional, but the special connotation which this term had come to possess made its application deceptive. The distinction between organic and functional, although at first useful, had ceased to serve its purpose. The necessary distinction was between symptoms caused by physical influences and those caused by mental influences only. Physical changes might be permanent—such as gross destruction of nerve fibres; or they might be temporary—such as circulatory defects or alterations in the volume or composition of the blood. Similarly with mental or psychical factors: these might induce bodily reactions which persisted and led to permanent changes in bodily functions; yet no one would speak of a disease like diabetes as functional. It was illogical, he maintained, to identify the term "functional" with the term "hysterical." The term "functional" could very well be dispensed with altogether, for all diseases of the central nervous system were functional in the sense that a disorder of function was produced. What it was really necessary to know was whether or not a disease was due to permanent structural change in the tissues, and, if it was, whether the cause was physical or mental, or both. The attempt to draw a hard-and-fast line between organic and functional nervous disease was fraught with no little danger. Equally misleading was the idea that there must be two classes of neurologists, one to deal with organic and the other with functional cases. It was to be hoped that no such distinction would be recognized. The nervous system functioned as a whole, and should be studied as a whole.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

HYPERPYREXIA DURING INFLUENZA.

G. C., a school teacher, aged 42, was some years before this recorded illness an in-patient at Bath Hospital suffering from haematemesis (and subsequent thrombosis of the veins of the leg), and during her seven months in hospital is stated to have registered a temperature of 110° F.

During the month of March, 1919, she complained of "pains across the shoulders, back of the head, and neck." Examination revealed nothing save a slightly furred tongue and an axillary temperature of 107° F. On advising the relatives to have a consultant's opinion, in view of the hyperpyrexia, they expressed little or no anxiety, as she had "had a higher temperature before at Bath."

Dr. Carey Coombs saw the case in consultation on the following day. There was no evidence of any organic disease. The axillary temperature registered 110° F. It was taken by me personally, using two thermometers, one in each axilla. The thermometers were both graduated to 113° F., and appeared capable of registering not more than 114.5° F. That same evening the thermometers registered 113° F. +, which fact I communicated to Dr. Carey Coombs. The patient at this time complained simply of headache. On the third day the temperature fell steadily to the region of 104° to 105° F., and remained so for several days, only to rise about the fourteenth day again to 113° F. +. Though the columns of mercury filled, in both cases, the capillary to the utmost limit, neither thermometer was broken as the result of the patient's hyperpyrexia.

After an interval of some two or three weeks a third rise, terminating as before by lysis, was registered.

After fourteen weeks' treatment at home (during which time the patient lost several stones in weight) I was sent for