learnt to expect to find a Babinski sign present for a few hours or even a day or two. Others have found it present also after coma from alcohol, or anaesthetics, or in saturnine encephalopathy. An excellent article by Dr. Hawthorne (published in the *Practitioner* for September, 1914) states that the sign is also sometimes found after diphtheria and other infections, after some drugs, such as strychnine and hyoscine, and with rheumatoid knees.

Let us now see if any useful deductions can be made from the fact that a transient Babinski sign occurs in various transient toxic functional affections of the nervous system. As an illustration we may take an elderly uraemic patient of mine who developed uraemic hemiplegia, and in whom the autopsy showed no gross disease of the brain. He had a well-marked Babinski sign. Contrast this with a case of hemiplegia due to hysteria. In both cases the hemiplegia is due to functional disease; one shows a Babinski sign, the other does not. Since we know that Babinski's sign is due to some blockage in the pyramidal tract, surely the inference is irresistible that hysterical hemiplegia is due, not to any deficiency in the pyramidal tract, but to loss of function in some higher levels of the brain. The only alternative to this conclusion, so far as I can see, is the highly improbable assumption that Babinski's sign in these multifarious toxic diseases is due to a selective action of their toxins for the pyramidal tract.

Although our inference (that hysterical hemiplegia is peculiar in being due to loss of function in levels of the brain above the pyramidal tract) is in keeping with our general notions of the nature of hysteria, yet I do not know of any evidence which in hysterical paralysis so clearly exculpates the upper motor neurons and so neatly incriminates the higher levels of the brain as does this deduction which I have just submitted to you for your criticism. If any one should reply that my thesis is already in a vague way embodied in our current views of hysteria, I would reply by quoting the definition of hysteria in the opening sentence of the chapter on hysteria in the most recent neurological textbook I have (Judson Bury, 1912, p. 716): He says "hysteria is a psychical disorder which often leads to disturbance of the lower centres of the brain and spinal cord and of the sympathetic system. . . . The exact nature of the cortical disturbance is unknown." My thesis asserts that the lower centres of the brain and cord are entirely unaffected in hysteria, and are as innocent as the peripheral nerves.

For the sake of clearness I have so far only spoken of hysterical or uraemic hemiplegia. But obviously my thesis applies equally to hysterical or uraemic paraplegia and monoplegias, or indeed to any hysterical paralysis of a cerebral type. And, after all, every hysterical paralysis is of the cerebral type, and is never ascribable to loss of function in a peripheral muscle or nerve.

But let us concentrate for a moment on hysterical paraplegia. You may object that paraplegia should, at any rate, be considered a disease of the cord rather than of the brain, and that this, therefore, condemns my thesis that hysterical paralysis is always cerebral in origin and superior in level to the pyramidal tract. But surely this habit of thought is only excusable in dealing with organic lesions, in which case one is correctly averse to explaining a paraplegia by two large symmetrical cerebral lesions when a single small lesion in the cord accounts for the paraplegia. Not so, however, in hysterical paralysis, where experience shows that the brain is just as readily affected symmetrically as unilaterally. So that hysterical paraplegia becomes no obstacle to my thesis if it is regarded as a symmetrical cerebral monoplegia of the legs, which I submit is the proper way to regard it.

Next, to speak of Babinski's sign after fits, hysterical and otherwise. A plausible explanation of Babinski's sign is that it is in some way an effect of lessened inhibitory control normally exerted by the upper motor neuron. If this be so, one may reasonably deduce that the transient Babinski sign after fits, etc., is in the same way an expression of diminished inhibitory control, but due in this instance to temporary exhaustion or poisoning of the upper motor neuron. Now, I have never seen Babinski's sign present after a hysterical fit, but I have learnt to expect to find it after fits due to epilepsy, uraemia, or eclampsia. Hence this sign is a useful guide as to whether or no a fit has been hysterical, provided one can observe this sign before it has passed off. This may be turned to practical account, especially in these days of the Workmen's Compensation Act, as a means of showing whether a fit has been genuine epilepsy or has been hysterical or malingered.

Next, as to the occasional presence of this sign in infantile paralysis. This I can bear witness to, and it shows that the myelitic process is not always confined to the anterior horns in this disease, but sometimes invades the white matter of the cord, or at any rate of the pyramidal tract.

Next, I have often noticed an apparent tendency for the flexor and extensor response to occur simultaneously, leading to a rivalry between the flexor and extensor muscles, and hence to a very equivocal Babinski sign. If I can see the tendon of the big toe pulling, even if the stronger flexors do not allow an upward movement of the big toe, I consider that the response is extensor. The distinction is easier when there is a normal flexor response on the other side with which to compare the equivocal response.

For three years I have been watching a very curious case in which I only obtained a Babinski sign twice. On numerous other occasions the soles were cold or unresponsive. She is a middle-aged, very nervous lady, with a doubtful diagnosis of disseminated sclerosis, characterized by a weakness and ataxy of the legs, tachycardia, and distressing "vasomotor storms" in the chest and abdomen. For years she has had to take small doses of bromide. Can any one tell me if the plantar reflex or Babinski's sign can be abolished by habitual bromide? Or could there be, in addition to the sclerosis, some variable spasm in the vessels of the cord which would account for the coming and going of her Babinski sign? It is suggestive of spasm that nitroglycerine and digitalis gave her great relief.

Finally, in a book on massage, Kellgren (*Technic of Ling's System*, 1890, p. 54) says: "In sclerosed conditions of the crossed pyramidal tract frictional massage given on the internal plantar nerve with a moderate amount of energy will, after a latent period of about half a second's duration, cause involuntary extension of the toes; if given more energetically, involuntary flexion of the hips occurs, accompanied, if the patient be in the half-lying position, by passive flexion of the knee-joint." Cyriax asserts that this sign may be present in organic disease when Babinski's sign is absent.

I record this observation because it is practically the same as Babinski's sign, but it was published eight years earlier. And since books on massage are so rarely read by medical men, the observation might probably escape attention.

To sum up my main thesis: We know from dissections of those dying with organic nervous diseases that Babinski's sign is due to a lesion in the pyramidal tract. Babinski's sign also occurs in certain diseases, such as uraemic hemiplegia, which is too transient to be organic; so that in this disease the hemiplegia must be due to a transient functional paralysis of the pyramidal tract. Yet in *functional* hemiplegia due to *hysteria* Babinski's sign is never present.

It follows, therefore, that hemiplegia in hysteria is due to loss of function, not in the pyramidal tract, but in the neurons at some higher level in the brain. And this reasoning as to hysterical hemiplegia can be extended to other hysterical paralyses.

NOTE.

OVER the paper of Mr. E. P. Cathcart, on The Rôle of Carbohydrates in Nutrition, published at page 503 of our issue for September 19th, should have appeared the word "abstract." A similar notification might, in fact, properly have been introduced into the headings of all communications to the Sections at the Aberdeen Meeting hitherto published, for, with the courteous co-operation of their authors, all have been materially abridged.

ERRATUM.

IN the report of the discussion on anoci-association the word "Plymouth" instead of "London" was accidentally inserted after the name of Mr. C. A. Pannett, of the Royal Free Hospital, when recording his remarks on page 353 of our issue for August 22nd.

A PSYCHOPATHIC annexe to the Los Angeles Hospital in California, with accommodation for one hundred patients, has recently been opened. The cost was £35,000.