

forego some margin of their advantages for the sake of a great public benefit which the precise determination of a drug's value undoubtedly is. But the physician, in the usual practise of his art, must, like St. Paul, be all things to all men, that by any means he may save some; he must vary and change and combine the powers he employs, so that when the patient is cured he may often be unable to say which drug, or part of his treatment, has had the most curative effect."

We have thus set before our readers some of Dr. Bucknill's views as to the treatment of insanity, especially the additions made in the present edition. The whole chapter, however, should be read. His experience of private, public, and official lunacy is so extended that his opinions on this subject are necessarily of the greatest weight and value.

CLINICAL NOTES AND CASES.

Two Cases of Intra-cranial Syphilis. By J. HUGHLINGS JACKSON, M.D., F.R.C.P., Physician to the London Hospital, and to the Hospital for the Epileptic and Paralysed.

CASE 1.—There was, in the following case, no history of syphilis, but the appearances *post mortem* seem to me to warrant the diagnosis. It may be asserted that the severe blow on the man's head was not merely, as I suppose, the determining cause of intra-cranial syphilis, but that it alone, without the predisposing help of syphilitic taint, led to the changes discovered *post mortem*. The nodules in the testis, however, were, I think, pretty conclusive evidence that all the blow did was to determine syphilis locally. The cicatrices in the liver were more than merely suggestive. Further, the disease of the cerebral arteries was quite like that seen in cases in which there is such evidence as nodes to demonstrate the existence of syphilis. The first attack of hemiplegia, if not the second, was due doubtless to local softening consequent on thrombosis of a syphilitic artery. Such an indirect mode of production of paralysis in syphilitic cases must always be carefully considered. It has been described in this country by Bristowe, Wilks, Moxon, Broadbent, Buzzard, and myself. I have recorded several cases.—"Lond. Hosp. Rep.," Vol. 4, 1868.

Valuable evidence of syphilis is in the clinical course of the case. *A random Association, or a random Succession of*

nervous symptoms, is very strong warrant for the diagnosis of syphilitic disease of the Nervous System. The case illustrates this well, as does also the second case.

A soldier, 30 years of age, was sent to the Hospital for the Epileptic and Paralysed by Dr. Jeffrey Marston, October, 1867. There was *complete* paralysis of both third nerves of five months' duration. According to the patient, these palsies came on *suddenly* one morning when he was out walking. When admitted there was nothing more than palsies of these nerves. His optic discs were normal; he was healthy-looking; took his food well, and went out daily. In any case of palsy of any cranial nerve the important matter is to search for syphilis. The patient denied having ever had syphilis, and no signs of its past ravages were discovered. But the testes were not examined. This should be done in doubtful cases; in this case, *post mortem*, two nodules of "deposit" were found in one of the testes. There was a history of a severe blow on the head *two years before* admission. He was one day unmanageable from drink, and, therefore, to use his own words, "they were obliged to stun him." He suffered very little afterwards from the blow, so far as he knew, and kept well until four months before the palsies came on—nine months before admission. During this four months he had much pain in the head from temple to temple.

Plainly there could be no certainty in the diagnosis of the case in this stage. There could be only the suspicion of syphilis. The fact that the palsies were very symmetrical is not, as would seem at first glance, in favour of syphilis, but is rather against it. Palsies of cranial nerves from syphilis are *not* usually symmetrical (Optic neuritis or atrophy following it, is not included in the expression "paralysis of a cranial nerve"). Mr. Hutchinson believes that tertiary syphilitic symptoms are mostly unsymmetrical. The history of a severe blow was not evidence against syphilitic disease, for syphilitic disease of the brain frequently follows blows on the head—is, so to speak, lighted up by injury.

On the mere suspicion of syphilis large doses of the iodide of potassium were given. The continuous current was used by Mr. Netten Radcliffe. Under this double treatment the paralysis of the left third nerve diminished slowly to a slight extent; the pupil on that side became smaller. But the improvement was of no practical value. The patient was as much incapacitated as before; in order to see he had to use his frontal muscles and throw his head back.

The ill success of anti-syphilitic treatment did not negative the suspicion of syphilis. After five months' paralysis benefit could scarcely be expected from drugs. No one would expect iodide of potassium to remove old syphilitic "lymph" blocking up a pupil. Indeed, during the treatment the patient had a new style of symptoms; he had, on Sept. 23, two convulsive seizures, in which he was insen-

sible. He quickly recovered from each of these, seeming just as before. As nothing was heard of the *kind* of convulsion we learned nothing of much value to further the diagnosis. A general convulsion—or what is practically the same to us, a convulsion about which we get no details—is of no great value in diagnosis. A general convulsion from tumour or from uræmia may be quite indistinguishable from the convulsion of so-called idiopathic epilepsy. The occurrence of convulsions was in this case of some value, however, for it was *another* nervous symptom very different from the former. A paralysis of the third nerve of necessity points to disease affecting the nerve *trunk*, or the crus close to the implantation of the nerve; a convulsion is due to discharge of some nerve *centre*.

On the morning of January 5 the patient was found paralysed on the right side, face, arm, and leg; there was the “common form” of hemiplegia. Sensation was slightly diminished. His speech was unaffected. There had been no convulsion in the night so far as was known, and it probably would have been noticed, at all events had it been severe. The hemiplegia was too persistent to be the result of a convulsion; it was not the epileptic hemiplegia of Dr. Todd. It was, no doubt, owing to local softening from thrombosis of a syphilitic artery. Although the patient could speak, and was quite conscious, he was obtuse or apathetic. I now felt more confident as to the nature of the case. For now we had had three *different* symptoms—palsies of cranial nerves, convulsive seizure, and hemiplegia, which last was almost certainly an independent symptom, that is, not the result of a convulsion.*

He afterwards left his bed, and used to sit by the fire, but the hemiplegia remained. His appetite kept good until about a fortnight before his death. But after the attack his mental condition deteriorated. He became more and more listless. He occasionally wandered in his talk (his speech, in the strict sense of the word speech, being unaffected), but could always pull himself together to reply to simple questions about his case. On March 21 he had permanently settled in his bed, and on that day he did not know his wife. The optic discs were still normal.

On the morning of April 7th he was found to be paralysed on the left side. His mouth was a little turned to the right side, and from this time he never moved his left arm and leg. He had had some

* It is not uncommon for paralysis to follow convulsions; as I believe the paralysis is *produced by the discharge* in the convulsion. I use the expression “result of a convulsion” in the text. The after effects of strong epileptic discharges deserve careful consideration. I believe that in epileptic mania, and in so-called “masked” epilepsy, the highest nervous processes are put *hors de combat* by a strong nervous discharge, just as the corpus striatum is in epileptic hemiplegia. On this view the raving in epileptic mania is not owing to the epileptic discharge; it begins when that discharge is over, and results from uncontrolled action of processes more automatic than those temporarily paralysed by the discharge.

kind of fit, no doubt a convulsion, as his mouth, lips, and gums were bloody. I could not get a look at his tongue, although he seemed to make efforts to put it out when I asked him; although he seemed conscious, he never spoke afterwards; he lay like an inert mass, and gradually became comatose. It is to be observed that both pupils became small and equal before he died. He died April 11.

Autopsy.—There were two yellowish-white—wash-leather like—nodules in one testis. In the liver were several puckered cicatrices, which I suppose to have been the result of past syphilitic disease, but there were no nodules like those in the testes—no decisive evidence in the liver of syphilitic disease.

Examination of the Brain—The Arteries from back to front.—The right vertebral artery seemed to be healthy until about a quarter of an inch before it joined its fellow to make the basilar, where it began to swell—so to speak—to three times its diameter, and this condition was carried on so as to involve the basilar itself for about one-fifth of an inch. The swelling was of the usual gummatous stuff, but more friable than is common. The left vertebral was similarly thickened, and to it were firmly welded, by yellowish gummatous matter, the rootlets of the ninth nerve. The trunk of the left anterior cerebellar artery ran back parallel to the left vertebral, and was glued to it, and also to the sixth nerve. The anterior fifth of the basilar was here and there lumpy, and at its division it was much thickened, flattened, and whitish. Both posterior cerebral arteries, and both superior cerebellar and the two third nerves were all fixed to one another by material similar to that thickening the arteries. The left posterior communicating artery had three nodules on it. Through the arachnoid over the inner end of the right fissure of Sylvius was seen a yellowish mass the size of a horse-bean. The right middle cerebral artery was enlarged and nodose, and it and all its principal branches were firmly welded together. But on cutting up these vessels I found no plugs in their channels. The left middle cerebral artery was much thickened, but was quite pervious; its branches were a little nodose here and there. The anterior cerebral arteries soon after entering their fissures were thickened and glued together. They were both blocked up.

The Brain Substance.—The convolutions of the frontal lobes were healthy. The floor of the fissure of Sylvius was imperfectly softened for about an inch from its commencement. The *right* corpus striatum was slightly depressed on its inner and anterior part, and below this part there was softening of the size of a hazel nut. The *left* corpus striatum was softened in the lower and external part, about midway from front to back (the frontal convolutions were not affected), and the lower part of the thalamus was slightly softened. There was no disease in the other parts of the encephalon.

In the following case, also, there was no direct evidence of syphilis; there was indirect evidence in the state of the

patient's daughter ; she presented signs pointed out by Mr. Hutchinson as indicative of inherited syphilis. The post-mortem examination, to say nothing of the "Random Succession of Symptoms," is decisive.

I would here remark that, as this case illustrates, double optic neuritis frequently exists when there is no evidence to show that sight is affected, and indeed when there is clear evidence that it is good.

I have asserted this over and over again, but it is a thing hard to believe. Besides other reasons, the importance of recognising this is that we shall often discover optic neuritis too late for successful treatment—too late, I mean, for the *prevention* of amaurosis—unless we examine the eyes by routine. If a patient has any kind of nervous symptoms, especially pain in the head, we must not wait until his sight begins to fail ; we should use the ophthalmoscope by routine. If we do we shall discover optic neuritis in its pre-amaurotic stage. Another thing to be mentioned is that optic neuritis from syphilitic disease in the brain differs in no way from optic neuritis the result of a glioma or other "foreign body." Optic neuritis tells us nothing more than that there is coarse organic disease of some kind within the cranium. Its diagnostic value is the same whether sight be affected or not. There is no difference in the optic neuritis, whether the tumour or other foreign body causing it be in the cerebrum or cerebellum. It is of no value whatever in Localising beyond that it points to disease within the cranium.

Joseph Mx., aged 45, was first seen by me in the London Hospital, October 15, 1866. He was then lying in bed, as if half stupefied ; probably, in part, from pain in the head, which seemed to be intense. He kept crying out, "Oh ! my head !" and paid little attention to any questions. His speech, however, when he did talk, was quite good ; there was no paralysis anywhere. There were no obvious symptoms, except the pain in the head and the great apathy. However, I examined his eyes by routine, and found that he had double optic neuritis. There was no evidence that his sight was affected, but it was only possible to test it in the very roughest manner.

From the intense pain, and from the double optic neuritis, I could only infer that there was *some* kind of adventitious product in some part of the encephalon. As there was no paralysis, no localising symptom, it was impossible to fix the seat of this adventitious product, except, perhaps, negatively ; that as there was no paralysis, it was very unlikely to be of the motor tract. Nor was there evidence as to its particular nature ; for any kind of "foreign body" (glioma, syphiloma, abscess, hydatid cyst, &c.), would produce severe headache and double

optic neuritis. To anticipate, when recovered from his acute illness he denied having ever had syphilis. Nor did the history which his friends gave help us. Three weeks before, when selling fruit in the streets, he fell, and cut his head; he was brought home, managing to walk. He had "no senses" until next morning; until that time his wife could not tell what he said. He then told her that he had had a bad fit, but that it occurred after the injury to his head in the fall. He continued, however, to be in a "stupefied state," and at one part of the first week passed his motions under him. As such was his mental condition, it would not do to trust his account of the onset of his symptoms. He had during this week severe pains in the head, and—but only on one day—a few minutes' vomiting of "slimy" stuff. His only previous ailment had been "rheumatics"—possibly pains from syphilis.

Supposing the patient had had a convulsion, either before or after the fall mentioned, it would have furnished no evidence whatever as to either the seat or as to the nature of the lesion. If I had heard that the fit affected solely or almost solely one side of the body, I could have inferred that the adventitious product—which, be it observed, the severe pains in the head and the double optic neuritis declared to exist—~~was~~ of the cerebral hemisphere on the side opposite the side of the body convulsed or most convulsed. Moreover, this would have been *empirical* evidence as to the nature of the disease, because convulsions of this kind in association with double optic neuritis do point, in *most cases*, to *syphilitic** disease of convolutions—to coarse gummatous "deposits." The convulsion alone, or the optic neuritis alone, has not that value.

However, the only evidence I could obtain bearing clearly on the particular *nature* of the disease was that the patient's wife had had three children, of whom one was born dead, another lived half an hour, and the other was still living. But neglecting this, and it was, of course, only suggestive, there was nothing to show that the patient's illness was not owing to glioma, which might be in either the cerebrum or cerebellum. There was only evidence of "coarse" intracranial disease of some kind.

Nevertheless, I decided to give large doses of iodide of potassium; not because I knew the man was suffering from syphilis, but because there was a chance that his symptoms might be owing to syphilis. I have not only acknowledged that I did not know, but I have tried to shew that I *could* not know that the adventitious product was syphilitic. I repeat, the acute illness the man had was just like that which glioma or any other growth or mass might have produced.

I gave him large doses of iodide of potassium three times a day. He improved with marvellous rapidity, and in about a week was apparently well. The edges of the disc were ill defined, the discs were reddened,

* I am glad to find that Dr. Buzzard agrees with me in thinking that this association (of optic neuritis with one-sided convulsion) is "an important diagnostic feature." See his lately published work on "Syphilitic Affections of the Nervous System."

and the veins were tortuous. His sight was good. His sense of smell was unaffected. He went out apparently in good health.

It may be said that now, at least, I should be convinced that the patient's intra-cranial disease was syphilitic. That he recovered so rapidly after the use of syphilitic remedies was strong evidence, and the only evidence. The rapid recovery—not the recovery merely—would be, for instance, the only evidence rendering it worth while to publish the case as one of “recovery from syphilitic disease of the brain.” It is quite a theoretical conclusion to suppose that patients do not recover temporarily from such symptoms as this patient had, even when produced by cerebral tumour of a non-syphilitic kind—a remark only likely to appear strange to those who do not bear in mind that a large tumour may exist in the cerebrum when there are no obvious symptoms of any kind whatever.

Another matter is that had we been certain that his illness was owing to syphilis—had there been a node on the head, and a large gap in his palate to declare it—we could only say that the patient had got rid of his symptoms. We could not suppose that he was cured of syphilis, nor do I think we could conclude that the effused syphilitic material was absorbed. A patient who has recovered from a syphilitic affection of the nervous system is *very likely to suffer again*—not necessarily from the same kind of symptoms, but more likely from a new style of symptoms.

I have many times expressed the opinion that the therapeutics of syphilitic affections of the nervous system is not so triumphant as is often supposed. I do believe that symptoms pass off rapidly under the use of remedies, and have just given an example. But if we keep an eye on our patients we find them suffering again; we often “cure” them many times.*

In April, 1867, five months later, he came to the out-patient room, saying that he had had three fits during the month, and for one month pains in the head. But I learned nothing as to the *kind* of these seizures, and soon lost sight of the patient again.

But in July of that year I obtained indirect evidence bearing on his case. His only living child, a girl, 12 years of age, was brought to me for convulsive seizures, to which she had then been subject eight weeks. She had no warning, fell suddenly, was convulsed, did not bite her tongue. Now this girl had narrowed upper central incisors, and they were slightly notched. Mr. Hutchinson was kind enough to look at them,

* I have recorded a striking case of this kind “*Med. Times and Gazette*,” March 29, 1873. The patient was cured four or five times.

and declared them to be characteristic of congenital syphilis. She had nebulous corneæ, but this was doubtfully the result of past interstitial keratitis. The child's nose was sunken. She afterwards died of typhoid fever; no traces of visceral syphilis were discovered.

From this indirect evidence I conclude that the patient—the father I mean—had syphilitic disease of the brain.

On October 16, 1867, I found him in the hospital again. He was lying in bed, apathetic and miserable looking. There was palsy of the right side of the face and slight weakness of the *left* arm and leg. The facial palsy was not owing to affection of the trunk of the portio-dura nerve, nor of its nucleus. The paralysis was slight, but this would not shew it to be not due to affection of the trunk of the portio dura, for this nerve may be slightly affected. But the palsy was very unequally distributed. There was great palsy of the cheek, whilst the orbicularis palpebrarum was slightly affected. It was certain from this that there was a central lesion, but unfortunately no history was obtainable of the mode of onset of the hemiplegia. Had both the hemiplegia and the facial palsy occurred together we should have supposed there to be a lesion in the right side of the pons; if at different times, possibly two lesions—one of the left corpus striatum and one of the right corpus striatum or thalamus. Had the symptoms come on suddenly we should have inferred softening from thrombosis; if very slowly, a growth.

I gave him fifteen grains of iodide of potassium three times a day, but a week later he would leave the hospital. He was confused, scarcely knowing, except from minute to minute, what he did.

On Nov. 27th, he was brought back by his wife. He still had the paralysis mentioned, under date of Oct. 16, and now also palsy of the *left* third nerve; his general condition was about the same. Under iodide he again improved; the palsy of the levator palpebræ passed off, and the palsy of the recti diminished. The optic nerves—not examined by the direct method—looked normal.

He went out Feb. 11 (1868), but was brought back Feb. 18, for complete hemiplegia of the right side, and loss of speech. His wife said that at two o'clock that morning he asked for the chamber-pot, and seemed to have a "struggle" to get up to use it. He never spoke again, and at three o'clock he had some kind of seizure, possibly a convulsion, and was found to be paralysed and speechless. I saw him

February 22. The palsy of the left third nerve was as last noted. The hemiplegia was of the ordinary form, the face, arm, and leg being paralysed, as is usual, and *the head and eyes being turned to the left*. These two deviations point to a grave lesion. He was not insensible, but could not put out his tongue; yet as he could swallow well it was clear that his tongue was not paralysed—that is, not paralysed in the ordinary sense of the term. His appetite was fair; he could take meat. He passed his fæces and urine in bed. Gradually he became more and more apathetic, his appetite failed, he swallowed badly, and a bed sore formed. He died March 27, 1868.

Autopsy.—There were found two gummatous masses, each of about the size of a hazel nut, in the right posterior lobe; they affected the part lying on the tentorium, close to the petrous bone. There was much softening round about them. There was a small gumma, size of half a pea, on the surface of a convolution of the left parietal lobe. There was a focus of softening the size of a horse bean in the right (*sic.*) crus cerebri; there was a focus of softening, the size of a pea, in the right thalamus opticus. The corpora quadrigemina, pons Varolii, and medulla oblongata were healthy.

There were several small yellowish raised patches on the basilar artery, but the main arterial disease (gummatous, not atheromatous) was of the left middle cerebral artery. One of its main branches was nodose, running, as it were, through several lumps—gummatous thickenings of the outer coat. Its channel was not occluded. There was considerable softening, however, of the hinder part of the corpus striatum, and of the under and fore part of the thalamus. The frontal convolutions were not damaged.

Mania as a Symptom of Bright's Disease. By SAMUEL WILKS, M.D., F.R.S.

Having had under my care in Guy's Hospital several cases of Bright's disease in which maniacal symptoms have occurred, I wish to draw attention to the fact, in order to elicit information and opinions from others, as to the probability of this form of cerebral disturbance being a result of uræmia.

The most striking morbid phenomena referable to the nervous system in this disease are, as is well known, convulsions and coma. These brain symptoms were first accurately described by Addison in the Guy's Hospital Reports for 1839,