It was only with extreme care that the contracted bladder was safely opened on the point of a staff, and the calculus extracted.

All went well in regard to the operation, but on October 16th he had a severe attack of bronchitis and asthma, to which he had been subject for years. His asthma had long prevented sleep in a recumbent position, and an upright one in bed was necessary after the operation. He died exhausted on October 24th.

CASE XXIX.—C. R., aged 29. Had suffered for many years with symptoms of stone. He had been sounded frequently without result.

November 14th, 1888. Examined under ether, and a large and very flat oval calculus detected. The following day the suprapubic operation was performed. The calculus was composed of uric acid, and weighed one ounce and a third. His convalescence was somewhat prolonged, but ultimately he returned home perfectly well.

It will thus be seen that among the eleven cases of tumour only one was fatal, and that from pyemia. Among the eighteen calculous cases there were three deaths, if the fatal result in Case XXVIII is not included, in which death was clearly due to the severe attack of bronchitis and asthma, to which the patient had long been subject. The whole of these cases represent the most advanced and the most complicated examples of disease, and were on that account not amenable to lithotripsy, or to a perineal section of any kind.

NOTES ON ACQUIRED LEPROSY AS OBSERVED IN ENGLAND.

By JONATHAN HUTCHINSON, F.R.C.S., LL.D., F.R.S.,

Consulting Surgeon to the Hospital for Diseases of the Skin, to the Royal London Ophthalmic Hospital, and to the London Hospital.

II.

THE case with which I concluded my paper last week was of great importance as proving that the leprosy process (that is, the life and multiplication of the bacillus) may come to an end if the patient is removed to a place where the disease is not endemic. I have another yet to relate which is quite as definite, and I have, I believe, several patients under observation in whom the process of cure in England is slowly in progress.

CASE II.—The case to which I allude is that of a Methodist clergyman, of high endowments, who was born in Canada, and who, in 1850, at the age of 24, went on missionary service to reside in the Bermudas. Five years after going to the Bermudas he married, and, being then in excellent health, continued to reside there. In 1857, owing to some nervous failure, he became unable to write with his right hand, and in 1859 an eruption appeared. In 1863 he saw Dr. Brown-Séquard, who said that it was a confirmed case of leprosy with paralysis. During the next ten years he continued to suffer from the disease in an aggravated form. On one occasion, after an injury, he had an anæsthetic toe amputated. In 1875 his eyes began to suffer, and during the next five years he became quite blind after repeated attacks of inflammation. Several iridectomies were, I believe, done by a most skilful ophthalmic surgeon (Dr. Bullar, of Montreal), in the hope of arresting the disease, but without any permanent result. There are many interesting details in this case with which I forbear to trouble the reader. It is sufficient for my present purpose to say that the Rev. Mr. D. came under my observation about twenty-five years after the beginning of his leprosy, and that he was then, at the age of 55, an example of recovery. All leprous processes had come to an end. There had been no fresh manifestations for some years. He still enjoyed the use of a powerful intellect, but he was blind, much disfigured, and crippled in both hands and feet.

The case proves (1) that one not inheriting any taint may acquire leprosy by residing in a locality where it is endemic; (2) that such acquisition may be effected wholly without exposure to hardship or poverty; (3) that the malady may come to an end in the course of years during residence in a healthy district.

I do not see that it is possible to suggest more than two ways in which it is probable that the disease was acquired. Clearly Mr. D. in some way took in, while residing in the Bermudas, the specific germs of a specific malady. He must have taken them either by direct contagion, or he must have received them in food. A similar proposition may, I think, be made in reference to most of the cases with which I am at present dealing.

CASE III.—My third case is that of a young English lady who went to reside in Burmah in connection with a mission school. She went out at 19 and returned at 30 with her features so disfigured by "tubercular" leprosy that she looked like a woman of 60. She was for some months under my care in the London Hospital. It is now eight years since I saw her, and I do not know anything as to her present state.

CASE IV.—A woman, Mrs. C., was under the care of my colleague, Mr. Waren Tay, and presented a marked example of leprosy. Her age was 31 when I saw her. She had been taken to India at the age of 5, and had lived there twenty-two years. When she returned to England she was, as far as she knew, in sound health, and it was not till two years later that the signs of leprosy became definite. The first manifestation was the appearance of brown patches about the neck and shoulders, and when I saw her these brown areas covered almost the entire surface. I have not kept any record as to the precise part of India in which this woman had resided, but she told me that it was inland, and that there was abundance of fresh-water fish. She herself did not like the fish, but she believed that she had nevertheless partaken of it on an average two or three times a week.

CASE V.—A gentleman born in England, of English and Irish descent, named L. G. H., consulted me in 1880 for leprosy. He went to live at Bombay in 1856, and in 1875 his first patch was observed. It was a discoloration on his right leg. It was not until eighteen months later that his face was affected. The disease was in its seventh year when I saw him, and was well characterised. He said that in India he had eaten fish and prawns occasionally.

CASE VI.—A gentleman of Scotch descent consulted me on account of well-marked leprosy. He had been engaged for some years on the newspaper press of Calcutta, and there his disease had commenced. This patient had never been subjected to any kind of hardship, and he had not left England until he was 20 years of age.

CASE VII.—Colonel H. P. C. was born in England, of English parents. He went to India in 1849, and returned in 1869. In 1870 he consulted a London surgeon for an eruption which was diagnosed as syphilis, but in respect to which he denied that he had had any chancre, or been exposed to any risk of contagion. He was married, and had healthy children. In 1874 he consulted me, and he was then the subject of leprosy in an advanced form, his skin being almost universally discoloured, and of a brown tint. The lobes of his ears were thickened and dusky, and the soles of his feet painful. I saw Colonel C. several times during the next year, but do not know anything as to his subsequent history. He told me that whilst living in Burmah he had eaten fish moderately, and that he had been particularly fond of a condiment prepared from prawns, which he had special facilities for obtaining. This gentleman gave me interesting information as to the prevalence of leprosy in Burmah, with the facts concerning which he was officially well acquainted. He said that it was very common, and especially so among the coast population, and that the Government was at that time engaged in making extensive provision for the care of lepers. He drew my attention to a passage which had been recently published by Mr. Francis Day, and expressed his unbounded surprise that anyone who had travelled in Burmah could have written it. The passage to which I refer is as follows: "If, however, we look a little further to the east, we find the Burmese race, who are most assuredly a fish-consuming one, and who like their odoriferous nga-pee far better than fresh fish. Yet in my recent travels through Burmah I never saw an instance of leprosy amongst the indigenous population; neither did I see any cases of that congenital malformation of the fingers and toes so common among the natives of India born with a leprous taint. In fact, it has been frequently observed that this disease does not exist amongst the Burmese, and the same remark applies to the Andamanese, whilst it is as common in India amongst the inland classes who do not eat fish, as it is amongst those who consume it." I quote this passage as an example of the very misleading information

"On Fish as Food." By Surgeon Francis Day, F.L.S. and F.Z.S. *Indian Medical Gazette*, February 1st, 1871.

which the student of medical geography has occasionally to encounter. It is only one of many which have met me in my investigations as to leprosy.

CASE VIII.—A colonel in the army, aged 53, who had been born in England, but had lived twenty-seven years in different parts of India, Burmah, etc., became leprosy. The beginning of his disease was three years before I saw him, and occurred in the left foot. He was a man of splendid physique, of most temperate habits, and in excellent general health. Although both feet were numb, he had only about a year before I saw him engaged in athletic sports, and won a race. Both his ulnar nerves were affected, but on the right side the paralysis was only commencing. This gentleman had been exposed to the ordinary dietetic and climatic influences of India and Burmah, but had never suffered any hardship. He had eaten fish, etc., when he could get it.

CASE IX.—Colonel B. F., aged 45, consulted me first in May, 1879, having been then the subject of leprosy for about six years. He was of English parentage, but had been engaged in service in India for twenty-four years. He had been stationed chiefly on or near to the sea-coast, and he said that he thought he had rarely been a day without taking fish at dinner. He had been very fond of prawns and of prawn curry.

CASE X.—A gentleman, Mr. P., aged 48, had resided twenty-seven years in India. He had been born in England, and of English parents. He had lived in different parts of India, having been engaged for six years on the Ganges, and subsequently at Allahabad, Oude, and Scinde. He stated that he had been accustomed to eat fish whenever he could get it, and had been fond of prawns. His leprosy, which was very severe when I saw him in 1883, had commenced six or seven years prior to that date. There was a very interesting fact in this gentleman's family history. His wife, an English lady, had never suffered in any way, but one of his children, who had lived with him in India and was, I believe, born there, showed the disease at the age of 9. This child had returned to England a little before the symptoms had shown themselves. He was seen and the disease diagnosed by Sir Joseph Fayrer and Mr. Balmanno Squire. He subsequently returned to India, where he died of heat apoplexy. Having regard to the dates at which the disease appeared in father and son, and to the entire immunity of the mother, it would appear improbable that the child acquired the disease by inheritance. It would seem more likely that he and his father got it at the same time from one common source. This supposition is also supported by the fact—one to which I shall have to draw attention shortly—that it is not very uncommon for the children of English parents born in leprosy districts to acquire the disease while their parents remain free. It would seem that the bacillus develops with facility in the young.

Both the portraits which are given in the New Sydenham Society's *Atlas*—one showing the anæsthetic and the other the tubercular form of leprosy—are from patients with English parents who had acquired the disease in foreign parts. One of these (Case XI) a Scotch sea-captain, in vigorous health, had traded for many years to and from Barbadoes. He had usually stayed in the island about six weeks at a time. He had never suffered from any kind of hardship, and, on the contrary, had led what would seem to have been a most healthy kind of life. At about the age of 45 he became a leper. In the other case (No. XII) a woman who had been born in Ireland went to India as the wife of a soldier. She resided there for many years and in various parts, being employed chiefly, I believe, as a washerwoman. She eventually became the subject of leprosy in a severe form.

The twelve cases which I have quoted do not, I believe, comprise by any means all the examples of leprosy beginning in patients of British birth and descent which have come under my observation. They are all, however, of which I am able at present to find record in my notebooks.² One and the same criticism may be said to apply to all. They are examples of the acquisition of a specific disease by healthy persons who had no inherited predisposition. In no single instance had the person so acquiring it been exposed to any degree of hardship, or deviated in any definite manner from the ordinary conditions of a cleanly and well-regulated life. In every case the acquisition of the disease had occurred in some country where it was known to be prevalent, the East and the West Indies being the chief localities. I submit—as, indeed, I have already suggested—that there are

² In particular I well remember to have seen two or three soldiers who had become lepers in the Indian service, but of whose cases I cannot find any record.

only two suppositions open to us by which to explain the *de novo* acquisition of such a disease in cases such as these. The patients must either have received the specific contagium of leprosy on some part of the skin or mucous membrane, or they must have swallowed it in connection with food. Both these suppositions are possible. I may confess, however, that to my own mind one of them seems far more probable than the other. In no single instance had there been any known exposure to contagion. In no case had the patient associated with anyone suffering from leprosy, and in most instances the statement given was that they had but rarely seen lepers, and had certainly never come near them. All had, however, eaten the food common in leprosy districts; and if we incline to suspect that fish, and especially potted fish, is an ordinary vehicle for the contagion, all had undoubtedly been exposed to this risk.

Another and perhaps larger group of cases, very similar to those I have just quoted, consists of examples of leprosy beginning in persons of European blood, but born in leprosy districts. The evidence which these cases give is somewhat less conclusive than that of the group which we have been considering. Thus it is obvious that in some instances the parentage may have been mixed, and thus the disease may have been inherited. Where the admixture of native blood is but slight, and its evidences not very obvious, our patients are not always eager to divulge the truth, and may be willing to pass as of pure English descent when not really so. Again, it is possible that a child born in India may inherit something which has never as yet made itself manifest in his parents; there are also the perils of vaccination; and lastly, we have the fact that a child born in India, of English parents, may have been suckled by a native woman, and possibly by one having a taint of leprosy. I do not myself attach much importance to any one of those sources of fallacy, but it is right that they should be named. My belief is that the cases which will form the subject of my next series may most of them be placed side by side with those already quoted as being examples of leprosy acquired by Europeans as the result of residence in affected districts.

THE CROONIAN LECTURES

ON THE RELATIONSHIP BETWEEN

CHEMICAL STRUCTURE

AND

PHYSIOLOGICAL ACTION.

Delivered before the Royal College of Physicians of London.

By T. LAUDER BRUNTON, M.D., D.Sc. EDIN., LL.D. ABER., F.R.S.,

Fellow of the Royal College of Physicians; Assistant-Physician and Lecturer on Materia Medica at St. Bartholomew's Hospital.

LECTURE IV.

ANALGESICS.

Non-reception and Non-perception of Painful Stimuli.—The two classes of drugs which we have already considered, general anæsthetics and local anæsthetics, both abolish pain: the latter by acting upon the peripheral terminations of sensory nerves, so that they do not receive the painful stimuli; and the former upon the nerve centres, so that they do not perceive them.

Non-transmission of Painful Stimuli.—But pain will also be relieved if the conducting power of the sensory nerve trunks or of the sensory paths in the spinal cord be so altered that painful impressions can be no longer transmitted.

Action of Cocaine on Nerve Trunks.—Cocaine has this effect upon nerve trunks when locally applied, for, when injected subcutaneously near a nerve, it may produce anæsthesia in all the district to which that nerve is distributed.¹

Action of Cocaine on the Spinal Cord.—It has a similar action on the sensory tracts of the spinal cord, for Hughes Bennett² found that it rendered these tracts insusceptible to stimulation; and Mosso³ found that both conductivity and reflex excitability in the cord are destroyed by it before the nerve trunks, either sensory or motor, are paralysed.

Action of Cocaine on the Cerebral Cortex.—It lessens the irritability of the cerebral cortex when directly applied, for if a 4 per

¹ Hall and Halsted, *New York Medical Journ.*, Dec. 6th, 1884.

² Hughes Bennett, *Edin. Med. Journ.*, Oct., 1873.

³ Ugolino Mosso, *Arch. f. exp. Path. u. Pharm.*, vol. xxiii, p. 153.