NOTES ON OCULAR LEPROSY.

By ARTHUR NEVE, F.R.C.S.E.,
Joint Superintendent Kashmir State Leper Asylum.

THE eye is affected in 20 of the 80 lepers now in the Kashmir State Leper Asylum. As would be expected, the proportion is larger in the tubercular cases than in the anæsthetic. In tubercular cases the ocular affection is essentially one of infiltration, and in anæsthetic cases of injury from exposure. The extent and position of the lesion vary greatly. Among the present inmates the ocular manifestations may be classified as follows:

LIDS.

1. *The skin of the lids* is liable to the deposit of nodules just as that of other regions; these seldom attain any large size, and very seldom ulcerate. At present there are only 3 cases of marked tuberculous nodules.
2. *The margin of the lids* is occasionally affected, and there is atrophy of the ciliary follicles. In 3 cases this is complete, in 5 partial.
3. *Paralytic ectropion* is the characteristic eye lesion in anæsthetic leprosy. It is rather a drop of the lower lid than an eversion. It falls away from the eye and leaves it exposed. Owing to displacement of the lower punctum there is lachrymation. The exposed conjunctiva becomes irritated, and often there is ulceration of the lower edge of the cornea. At present there are 9 such cases in various stages.

CONJUNCTIVA.

1. *Conjunctivitis* frequently occurs in lepers, as in all Kashmiris, and there seems no special predisposition, except in those who have paralytic ectropion. Anæsthesia of the lids exposed to special danger from the bites of flies and mosquitos, and to injury by foreign bodies.
2. *Granular conjunctivitis* is only represented by 2 cases—a small proportion for this country.
3. *Leprous nodules* appear in the conjunctiva as elsewhere, but we only have 3 primary cases. On close inspection it is seen that the deposit has extended from the deeper tissues. *Pterygium* is occasionally seen; there is 1 case now, it is not specific.

CORNEA.

1. *Keratitis* sometimes occurs, but is not specific, being usually due to traumatism. From the same cause we have
2. *Ulceration*, which is most frequent in the lower limbus. This is seldom deep or actively septic, and under treatment, especially by temporary closure of the lids, it readily heals. There are 2 such cases now.
3. *Infiltration*, which commences peripherally, usually in the sclerotic margin, and gradually extends more or less circumferentially. At the outset there may be a little dotted deposit of a yellow colour, possibly vascular. More often it grows as a distinct nodule, involving the sclerotic, cornea, and iris simultaneously. There is a tendency for both eyes to be somewhat symmetrically affected. There are now 8 such cases in the asylum. I regard this as the characteristic lesion of tubercular leprosy. As stated by Hansen and Looft, the inner margin of the cornea is seldom affected, while a low rampart is formed round the rest of the cornea. We now have one typical case of this condition. The infiltration is usually superficial to Descemet's membrane, and may be dissected off it. In one case where this was done a staphylococcal protrusion has resulted. The infiltration consists partly of the bacilli, but chiefly of cells in a state of brown granular degeneration.

IRIS.

In ocular leprosy the iris is frequently attacked. This is especially the case when infiltration has commenced in the corneal margin. Nodules form in the angle of the anterior chamber, which becomes partially obliterated by them. Less often the nodule may be single, but seldom on or near the edge of the iris. The whole tissue of the iris becomes softened by a chronic inflammatory process, and the deposit of lymph tends to occlude the pupil, causing permanent blindness. When the infiltration begins in the lower angle it closely resembles a hypopyon.

LENS.

I do not remember seeing any case of cataract consecutive on ocular leprosy.

DEEP STRUCTURES.

Deep-seated eye affections are rare in leprosy, and are always secondary to corneo-scleral lesions. I doubt whether nodules ever occur on the retina or choroid primarily. I have seen cases of general infiltration of the eyeball which might be called pseudo-glioma, and atrophy of the globe is, of course, met with as the result of suppuration. The sight as tested in sixty lepers, illiterate, without visible ocular disease, was good. The optic nerve is seldom or never the seat of leprosy deposit, but the infiltration spreads to the ciliary nerves from the corneo-scleral region, as also to the ora serrata of the retina (Pollock). Blindness is not as frequent in Kashmir from leprosy as appears to be the case elsewhere. There are only 13 blind eyes; of these, 2 were caused by small-pox, 2 are due to total pannus consecutive on trichiasis, 2 to perforating ulcers, 2 to glaucoma, and the rest to tubercular deposit and occlusion of the pupil.

ACCIDENTS AND INJURIES CAUSED BY
LIGHTNING.*

By OSCAR DUNSCOMBE-HONIBALL, M.D.,
President of the British Guiana Branch.

AFTER some introductory remarks, Dr. Dunscombe-Honiball proceeded:

The literature on the subject of injuries caused by lightning is extremely scanty. Holmes, in his *System of Surgery*, writing on accidents from lightning, says they are very rare at home (about 44 per annum in England, Bowdin), and that it had never fallen to his lot to witness immediately on their occurrence any which called for serious surgical treatment, and he goes on to say that it does not appear to have occurred to any of his countrymen whose experience of this form of injury in tropical climates may have been great to give to the profession any systematic account of the matter. As I have in my more than thirty years' tropical practice seen many cases both fatal and otherwise, I may possibly be excused for taking this somewhat overlooked subject as the text for my address. I am well aware that very little practical result can be looked for as the outcome of an address and discussion of such a subject. Those who die from lightning stroke die with awful suddenness, and for those who recover little beyond stimulation and the usual surgical treatment for injuries received seem to be required. But I will ask your kind indulgence if I do not appear to treat the subject in a very scientific and speculative mode, but rather give a plain account of the appearances and symptoms I have met with in my experience of lightning stroke, both *post mortem* and in those who have not succumbed to this injury. I have not, I regret, kept an exact account of all the cases that have come under my notice, but, roughly speaking, I should say I have had experience of upwards of a score, here and elsewhere. Fortunately, however, I have very accurate and extensive notes, both *post mortem* and clinical, of the last 11 cases coming under my notice, which have occurred within the last half dozen years. Possibly the best plan for me to pursue will be for me to briefly detail these cases, give *post-mortem* appearances and clinical histories, and then make some comments in general on the nature of the injuries.

The first group of cases—I say group of cases because 10 were struck at the same time—to which I wish to draw your attention occurred on November 28th, 1894, on Plantation, Windsor Forest, West Coast, Demerara. A party of male East Indians, to the number of 15, had sought shelter from a severe rainstorm in a small brash hut; in a second, without any warning—I am using the words of one of those present—a sharp blaze of light seemed to strike in, over, and through the hut, accompanied by a terrific thunderclap. All present were blinded for a second or two, but when they had recovered from their first fright and shock it was found that four of their number were insensible (two, as a matter of fact, were dead), and six others injured more or less. They were all brought to the Estates Hospital, where I happened to be at the time. On examination I found two dead, one recovering from insensibility, one still insensible, and six

* Presidential Address of the British Guiana Branch of the British Medical Association.

others showing in varying degrees signs of shock. They (the latter six) appeared dazed, and presented spots, like wheals, on their bodies, in different situations, as though they had been sharply struck with a broad piece of leather, or like exaggerated and isolated spots of urticaria. These six required only temporary treatment, such as the exhibition of stimulants, and a confident assurance that they were all right; next day they appeared quite well, and the wheals had disappeared.

I will now describe the appearance and condition of the two men—Parmessur Sing and Adjoodiab—admitted into hospital. Parmessur Sing, when I examined him in the ward, had recovered sensibility, and was conscious; he was, however, nearly pulseless, suffering from intense shock, cold skin, cold tongue, dazed, and, I think, evidently not quite—or at once—understanding questions asked; his temperature, taken an hour after, was half a degree below normal; the pupils were dilated, and there was lividity of lips and nails; the heart sounds were scarcely audible. The only external injury he had was a fire blast or abrasion (the epidermis only was removed) down the outer aspect of the left arm. Under the use of stimulants, blankets, and hot water jars he rallied in a few hours. The next day, when I saw him, he was quite sensible and self-possessed, but had a frightened look and a peculiar slowness in answering; and I do not think he has ever quite lost the look of fear, although now it is an aspect more of anxiety. The fire blast or abrasion, which after a couple of days looked exactly like a burn in the first degree, was most difficult to heal, and he remained in hospital until December 31st. When discharged he still seemed wanting in rapidity of thought, taking an appreciable time before answering simple questions, and with an evident appearance of forcing himself to comprehend what was said to him; it looked, so to speak, as though the electric wires had been damaged. All along he complained of numbness in the left upper extremity and left side, and had lost some muscular power in the limb. I saw him quite recently, and he still suffers from loss of power, or, to use his own expression, he has never been as good a worker as before the accident.

As to Adjoodiab, who was insensible when I first saw him, he very soon became conscious, but seemed muddled or dazed to a much greater degree than Parmessur Sing. He was utterly depressed, with sighing respiration; pulse fairly good; temperature normal; a most marked expression of fright on his face; he had a slight wheal on the right thigh, which was gone the next day, but on the third day he complained of numbness in this situation, and has never lost this anæsthesia; his mental condition has also been permanently affected; he became irritable and childish, and, although the irritability passed off, he remains decidedly weak minded up to now.

The two men killed were Phakoo and Malhar Sing. Both had been two years in the colony, and were remarkably fine able-bodied men. The *post-mortem* examination was made twenty hours after death. Phakoo: Rigor mortis present and well marked; hæmorrhage from right ear, or, more correctly, the marks of hæmorrhage; perfectly calm and quiet expression of face. An irregular wound of right temple $1\frac{1}{2}$ inch in length, with slightly depressed fracture underneath. On removing the skullcap this fracture was found to pass through the base in the region of the middle fossa, breaking through the petrous bone and tearing the membranes; there was very little extravasation of blood; brain substance firm and normal, and also membranes beyond the zone of fracture; heart firmly contracted and empty. There was scorching or staining of the whole front of the chest, abdomen, and thighs, the marks on the chest being of a distinctly arborescent appearance.

Malhar Sing: Rigor mortis present; the eyes were staring, and there was a look scarcely of fright, more of surprise or astonishment. The only external marks were an abrasion or fire blast on left side of chest over cardiac region, with scorching or staining over left side and chest, and an abrasion below right knee; these abrasions were more like excoriations than anything else, the epidermis only being removed. The heart was empty and firmly contracted. The brain had been undoubtedly injured, its substance being of very much less firm consistency than usual; indeed, in parts almost (diffuent,

with scattered pinkish specks (*puncta sanguinosa*); other organs normal. In both these cases I noticed nothing unusual about the condition of the blood.

The next and last case to which I wish to refer occurred at La Grange, on West Bank, Demerara, on September 30th, 1897, during a heavy downpour of rain; and I may mention, in passing, that I have never known a case of lightning stroke to occur except during rain or immediately after, when the persons injured were wet and the surroundings humid. The patient was an East Indian man, named Salickram; he, with two other men, was walking about on the middle walk all within a distance of each other of from 1 to 2 feet; he had his agricultural fork, four prongs, over his right shoulder, with a tin saucepan slung on the handle. The overseer in charge was about 150 yards away, and from him I gathered these details. Without warning, a vivid flash of lightning, accompanied by deafening, simultaneous thunder, struck the group of three; they all fell down, but two immediately got up, and were not hurt; evidently, therefore, they fell from mere fright; the overseer says he saw smoke, and that the grass there was scorched. Salickram was found to be dead, and his death was instantaneous. I went back, and saw the body; the hat the deceased was wearing was scorched and torn to ribbons, and thrown some yards away; the tin saucepan was smashed in, and I found by means of a small key that the second prong of the fork, which he had over his shoulder, was highly magnetic; there was a ghastly, horrified look upon the face. I found a jagged, irregular wound at the back of the head, a little to the right side, 4 inches in breadth; under this I felt the bone fractured. Eight hours after death I made the *post-mortem* examination. Rigor mortis was present. The brain substance had lost its usual firm consistency, and was very soft and easily broken down. There were three fractures radiating from the right wing of occipital bone, one to vault of cranium, and two passing forwards and downwards to base of skull; no extravasation of blood. The heart cavities were empty and contracted, except the right auricle, which was partially dilated, and contained a little blood. In this case I noted an abnormal fluidity of blood. There was scorching of the right side of the neck and down the right side of the chest and arm; in some parts scorched the skin was like parchment.

I have no existent notes of my former experiences of lightning stroke to go upon, and could only draw upon my memory for any detailed accounts of them, which would be unsatisfactory. As to the oft-repeated statement—which, I think, originated with John Hunter—that there is an absence of rigidity in the cadaver, and also the constant presence of an abnormal fluidity of the blood, you will have seen that the *post-mortem* appearances I have described do not bear these out, but, on the contrary, offer a direct negative, and, although these points may seem slight and unimportant, yet I submit that any authenticated fact is well worth being recorded.

Dr. G. V. Poore says¹ that the effects produced by lightning differ only in degree from those produced by static electricity generated in the laboratory, and that by lightning a person may be killed outright and a *post-mortem* examination reveal no lesion whatever. Now, is this really so? and what are the grounds for the statement? Has an electric shock ever amputated a man's fingers, as I have seen in lightning stroke, or caused compound fractures of skull such as I have described? A case occurred here some little time ago where a stableman was killed by an electric shock. I think, Dr. Anderson made the *post-mortem* examination. I have endeavoured to obtain an account of the *post-mortem* appearances in death from electrocution, but unfortunately have failed. In no case of death from lightning which I have examined has there been a total absence of lesion. There may, it is true, be an absence of gross lesion of the brain, but there has always been some tangible injury. And in this regard I think it well to give one caution: Be not too sure that in every case found dead after a storm, and where no marks are found, that death was due to lightning stroke. A careful examination may show a condition of heart that would fully justify the assumption that death was due to cardiac failure induced by sudden and startling fright.

My own idea is that, although death from lightning stroke is nearly allied to and closely resembles death by electricity,

yet there is, on the one hand, something added, and on the other something absent. Of course this is purely my opinion, and must be taken for what it is worth.

REFERENCE.
¹ Quain's Dictionary of Medicine.

MEMORANDA: MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, ETC.

HÆMATURIA AS A CAUSE OF MISCARRIAGE.

On November 19th, 1898, I saw Mrs. R., aged 32, on account of profuse hæmaturia, with sharp pain in the right lumbar region, which had been continuous for over a week. On examination I found a six months' pregnancy, accompanied with extreme tenderness over the right kidney (unfortunately the enlarged womb prevented my observing the size of the organ. Her temperature was normal, there was no tenderness over the bladder, no frequency or pain on micturition, the amount of urine passed being normal, the blood being intimately mixed with it; neither fragments of tissue nor pus cells were discovered on microscopic examination.

This profuse hæmaturia continued unaffected by treatment until December 8th, on which date, as the patient was rapidly sinking—her anæmia having reached an extreme degree—I decided to induce labour, and by emptying the womb relieve the renal congestion, which I thought was a possible cause of the hæmaturia.

This I accomplished by Krauz's method; the child, a male, survived its birth only twenty minutes. The patient, on recovering from the shock of the operation, made an uninterrupted recovery, and on the seventh day following her confinement was absolutely free from all pain in the lumbar region, the urine was clear, and no trace of albumen was present; from this time she made a rapid recovery.

On December 5th, 1899, I was again called to see her, and found the womb enlarged to the level of the umbilicus, with pain in the right lumbar region and slight hæmaturia, which had been continuous for three days. The hæmaturia gradually increased in severity until January 7th (just five weeks from its commencement), when labour set in, and she was delivered of a female child, apparently of seven months' growth. As in the previous labour, with the emptying of the womb, all the symptoms disappeared, and she made an uneventful recovery. I have since made a most careful examination, and I can detect nothing abnormal in the size or position of either kidney, and the urine is absolutely normal. She enjoys exceptionally good health.

I am inclined to regard the hæmaturia as due to congestion of the kidney, from pressure on the renal vein by the enlarged womb, but why the tenderness and pain should have been confined to the right side I have been unable to decide.

If any member can suggest any possible treatment, so as to avert a recurrence of the hæmaturia in any subsequent pregnancy, I should be most grateful.

Manningtree, Essex.

J. S. JAMESON, M.D. (Dub. Univ.).

THE CONSTITUENTS OF HAFKINE'S ANTIPLAGUE VACCINE.

I HAVE carried out several experiments in order to find out the real immunising constituent of Haffkine's prophylactic. The prophylactic fluid was passed through a Pasteur-Chamberland filter.

1. The sediment found on the bougie consisted of bodies of dead plague bacilli. It was proteid in reaction, and protected rabbits from plague in doses of 300 mg.

2. The filtrate, a clear fluid, also gave proteid reactions, and possessed well-marked protective power even in small doses. It is possible to isolate, after Brieger's method of preparing tetanus toxin, the immunising substance from Haffkine's fluid in a more or less pure condition, and this immunising substance does not respond to any of the known proteid reactions, and possesses well-marked protective power in rabbits against plague in doses of 100 mg. This immunising substance is grey and amorphous, and is soluble in water.

It might be termed extracellular, as it is found dissolved in the surrounding media. The immunising substance present in the sediment might be called intracellular, as it is found in the bodies of dead bacilli.

The extracellular immunising substance is formed in the substance of the cells (plague bacilli), and is then thrown into the surrounding media, and this substance is formed most probably when plague bacilli disintegrate.

Plague bacilli produce besides the immunising substance a proteid which produces pus, and this proteid body is found in the bodies of the dead bacilli. I have found that induration at the seat of inoculation occurs whenever Haffkine's fluid is used, especially when it is used subcutaneously, or whenever the sediment on the bougie is used; whereas these indurations never occur when the filtrate of Haffkine's fluid or extracellular immunising substance is used.

I have found also that these indurations generally contain sterile pus. From this it is clear that the pus-producing substance is in the bodies of dead bacilli. This fact also coincides with the clinical facts observed by many.

1. Suppuration never occurs in severe cases of plague, but only in mild cases or in those cases in which the duration of the disease is long—that is, when plague bacilli are dying or disintegrating. Of course it must not be forgotten that suppuration occurs in cases of mixed infection in which plague germs enter the system with pyogenic cocci.

2. It is known that it is impossible to get a culture of plague bacilli, or even to find plague bacilli in buboes when suppuration has advanced. This, however, can be explained in two ways:

(a) The plague bacilli are dying or disintegrating.

(b) In cases of mixed infection pyogenic cocci might overgrow or outlive plague bacilli; but I have found from my experiments that pyogenic cocci have no direct deleterious influence on plague bacilli, and also they have no prophylactic power whatever against plague. As rabbits previously inoculated with pyogenic cocci invariably die of plague when subsequently inoculated with plague.

Medical School, Hyderabad.

S. MALLANNAH, M.B.

REPORTS ON MEDICAL AND SURGICAL PRACTICE IN THE HOSPITALS AND ASYLUMS OF THE BRITISH EMPIRE.

REGIMENTAL HOSPITAL, EIGHTH BOMBAY INFANTRY, AHMEDNAGAR.

POISONING BY CASTOR-OIL SEEDS.

(By Lieutenant L. T. R. HUTCHINSON, M.A., M.B., B.C. Camb.,
Indian Medical Service.)

IN THE BRITISH MEDICAL JOURNAL for February 10th, 1800, a fatal case of poisoning after eating a couple of castor-oil seeds is recorded. The two following cases may therefore prove of interest:

A havildar, aged 29, and a private, aged 22, of the 8th Bombay Infantry, were both admitted to the regimental hospital, Ahmednagar, on February 10th, 1900, for poisoning by eating castor-oil seeds.

The regiment had been out for a field day since early morning on February 9th, and, after bivouacking out for the night, were returning to their lines on the morning of the 10th. As they passed through some irrigated fields where the castor-oil shrubs were growing, these two men picked the seeds, and, finding them sweet, ate several. This occurred at 8 A.M. The havildar stated that he had eaten a handful, probably twenty-five to thirty seeds or more; the private that he had eaten eight or ten seeds only. About three-quarters of an hour later they noticed a bitter taste, and began to feel giddy. Shortly afterwards violent vomiting and purging commenced in each case, and they were placed on stretchers and brought to hospital with all possible speed.

They were first seen by me at 1.15 P.M. The havildar was then comatose, and almost pulseless; his skin was moist, his extremities cold, and his pupils dilated. After admission no further vomiting occurred, but he passed two stools which contained almost pure mucus and blood. The condition of