relief. — Mr. BELL said it was his practice to puncture in both, but that relief was better marked in cases of orchitis.

Dr. MILNER FOTHERGILL read a paper on the "Prospects of cases of Valvular Disease of the Heart." He pointed out that the well-marked cases, as described in text-books, were generally recognised and appraised correctly. But there were other cases marked by the presence of a murmur when the prospect was very much better. Much misery to patients, and considerable discredit to the profession, were the result of transferring to such cases the prognosis of the most serious forms. Practitioners scarcely ever erred about the grave cases, but they were not always so accurate about the less serious cases. Stokes long ago pointed out that there were cases where the mischief did not progress, or imperceptibly; and Latham differentiated three forms of mitral mischief after endocarditis of varying severity. It was important to recognise the fact that in many cases the injury done is quiescent, like a scar, and the patient is only conscious of something wrong on effort. All light work was not incompatible with length of days. He then related the history of four cases, all well known to him, of mitral disease. 1. A girl, who had a loud mitral murmur at the age of eight. She is now twenty-four, in fair health, as a village schoolmistress. 2. A man whose murmur was first heard in 1867, sometimes ailing, but at work as a blacksmith, only troubled when shoeing very heavy horses. 3. His sister, with disease of two years' standing, last year flooded in labour. She became anæmic, and then some dropsy followed, which quite disappeared under treatment. 4. A man with some mitral disease of twenty-seven years' standing, who had dropsy for the first time last autumn after a short illness. He is now free from any dropsy and feeling well again. Another case of severe mitral disease, lasting thirty-eight years, was cut short by bronchitis last spring. Even aortic regurgitation does not always progress rapidly, and he knew of a number of cases where no perceptible progress had been made in four or five years. Consequently we are not to take a hopeless view of all cases of valvular disease of the heart.—Dr. DE HAVILLAND HALL spoke of the life assurance aspect of heart disease, and gave instances in point. Death from bronchitis in valvular disease was directly due to the heart affection, and he agreed with Dr. Fothergill as to the advisability of great caution in prognosis.—Dr. GILBART SMITH said the difficulty of arriving at an accurate prognosis was in many cases very great. In one case of præcordial pain a soft bruit came out on exertion and the apex was displaced outwards. In such a case a reservation of opinion for further observation would be construed by the patient as a verdict of "heart disease." In another case, one of aortic regurgitation, apparently not advanced, sudden death occurred from the patient taking a journey contrary to advice. Such cases caused considerable hesitation in forming a prognosis. In life assurance, mitral murmurs, when unaccompanied by dilatation, accentuated pulmonary second sound, or venous engorgement, might readily form a good investment, for the slight risk would be more than compensated by the increased premium.—Dr. ALTHAUS thought the views in the paper confirmed by the fact that most of the deaths from heart disease in the Registrar-General's return were set down from sixty to seventy years.—Dr. BROADBENT agreed with the views of the author, and considered that heart disease was not necessarily fatal. It was his practice in such cases as those referred to to give a straightforward opinion as to the existence of heart disease, explaining at the same time that sudden death was not to be feared, and that with care a long life might be looked forward to.—Dr. FOTHERGILL, in reply, said many of these cases were fit cases for life assurance. He had been in the habit of testing the effect of exertion in evolving murmurs by getting the patient to lift a heavy weight.

EPIDEMIOLOGICAL SOCIETY.

AT the meeting of this Society, held at University College on Feb. 2nd, the President, Sir Joseph Fayrer, in the chair, Surgeon-General C. A. GORDON read a paper "On Certain Considerations regarding Cholera and Fever, more especially with reference to India and China." He took as his text weekly reports of health conditions in India, as published in the papers of the day, and from those reports argued that the phenomena of fever, cholera, and small-pox thus presented could not be explained either by the theory of specific poison or by that of contagion. He supported this view by numerous statements, some of which were the result of personal observation, but the greater number were expressions by writers and authorities to whom he referred. In summarising the results at which he thus arrived the following appeared to him the most important—namely, there is reason to believe that although no actual relationship exists between cholera and small-pox, yet the same, or a similar, law applies to the manner of their prevalence as epidemics. Between cholera and malarial fevers a relationship is indicated as existing, alike in respect to causation and phenomena. Both these diseases in India present a general relation to season. Both in India and in England a relationship appears to exist between the type of prevailing fever and locality. Bowel affections generally, in India and elsewhere, present a relation to season and locality, this relation being as defined as that of vegetation to the same circumstances. In endemic fevers intestinal lesions, and those of abdominal glandular tissues, occur as complications in particular cases. Cholera in India occurs under a variety of circumstances, and in different manners, but neither furnish a specific name applied to particular cases as indicating one particular manner of causation. Fevers vary in phenomena and intensity, according to conditions, geographical, climatic, and individual. Among our soldiers in India fevers in the mass and in individual cases are less sthenic in type than they formerly were. Reasons for this circumstance are assigned. Specific poisons are of two categories, as described. Innocuous liquids can, by artificial means, become converted into poisons; hence analogy indicates the likelihood of corresponding changes being effected by means of vital action. But diseased liquids do not necessarily communicate their particular disease when inoculated or swallowed. Certain diseases named by him, which are propagated by infection or contagion, are induced by particular combinations of conditions. Fever in some of its types and forms is of this class; so is cholera. Between these diseases themselves, and with regard to several others occurring epidemically, an affinity is indicated. The expression *malaria* is but another term for climatic influences. The existence of organisms peculiar to malaria has yet to be confirmed. No specific poison is necessary to account for the phenomena of Indian fevers. The theory of specific poison does not explain those in "typhoid" or "enteric" fever. It is opposed as insufficient by a number of authorities. General insanitary conditions predispose to disease; but do they to any one specific disease more than another? It is not established that such is the case. Polluted air induces certain diseases, but not necessarily a specific form of fever. In China, air intensely polluted is compatible with health and activity. The theory of local pollution, if sufficient to account for the occurrence of some epidemics, is altogether insufficient in respect to others. Although contaminated water may induce cholera, the occurrence of that disease is not always thus accounted for. Water induces cholera after that disease has become epidemic; not before. The relation of water supply to cholera in India is not established by recent observations. Very striking instances are adduced where the use of extremely foul water has not resulted in fever, enteric or other. While instances are recorded, particularly in places where cholera is endemic or epidemic, of cases of that disease following the use of contaminated milk; instances are related of infants suckled by women ill of cholera, yet the former not becoming affected by it. Cases said to have been of typhoid fever, due to the use of diseased meat, were subsequently stated to have been cases of trichinosis and sausage poisoning. Although specific typhoid, like several other forms of fever, may be conveyed by milk, this circumstance by itself fails to explain the diffusion of that disease as an epidemic. As a rule, cholera has no relation to age. In India and the tropics generally, the young and the lately arrived suffer more from endemic diseases than the older in age and residence. Infant mortality of Europeans in India is excessive. Individual conditions and habits predispose to particular forms of disease and complications. Climate influences the geographical distribution of disease, and determines its type and incidence. Although cholera has undoubtedly been propagated by means of masses of persons and individuals in numerous instances, there are others equally numerous in which it has not been so. Fevers of particular kinds in India may be propagated by means of infection; but, of the cases

investigated by Dr. Gordon, not one was thus accounted for.

In the discussion which followed, Dr. Murray, Dr. Norman Chevers, Dr. Russell, Dr. Thorne, Dr. Thin, and Dr. Collie took part.

HARVEIAN SOCIETY OF LONDON.

A Case of Temporary Hemiplegia after Localised Convulsion.—On Types of Imbecility.

A MEETING of this Society was held on Thursday, February 5th, Henry Power, Esq., F.R.C.S., President, in the chair.

Dr. HUGHLINGS JACKSON read a paper on a case of "Temporary Left Hemiplegia after an Epileptiform Seizure, beginning in the Left Foot," from which the following is an extract:—"I will put the events of the case briefly in stages—1. A man, thirty-five years of age, came into my drawing-room at ten o'clock January 16th, 1881, apparently quite well: he felt well. He had walked from near Temple-bar to my house. 2. In about two minutes he had an epileptiform seizure, the spasm beginning in his left foot; in less than a quarter of an hour the fit was over; there was no defect of consciousness in the seizure. 3. Directly after the fit the left leg was so much paralysed that he could not stand; later on (but after a slighter fit almost limited to that limb), his left arm was weak. I did not test the arm carefully until after the second attack, as I had not finished examining his leg. At any rate, by the time of ending of the second fit (about 10.30) there was hemiplegia, the epileptic hemiplegia of Todd, or, as I think it is better to say, post-epileptiform hemiplegia; the face and tongue were not affected; there was no loss of sensation. 4. He left my house at twelve; by his own account his leg was all right again at ten minutes to two o'clock; possibly it had recovered before; but then only did he try to walk, and was astonished to find that he could. It had recovered to my observation when I visited him at a quarter past four. His arm had, then, also recovered its full movements; he could use it to button, &c.; the exact time of recovery of the arm I did not ascertain. At luncheon (two o'clock) his fork dropped out of his left hand; once he put his left hand on his chop instead of on his fork. Let us say he recovered in a few hours; not wishing to be indefinite, but wishing the reader to take the time as he likes, either from the patient's statement that he recovered in about four hours, or from my observation, that there was recovery in about six: on either account the recovery was prompt. The fact of quick recovery from so much paralysis is very striking, but the case is clinically pointless without noting that the paralysis recovered from was after a convulsion, and there is little point then, unless the particular sort of convulsion be noted. It was an epileptiform convulsion, the spasm beginning locally and unilaterally, and spreading comparatively deliberately; it was a seizure of a class described by Bravais in 1824. It is one of the class depending on disease of the mid-region of the brain. It is one of the seizures whose anatomy and physiology are so much elucidated by the researches of Hitzig and Ferrier into the so-called motor region. The fit my patient had was one of the rarest; it began in the foot. I said that the patient came into my room apparently well. He was not well. He had had three such seizures before the one I witnessed—proof-positive of persisting local disease. In the third one, as in the one I witnessed, there was no loss of consciousness. I got no account of any decided paralysis after the first three seizures; but then he said he was weak, and if this weakness were local he was paralysed. Five minutes after the last of the three he could walk. He had not bitten his tongue in any one of them. The mode of onset is a matter of extreme importance, and, therefore, it may be noted that he referred the sensation at the onset of his seizure, to the outer side of the ball of the big toe. During the paralysis the knee phenomenon on the paralysed side was in great excess, and clonus was easily obtained on that side. These abnormal conditions were not found on the patient's recovery." Dr. Hughlings Jackson then stated the several hypotheses as to the process by which post-epileptiform paralysis results. He adopts the hypothesis of Todd and Alexander Robertson, that there is temporary exhaustion of nerve fibres consequent on the excessive discharge in the paroxysm.—Dr. FERRIER and Dr. BUZZARD spoke.—Dr. HUGHLINGS JACKSON, in reply, said that he attributed the paralysis but little to "running down" of tension of the cells which discharged, but rather to exhaustion of fibres affected by the discharge. There was no evidence of exhaustion, in the sense of general bodily exhaustion; before the fit the man walked well. Dr. Jackson admitted that in cases of persistent cerebral lesions there might occur occasional symptoms from mere general bodily exhaustion, and in illustration mentioned a case of organic brain disease where the patient always spoke badly when he had gone a long time without food. He alluded also to the well known case of Sir Henry Holland, who, when fatigued, lost his German but not his English, regaining his German after rest, food, and wine. But in the case related there was no general exhaustion, and there was a convulsion. The case after the fit differed from a case of ordinary hemiplegia in that, during the prior paroxysm, effects had been produced from the cortex to the muscles convulsed. Dr. Hughlings Jackson believed that some fibres in the lateral column were exhausted, and he believed that the exaggerated knee-jerk and the foot clonus were owing to loss of cerebral influence—to "loss of control."

Dr. FLETCHER BEACH then read a paper "On Types of Imbecility." The author commenced by referring to the different systems of classification of cases of imbecility which have been adopted and brought forward, one of which by experience he had found useful in describing the disease. Cases under this system are classified under the headings of congenital and acquired imbecility, including under the former those occurring at the time, under the latter those supervening after birth. The following classification was the one suggested by the author:—*Congenital Imbecility.*—1, Simple congenital; 2, Microcephalic; 3, Hydrocephalic; 4, Scalphocephalic; 5, Pearly tic; 6, Cretinism (sporadic, endemic). *Acquired Imbecility.*—1, Eclampsic; 2, Epileptic; 3, Hydrocephalic; 4, Paralytic; 5, Inflammatory (*a*. Hypertrophic); 6, Traumatic; 7, Cretinism (endemic). It was shown that under the heading "Simple Congenital Imbecility" are included cases of a very low, and others of a fairly high, type. The cause of microcephalic imbecility was discussed, and the difference between the hydrocephalic and rickety head was pointed out. The cause of scaphocephalic imbecility was said to be obscure. Pearly tic imbeciles were shown to make good progress mentally, but little physically. The characteristics of sporadic and endemic cretinism were usually the absence of a thyroid gland, and generally the presence of fatty tumours in the posterior triangles of the neck. More improvement was stated to take place in cases of acquired than in congenital imbecility, and the reason for this was explained. Eclampsic imbecility was shown to be due to convulsions coming on soon after birth, continuing some years and then ceasing, but so injuring the structure of the brain during their continuance that the patient becomes imbecile. Under the heading "Epileptic Imbecility" are included some cases where with cessation of fits the greatest improvement takes place, and others who go on from bad to worse and finally end in utter dementia. Inflammatory imbecility was said to be due to measles, typhoid fever, and other acute diseases, as a result or complication of which inflammation of the brain and membranes was produced not sufficiently grave to be fatal, but serious enough to cause mental impairment. The reason for placing hypertrophic imbecility in this class was explained, and the difference between hypertrophy of the brain and chronic hydrocephalus was pointed out. Traumatic imbecility was stated to be due to some injury of the brain, hereditary predisposition acting as a predisposing cause in these, as in other, cases where a slight exciting cause seemed scarcely sufficient to produce so grave a result. Cases illustrating the different classes were related, and the paper was accompanied by photographs of patients under the author's care.

Dr. Hughlings Jackson, Dr. Buzzard, Dr. Ferrier, Dr. Mahomed, and the President thanked Dr. Fletcher Beach for his able and interesting paper, and briefly criticised various portions of it. Dr. Beach replied.