to their natural strength, when the joint is again fully used in the ordinary way there will be no tendency to a recurrence of the accident. Such a line of treatment is that which has been followed for years with the greatest success by Dr. Wharton P. Hood. In a certain percentage of cases it will fail and the trouble will recur. These must be operated upon. Similarly chronic cases that have already had numerous attacks when first seen require operation. Those cases, also, in which a quite loose body can be felt to move to widely different positions in the joint and those in which a loosened cartilage protrudes from the joint will not improve without operation. In all other cases such a course of treatment as has been sketched should be thoroughly tried before the question of operation is considered.

The operation.—A few particulars concerning the operation itself may be mentioned. A vertical incision of about three inches, one inch to the inner side of the patella and going down to an inch below the head of the tibia is the incision which I choose for opening the joint, unless, of course, there is special indication, as the presence of a foreign body, for incising at some other situation. An advantage over the transverse incision is that by the vertical cut, the extension of the vasti on to the tibia, an important supporting structure is not divided; moreover, the vertical incision is easily prolonged upwards when this is necessary for examination of the large synovial pouch about the patella. Care must be taken to clip and to tie every bleeding point, and the joint is to be literally squeezed dry before it is closed. The joint should not be washed out with antiseptics and no drainage should be employed. One exception to the rule of not washing out is presented by such cases as Case 35. Here a loose body, the presence of which had been determined with certainty before the joint was opened, could not afterwards be found in spite of the freest digital searching. On flushing the joint with boiled water the body literally floated out. Such a manoeuvre may occasionally be necessary and has led to no harm. In another case (Case 45) even this proceeding failed to discover a loose body about the presence of which there was absolutely no doubt. It became fixed presumably in the posterior part of the joint behind the crucial ligaments and gave no further trouble. The course of this case to a perfect cure seems to prove conclusively that rest, exercise, and massage may lead to the remedying of a condition within the knee-joint which might well have seemed incurable except by operation. It is, however, possible that the body became fixed owing to influences which would not have come into play had the joint not been opened.

It is hardly necessary to insist upon the strictest asepsis, which must, of course, be observed, but I cannot agree with Mr A. E. Barker that the finger should not be used within the joint. Without such use I cannot believe it possible in some cases properly to estimate or to remedy the fault that may be present. It is necessary in some cases to examine with the finger every part of the joint that can be reached. Even then a small loose body may escape detection as in Case 45.

My experience favours removing loose semilunar cartilages rather than fixing them, and the same applies to torn or hypertrophied alar ligaments or local hypertrophy of synovial membrane.

In closing the wound the sutures are to be passed so as to include the cut synovial membrane.

After-treatment.—The splints are to be taken off within a week. Passive movements are begun as soon as the skin wound is healed, and these are combined with massage. If in spite of exercises and massage there appears some stiffness of the joint it must be freely moved under an anæsthetic and then daily massaged and freely moved to prevent stiffness from reappearing.

Points concerning diagnosis.—The 59 cases here tabulated, all of which presented either the characteristic symptoms with locking of the joint, or else weakness with recurrence of pain and swelling, which is often the only evidence of damaged semilunars, illustrate the following conditions: (1) semilunar cartilage, internal or external, split, loosened or displaced, torn from either or both extremities or from coronary ligaments; (2) loose bodies—the body may be quite free or may be attached by a pedicle; (3) a torn or hypertrophied alar ligament or hypertrophied fringe of synovial membrane; (4) rheumatoid arthritis—roughened cartilage, thickened synovial membrane, and osteophytic outgrowth; and (5) no obvious derangement. There are some

points which help us in differentiating between these conditions, although it will happen not seldom that symptoms most strongly suggestive of one lesion will prove to be due to another. Thus Case 59 was apparently a typical example of semilunar damage, yet the lesion proved to be a perfectly loose body and the semilunars were natural.

In the cases of damage to a semilunar cartilage there are generally these features: (1) distinct history of injury originating the trouble; (2) a well-defined site of pain on the inner or outer side of the knee according to the cartilage damaged; (3) no foreign body palpable; and (4) no creaking in the joint. These same features also characterise generally cases in which the lesion affects an alar ligament or consists in localised hypertrophy of synovial membrane.

In the cases where a loose body is present the symptoms, again, may be exactly similar to those caused by semilunar damage (Case 59). Sometimes, however, the body will be able to be moved into widely separated portions of the joint (Case 45) and felt in its different situations. Similarly a very loose semilunar cartilage may sometimes be diagnosed with certainty through its being protruded at the side of the joint. Such a cartilage, however, may be most confusingly imitated by an excrescence due to rheumatoid arthritis.

Results.—Viewed generally these cases show that operation for internal derangement of the knee-joint is in most cases highly satisfactory. There are generally quick recovery and no recurrence of the trouble. In one case after several years of freedom another operation was required and a large number of loose bodies were removed from the joint. I am, however, very anxious not to minimise the gravity of the operation, which I hold to be considerable, and would lay stress upon Cases 22 and 51, which illustrate failure such as is probably inseparable from a certain proportion of all serious operations. Even short of the unhappy event of permanent stiffness, considerable trouble may be caused in convalescence by profuse secretion of synovia (Case 37) and by a tendency to stiffness of the joint (Case 23). Such cases cause great anxiety and require the most early careful and persistent treatment if a perfect joint is to be finally secured.

Grosvenor-street, W.

A CASE OF ACUTE MYASTHENIA GRAVIS.

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WE have thought it desirable to place the following case on record, partly on account of the rarity of the condition and partly on account of the fact that our case presented some unusual features.

The patient, aged 71 years, was a contractor of Plymouth. He had lived a life of considerable mental and physical activity for about 30 years and during this time his health was above the average. At the age of 60 years he relinquished the greater part of his work; he became easily excited and fatigued and his business capacity obviously lessened. At the age of 63 years he began to suffer severe neuralgic pains in various parts of his body and limbs, for which there was no apparent cause. Finally, however, these pains became limited to the left leg and were sufficiently severe to confine him to bed for many weeks. At this time the urine was found to be of high specific gravity and to contain a considerable quantity of sugar. The glycosuria never quite disappeared, but it became so inconsiderable as to be scarcely appreciable by the ordinary tests.

About 12 months before his death albuminuria set in and continued to the end. During the 12 months prior to his death he steadily lost energy and strength; but no local symptoms presented themselves till seven weeks before his death. He then relaxed his diabetic diet and his final illness appeared to date from this time. He developed a gradually increasing general weakness without malaise. He became exhausted during the process of dressing, the distance which he was able to walk grew daily shorter, and slight bilateral ptosis developed so that he was unable to read for any length of

time. As he did not improve, for the sake of a holiday, he paid a visit to his daughter in Essex. Shortly after his arrival he developed difficulty in swallowing and soon had to limit his food to semi-solids and finally liquids. The latter, however, frequently regurgitated through the nose.

After he had been in Essex about three days he was seen by one of us and again carefully examined. He was then well nourished and when sitting down looked in good health except for his ptosis. He complained of weakness; but on examination it was found that he could fairly well perform any movement of the limbs or trunk. Any such movement, however, was almost immediately exhausted; for instance, he could rise from his chair fairly briskly for a man of his age; a second attempt to do this involved great effort and a third attempt was quite unsuccessful. There was no muscular wasting and the electrical irritability of the muscles was not examined. There was no loss of sensation in any part of the body, nor was there any incoördination, tremor, or involuntary movement of any kind. The abdominal and plantar reflexes were normal. The knee-jerk was at first somewhat exaggerated but became partially exhausted on repeated stimulation. As far as could be determined, however, the knee-jerk could not be absolutely abolished. There was no clonus. There was no ocular palsy, but some nystagmoid jerking occurred on lateral deviation of the eyes, which jerking increased if the lateral deviation was prolonged. There was no history of diplopia. Bilateral ptosis was present to a degree sufficient about half to cover the pupils, but the ptosis increased if the patient attempted to read. The pupils were equal and the pupillary and conjunctival reflexes were normal. There appeared to be no weakness of the muscles of mastication or of the muscles of the face. The patient's voice was fairly strong after a rest of half an hour or so, but aphonia developed after a few minutes' conversation. He was unable to swallow solid food. The pharyngeal reflex could not be obtained. Liquids regurgitated through the nose towards the end of a meal. The heart and lungs were normal except that the aortic second sound was louder than natural. His arteries were normal for his age. The patient had full control of his rectum and bladder. At this time the urine contained both sugar and albumin in moderate amounts. The patient rapidly grew worse and returned to Plymouth on June 27th. On the 29th difficulty of swallowing became more pronounced and it increased daily until his death on July 13th.

No necropsy was obtained in this case, but there appears to be little doubt that our patient suffered from myasthenia gravis or asthenic bulbar paralysis. The unusual features to which we would draw attention are the patient's age, the exceptionally acute course of the disease, and the association with diabetes. From previously reported cases (about 70 in number) it would appear that the disease belongs to the third decade of life, and so far as we have seen in the literature no case is on record of such a patient who has reached the seventh decade. Erb's first case—the oldest at present on record—was 55 years of age.

The present case ran an exceptionally acute course. The only undoubted case that has run a more acute course is that recorded by Vidal and Marinesco; their patient lived only 14 days after the onset. The association with diabetes in our case appears to have been somewhat intimate since the myasthenia set in almost as soon as the patient relaxed his diabetic diet. So far as this observation goes it is in favour of the prevalent view that myasthenia gravis is due to the effect of some toxic agent upon the peripheral neurons.

MILK OR WHEY IN ENTERIC FEVER?

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IN THE LANCET of Nov. 2nd, 1901, p. 1182, Mr. Prideaux G. Selby of Teynham, Kent, published a paper concerning the treatment of enteric fever by whey instead of milk as a staple food, his death-rate for 73 cases being 2.7 per cent. Since that time Dr. S. J. Gee of St. Bartholomew's Hospital has treated four cases entirely, and one case partially, by this method. All did well, but one case perhaps deserves notice on account of its severity and the favourable issue under what is more or less a new departure in treatment, though I believe that Dr. Norman Moore has for some

time past treated his cases of enteric fever chiefly on whey. The patient in question was a man, aged 23 years, a fish-porter, and previously to this illness he had been in good health. On Nov. 3rd, 1901, he caught cold and suffered from headache, and on the 10th he took to his bed. On the 16th he was admitted into St. Bartholomew's Hospital, with the history that for the previous week he had been feverish and his bowels constipated. On admission his temperature was 100.5° F.; his pulse was 84 per minute, soft and regular; his radial artery was thickened; his urine was of specific gravity 1025, had an acid reaction, and presented no abnormality; and his tongue was dry and brown. Examination of his heart and lungs showed nothing abnormal. His abdomen was not distended or tender; his spleen could just be felt. On the 17th his blood gave a well-marked Vidal's reaction. On Dec. 2nd there were many typical enteric fever spots on his abdomen and chest. His evening temperature was usually from 103° to 104°, and his pulse was from 110 to 120 per minute and very soft; he was sponged night and morning. He was taking four ounces of brandy and a small quantity (about half a pint) of beef essence; otherwise he had nothing but whey, of which he consumed from three to four pints in the 24 hours. On Dec. 4th there were some indications of heart failure and the first sound became faint. The patient therefore began to take four-minim doses of liquor strychniæ, given at first three times a day and afterwards every six hours. On the 6th his pulse was very soft and dicrotic and he had occasional shivering fits. His bowels were open about three times daily; on the 6th the motions were stained with blood, and he passed one very large black slough measuring one inch by half an inch, together with several small sloughs; they were of considerable thickness, showing the ulceration to be very deep. His abdomen presented no distension and was natural in appearance. On the 9th several large blood-stained sloughs were passed. On the 10th the man was very ill and the brandy was gradually increased till he now took eight ounces. About 3 A.M. on the 12th, which was approximately the fortieth day of the disease, there was copious hæmorrhage from the bowel amounting to at least one and a half pints; the man was very ill, but he quieted down with morphia given hypodermically. On the 13th the patient was so collapsed and blanched that Dr. Gee gave an unfavourable prognosis. There was, however, no delirium or distension of the abdomen. On the 16th the patient was very restless, he passed urine frequently, and was with great difficulty made to sleep by hypodermic injections of morphia, hot sponging, and paraldehyde. On the 17th his urine was alkaline and contained much pus and the bacillus typhosus; he was given seven grains of urotropine three times a day. On the 18th he was better and the urine was clearing. On the 19th there was an obvious improvement. His bowels were opened twice, the motions containing no blood or sloughs. His tongue was cleaner and for the first time since the hæmorrhage he had strength to put it out when asked to do so. On the 20th the man was better. His temperature was intermitting well and his urine contained no pus. On the 21st (the forty-ninth day of the disease) his temperature was normal, his subsequent convalescence was uninterrupted, and the brandy was gradually reduced. He continued to take nothing but whey until the 31st, when he began to take milk. He was very emaciated, but not more so than might have been expected from the severity of the disease.

The most noticeable points in the case were the following.

1. Distension of the abdomen was entirely absent. Another patient with enteric fever who was taking half a pint of milk with his whey showed more distension on the third day after admission than this patient did at any time throughout his illness. From the size and thickness of the sloughs I am convinced that had the patient's intestines been distended with gas when he passed these sloughs perforation must have followed.
2. There was an absence of delirium almost throughout the illness, except for a short time on one or two occasions.
3. There was an absence of serious diarrhoea in spite of the severe ulceration; the bowels were loose but never became troublesome. It is probable, as suggested by Mr. Selby, that these three symptoms are often much aggravated by, or due to, toxins formed by decomposing milk curd.
4. With regard to the state of the mouth and tongue it did not correspond to Mr. Selby's experience and differed little from that of an enteric fever patient treated with milk, being dry and cracked and difficult to keep clean.

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