of time. Fortunately it was very promptly controlled by means of the icebag, and care was taken not to allow it to reach anything like such a height again. I have great confidence in the icebag for controlling pyrexia in children, and in this case it acted promptly and never failed in bringing about the desired effect. The intracerebral method of injecting the serum has been used in many cases in adults, on the ground that the remedy is more quickly and more effectually brought into contact with the nervous system. Full accounts of cases are given by Dr. Semple¹ and Dr. Gibb.² The operation could have been easily performed through the open fontanelle of the infant.

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A NOTE ON THE KNEE-JERK IN CHOREA.

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THERE is a peculiar modification of the knee-jerk very common in chorea, which, although it has attracted no attention, has, I think, some value in the diagnosis of slight and doubtful cases. It is not present in every case, it is not constantly present in the same case, but when it is present it is, so far as my experience goes, peculiar to the disease.

With the patient recumbent, if one raises the knee, allowing the heel to rest on the couch, making sure that all the muscles of the limbs are relaxed for the time being, and if one then tests the knee-jerk in the usual way, the foot is found to rise more or less smartly, but, instead of falling back immediately, it remains suspended for a variable time—hung up as it were—and then slowly sinks back to its initial position.

There are also variations. Sometimes the peculiarity amounts merely to a sluggish descent following an ordinary ascent, sometimes an ordinary knee-jerk is obtained, but just as the foot is beginning to fall again, it is caught in mid-air—is, as it were, hung up by after-thought—and held for a time or even raised to a higher level than that reached in the first jerk. Sometimes the knee-jerk passes at once into an active more or less persistent—even apparently voluntary—rigid extension of the limb. And between all these varieties there is every gradation. Mild cases show the effect best, as in them it is not interfered with by frequent and extensive movements. In "hemichorea" we find it only on the choreic side, provided the movements on the other side are practically *nil*.

I have looked for this knee-jerk in many cases of chorea, in many cases of other nervous diseases (especially in children), and in healthy people. In chorea, as I have said, I have found it common. In non-choreic cases I have never once seen it. In chorea, as Professor Osler says:—"The condition of the reflexes has not often been studied," and whilst recent writers have either altogether ignored the subject or devoted themselves to tabulated lists of increased, decreased and absent knee-jerks, Dr. Risien Russell alone makes any reference to the condition I describe. "In some cases," he says, "the resulting contraction of the quadriceps extensor is unduly sustained."² This, however, I venture to think, is not quite the whole case, and the peculiarity is one, it seems to me, which deserves more than a passing notice.

First, in its bearing on our conception of choreic movements generally, some interest may fairly attach to it, seeing that we have still no very clear conception of the evolution of choreic movements. The hanging up of the foot when it has risen in the knee-jerk is not merely due to the substitution of a long contraction of the extensors for the ordinary short one, such as one might be led to attribute to some change in the muscle irritability. The cases in which it is hung up by after-thought, that is, when there is a distinct interval between the summit of the knee-jerk and the subsequent suspension, and still more the cases in which this suspension passes into or is replaced by a definite active extension, show that the knee-jerk itself has evoked an involuntary movement.

I cannot but think that the textbook descriptions of choreic movements lay far too much stress on the interference of

"spontaneous" involuntary movement with attempted voluntary movement, and do not give sufficient prominence to the actual origination of involuntary movement in what seems a sort of overflow of impulses. Consider, for instance, the effect of the attempt at voluntary movement itself. No one can watch attentively for long a case of moderate chorea—that is, a case with well-defined but not quite bewildering movements, without seeing that at least many involuntary movements grow out of voluntary effort, and that obvious voluntary effort may actually call into existence a whole series of disturbing involuntary actions. If we were to assume that the nervous centres in chorea (I purposely use a vague term), are in a roughly similar condition to the cortical areas of an animal when the electrodes have been applied to them for some time—so that stimulation of a given area no longer produces the movement special to that area, but the nervous impulses overflow into neighbouring areas—we might perhaps more easily account for the phenomena of the complaint. That such an overflow can occur in chorea is capable of demonstration, and indeed furnishes us with a very delicate test for the presence of the disease. Let a patient in whom suspected chorea exists raise both arms vertically with the hands open. There may or may not be slight occasional choreic movements of thumb or fingers, or both. Now tell him (still keeping the arms up) to put out the tongue. At once existing movements of thumb and fingers are intensified, or if previously absent are now evoked. Here we have cortical centres closely neighbouring on each other, and voluntary movement initiated in one overflowing into involuntary movement due to impulses communicated to the others. The hung-up knee-jerk seems to point in the direction of overflow of impulse.

Secondly, in its bearing on diagnosis, I venture to think this reflex merits some consideration. A small boy was recently brought to me with the single complaint that he sometimes gave way at the knees without warning, and landed on the floor. I admitted him to hospital, and watched him for some time without arriving at any conclusion. The rest, care, and feeding seemed to prevent his falls. On his return home the symptoms recurred, and on readmission I found a very slight tendency to involuntary movement. His right knee-jerk was hung up in the manner I have described, and on testing him with his hands up and the tongue put out, it became clear that he was suffering from chorea. This diagnosis was borne out by the satisfactory results of treatment adopted in accordance with it.

I do not think that cases so doubtful will commonly occur. Quite the contrary. But when they do it seems to me that the sign may occasionally be of value. My friend, Dr. Peacocke, of Dublin, to whom I mentioned this form of knee-jerk some years ago, has told me he has found its presence a help in diagnosis.

If Osler can sum up, as he does,³ our present knowledge of the nature of chorea in the old words of Bouteille, namely, "Tout est extraordinaire dans cette maladie; son nom est ridicule, ses symptômes singuliers, son caractère équivoque, sa cause inconnue, son traitement problématique," it may not perhaps be thought unnecessary to bring forward even the most trifling contribution to its symptomatology.

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NOTES OF A CASE OF CONGENITAL HYPERTROPHY WITH STENOSIS OF THE PYLORUS.

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W. M., aged 8 weeks, was admitted to the Belgrave Hospital for Children on January 24th for wasting and vomiting. He was put under the care of Dr. Ewart, to whom I am indebted for permission to publish the case.

History.—The parents are healthy. The patient was the first child, and there were no miscarriages. The child was born at full time without instrumental aid, and was "a beautiful baby," weighing 8 lbs. The first symptom which attracted the mother's attention was extreme constipation. Usually there was no movement of the bowels for three days at a

time, and then the stool was scanty and of a light colour. At first the child was fed on the breast, and appeared to put on flesh. Not until it was a fortnight old did it begin to vomit after every feed. Cow's milk and barley water and Nestlé's milk were then tried. Occasionally there was no vomiting for one day or two days; then it vomited a large quantity at once.

State while in Hospital.—The following is a summary of the notes taken during the time the case was under observation in hospital.

The Vomiting and its Relation to the Diet.—On admission (January 24th) the child was put on condensed milk (Milkmaid brand) 3ss, water 3j, and brandy mx, every two hours. After every feed vomiting occurred in small quantity. Hiccough, present on admission, remained troublesome until a few doses of sod. bicarb., gr. j, had been administered.

January 25th.—As vomiting continued, raw meat juice was given, 3j every quarter of an hour, for eight hours. Cocain. hydrochlor. gr. ʒss in 3j water was given every four hours to allay the gastric irritability. Vomiting became less frequent, occurring every three or four hours.

January 26th.—On resuming the condensed milk, vomiting occurred after almost every feed. "Ideal" milk 3ss, water 3j every two hours was then tried. As the "Ideal" milk contains no sugar, a small quantity of sugar was added to most of the feeds. Vomiting at once diminished in frequency, and the hiccough ceased.

On January 29th the condensed milk was resumed. Frequent vomiting at once set in, and a considerable amount of blood-streaked mucus was brought up. On January 30th and 31st, however, vomiting occurred only at long intervals, once or twice during the day, and a very large quantity was brought up at these times.

As vomiting became more frequent again on February 1st, whey 3vj, with brandy mx, was given every hour. This gave no better result, even when bismuth was taken before alternate feeds. Therefore the "Ideal" was ordered, 3ss to water 3vj every hour during the day, and every two hours from 10 P.M. to 8 A.M. This diet appeared to agree better than any other, and it was continued up to the time of death (February 5th). The size of the feeds during the last three days of life was regulated from hour to hour as the occasion required. Vomiting after February 1st occurred only once or twice a day: the vomited matter was large in quantity and contained mucus. No bile was ever observed in the vomit.

Throughout the short time the case was under observation it appeared that foods which caused fermentation, such as sweet condensed milk, excited frequent vomiting; while foods such as "Ideal" milk and raw meat juice were retained in the stomach several hours longer. On the other hand, while whey was being taken there was frequent vomiting.

Condition of the Bowels.—On admission, hyd. c. cret. gr. ss morning and evening was ordered, and on the following day a drachm of castor oil was given and retained. No result followed. On the third day a glycerine enema was administered, with but scanty result. The bowels moved spontaneously only three times during the thirteen days the case was in hospital. Water or glycerine enemas were given on six occasions. The stools were always very small in amount, of dark colour, and sometimes constipated.

General Condition.—On admission there was great emaciation; the fontanelles were depressed, the sutures overlapped. The complexion was of a greyish pallor, the eyes sunken, half-closed, the features pinched. The infant had not, however, the apathetic expression associated usually with severe gastro-enteritis. The tongue was clean. There was constant hiccough. The infant weighed 4 lbs. 8 ozs. For a few days after admission he appeared to improve; the cheeks gained a rosy colour, and in five days he weighed 4 lbs. 11 ozs. On January 29th the child began to lose weight, and on three occasions (January 29th, January 31st, and February 1st) he became collapsed, gasped for breath, and was expected to die every moment. With hot fomentations, brandy, and hypodermic injections of strychnine and ether he rallied, took his feeds well, and cried vigorously at intervals. On one occasion a rectal injection of normal saline solution was administered; of 5 ʒss. given, about 3 ozs. were retained. Death took place unexpectedly on February 5th. During the thirteen days the temperature in the rectum was always below 98°, and only on one day did it rise above 96°.

Local Signs.—The abdomen was never distended; its wall was rigid, and the superficial veins were unduly visible. Nothing was made out by which one could diagnose the existence of a dilated stomach. No tumour was palpable.

Post-mortem Examination.—The body was greatly emaciated. The abdomen was not distended. On cutting through the abdominal wall the stomach bulged out, and was seen to occupy almost the whole of the abdominal cavity. The intestines were crowded into a small space corresponding to the hypogastric and left iliac regions. On raising the stomach a quantity of slimy and curdy fluid flowed from the mouth and nostrils. At the pyloric region there was felt a thickening, sausage-shaped, of cartilaginous consistence, seven-eighths of an inch in length. While the stomach was being removed a considerable amount of the contents escaped into the abdominal cavity from the cardiac orifice, thus rendering it impossible to measure accurately the quantity of food contained in the stomach at the time of death. About 3 ʒss of thick curdled fluid mixed with mucus remained in the stomach. On filling the organ with water, closing the cardiac orifice with the hand, and exerting pressure towards the pyloric region no fluid could be seen to flow through the stenosed orifice. On cutting along the lesser curvature and so exposing the interior of the stomach, the lining membrane was seen to be healthy, but covered with stringy mucus. The intestinal canal contained a small quantity of semisolid faecal matter; the greater part was quite empty.

The stomach was placed in spirit, and after four days, when it had undergone a certain degree of contraction, was again examined. A probe about the size of a thick hairpin could be easily passed through the pyloric orifice.

REMARKS.

Congenital hypertrophic stenosis of the pylorus is regarded as a rare condition, inasmuch as only about 45 cases have been described. Of these 45, nearly half have been recorded during the last three years. It has been suggested, therefore, that many cases of so-called marasmus are in reality due

to pyloric hypertrophy. References to the literature of the subject are given in the papers by Dr. J. Thomson¹ and Dr. Cautley,² and in the more recent papers which have appeared in the *Lancet*, *BRITISH MEDICAL JOURNAL*, and other magazines, by Batten, Nicoll, Still, and others.

Etiology.—In the absence of further knowledge on the etiology of this interesting condition, it would be out of place to discuss here the various theories that have been ably put forward. They may be broadly divided into three: (1) Developmental hypertrophy; (2) muscular spasm of nervous origin, or due to some irritant in the stomach, with subsequent hypertrophy; and (3) congenital narrowing of the pyloric lumen, with resulting compensatory hypertrophy of the stomach. Batten³ published an interesting case in which a child 11 weeks old, presenting symptoms of pyloric stricture, recovered under treatment by nasal feeding. The child died at 11 months, with broncho-pneumonia, and the *post-mortem* examination revealed pyloric hypertrophy with moderate stenosis. The fact that the symptoms disappeared with nasal feeding points to this case as an illustration of Dr. Thomson's theory: "A functional disease of the nerves of the stomach and pylorus, leading to an ill co-ordination and therefore antagonistic action in their muscular arrangement." The theory of muscular spasm may account for the slighter cases, but in the more marked cases it is difficult to believe that over-action of the muscle could in so short a time result in so great a degree of hypertrophy.

Diagnosis.—The diagnosis is simple where a tumour can be felt. Constant vomiting, especially when it assumes the character peculiar to the vomiting of dilated stomach, and when it is associated with obstinate constipation from birth, is very suggestive of the presence of pyloric hypertrophy. Marked constipation alone may be due to fissure of the anus, or to spasm of the sphincter ani;⁴ in such cases, however, there is increasing abdominal distension, and local examination usually settles the diagnosis.

Treatment.—In the slighter cases, where the condition is probably due to spasm, the indication is to avoid exciting any unnecessary peristaltic action. The child should be fed by the nasal tube (Batten), or *per rectum*. Rolleston and Crofton-Atkins recorded a case⁵ which put on weight while being fed by the rectum. In the more marked cases it is difficult to believe that other than operative treatment can be successful. Nicoll⁶ records a case which recovered after Loreta's operation. Gastro-enterostomy and pyloroplasty have been performed in Germany and in America.

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

SEA-SICKNESS.

FROM notes made of several cases while at sea, I have come to the conclusion that the cause of sea-sickness is purely reflex and physiological, acting primarily through the semi-circular canals.

The affection commences in a very large percentage of cases with a feeling of giddiness, much increased if the weather be cold.

As the malady develops, the patient becomes faint, the face assumes a sallow appearance, and there is diminished desire for exertion. In some cases symptoms will remain at this stage until the patient becomes convalescent by becoming used to the motion of the ship; in others it passes on to a stage of secretion of saliva, with contraction of the stomach and actual vomiting.

From my observations, I believe the whole of the symptoms can be traced to irritation of the two terminal branches of the auditory nerve to the cochlea and semi-circular canals. The upper division of the auditory nerve is connected at the base of the internal auditory meatus