abnormal constituents. There was some weakening of left grasp; no sensory disturbance. He complained of severe headache only. Vomited twice on tenth day.

12th day: Had two fits, not observed, during night; also vomited several times. Speech was now markedly impaired, slurring. Whole of left upper limb paralysed. 13th day: Vomited several times during last 24 hours. Headache less severe; weakness of left lower limb. 14th day: Discs examined "? vascular thickening, no limitation of field of vision."

15th day: Paralysis complete both left limbs, no paresis of face or ocular muscles. 18th day: Difficult to rouse; understood speech and could reply correctly. Discs again examined; "? slight burying of vessels left disc." 21st day: Unconscious, no knowledge of passage of fæces and urine; increased vomiting. 23rd day; Fit observed Jacksonian in character and commencing in orbicular muscles right side of face. 24th day: Died suddenly in congestive attack; great cyanosis of face.

Autopsy.—Cerebro-spinal fluid sterile, cytology negative. Blood in lateral sinuses sterile. General venous enlargement of venous sinuses, &c. Abscess right side cerebrum, pointing slightly anterior to right Rolandic area. Abscess was solitary, size of Tangerine orange; filled with greenish pus. It was well walled in with a thick capsule; no cerebral œdema except at extension to cortex; this appeared to have been acute and recent. The petrous temporal bones showed no disease; no area of infection in accessory sinuses. Route of infection was not discovered. Cultures from abscess gave *M. tetragenus*.

TWO CASES SHOWING THE EFFECT OF THE INCIDENCE OF ENGLISH MEASLES UPON PRE-EXISTING GRAVES'S DISEASE.

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THE onset of an acute infection in a patient already suffering from Graves's disease is to be recognised as a very serious state of affairs. The first case described is an example of this combination which ended fatally, and is of interest in that the acute infection, as such, was mild in character. Graves's disease is due to an excessive secretion of the active principle of the thyroid gland, which in itself may be a perversion of the normal secretion.[1] The exact process initiating this hypersecretion is unknown, although some authorities have suggested an auto-intoxication.[2] Granted that the initial process in Graves's disease is an infection, the presence of a super-added infection might so lower the resistance of the patient as to allow the original infection full sway, causing death by acute hyperthyroidism.

Charts of Two Cases of English Measles Complicated by Graves's Disease.

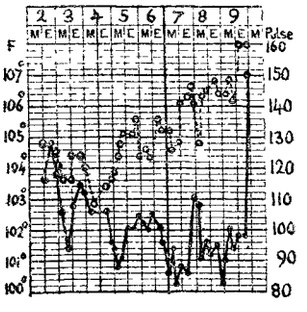

FIG. 1.—Fatal case of measles complicating Graves's disease. Case 1. Showing temperature (continuous line) and pulse-rate (interrupted line). Death occurred on the ninth day.

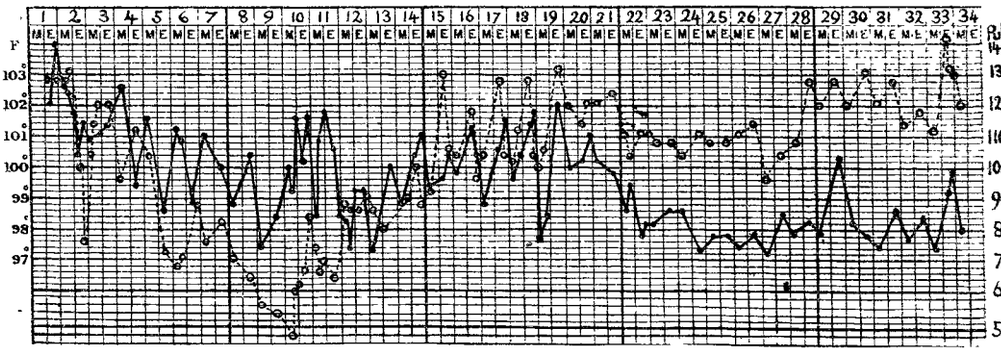

FIG. 2.—Measles aggravating Graves's disease. Case 2. Showing temperature (continuous line) remaining raised for three weeks, and pulse-rate (interrupted line), still 130 per minute, on discharge.

CASE 1.—The patient was a woman aged 31. Her past history was unimportant with the exception of an attack of diphtheria when quite young, certainly prior to the onset of any symptoms of Graves's disease. The latter were said to have begun when the patient was 28 years of age, exophthalmos being the first thing noticed. She manifested the classical signs and symptoms of Graves's disease. The thyroid was not excessively large, but a definite thrill was obtainable over the gland. The onset of the attack of measles was atypical in the absence of catarrhal symptoms, malaise being the only disability of which she complained. On the appearance of the rash, which was remarkable for its pale colour and first suggested rubella, the temperature rose to 104·4° F. The rash had disappeared on the third day, leaving a faint staining and some slight branny desquamation on the forehead and face.

During this time the patient was highly nervous, easily excited, and distressed by profuse sweats following the disappearance of the rash. On the seventh day respiratory embarrassment with basal crepitations, suggesting back pressure in the lungs, appeared synchronously with a small rise of temperature and cardiac dilatation. She then fell into a low muttering delirium, which was broken at intervals by transitory periods of consciousness. From this time until the day of death (9th) the pulse-rate steadily mounted to 160 per minute, it previously never having fallen below 110 per minute. The temperature fluctuated between 100° and 102°, and the respiratory efforts became more rapid and shallow. The temperature rose to 107° just before death, and during the 24 hours preceding death the patient was sweating profusely. The urine contained a small quantity of albumin, but no sugar. Examination of the fundi revealed no abnormality.

The condition of the patient towards the end left no doubt whatsoever that death was due to acute hyperthyroidism.

CASE 2.—This patient was a young woman aged 18. In this instance the thyroid gland was only slightly enlarged, but tremor and tachycardia were present. The attack of measles was mild, although it was three weeks before the temperature reached normal; there was slight tonsillitis, but no respiratory complication to account for this prolonged pyrexia. The tremor increased, and she became very anæmic; slight exophthalmos was present. On discharge her temperature was normal, but her pulse-rate was 130 per minute.

In this instance the attack of measles undoubtedly aggravated the pre-existing thyroid condition. (Four weeks later two-thirds of the thyroid gland were removed under local anæsthesia by Mr. S. H. Rouquette, at St. Thomas's Hospital.) It is interesting to note that a patient suffering from Addison's disease was under treatment for an attack of measles a short time after the preceding patient. The attack of measles ran a normal course, except that profuse staining was present until the twelfth day. On discharge, apparently, the course of the Addison's disease had not been adversely affected.

Dr. T. G. Nicholson recently reported[3] a case of hyperpituitarism, in which an attack of measles produced a fatal result.

I am indebted to Dr. C. R. BOX for permission to publish these notes.

[1] McCarrison: The Thyroid Gland and its Diseases.
[2] H. Mackenzie: Allbutt's System of Medicine, vol. iv., Part I., p. 361.

A CASE OF TRAUMATIC RUPTURE OF THE ILEUM.

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IN THE LANCET of April 6th there is a case of traumatic rupture of the jejunum by Lieutenant J. D. Oliver. The following case presents several features similar to those found in Lieutenant Oliver's case, and, apart from this, traumatic rupture of the intestine is not a very common accident.

Patient, aged 61, admitted to hospital at 8.30 A.M. on March 13th, 1918. At 7 A.M. the same morning, when pushing an iron bar into furnace fire, progress of bar was arrested, and lower part of abdomen struck end of bar. He was immediately seized with severe abdominal pain, and lay on ground until ambulance brought him to hospital. He had not vomited up to admission, and did not do so until after operation.

On admission patient lay on his back with knees drawn up, obviously in great pain. T. 99°, P. 80, of good volume. Abdomen rigid; no distension. Liver dullness not diminished; no dullness in flanks. Tenderness well marked in suprapubic region and epigastrium; more marked in former region. No sign on abdominal wall of bruising or abrasion. Patient passed urine freely; no trace of blood in it. Admitted for observation.

Eight hours after admission pain had increased and spread; still no distension nor loss of liver dullness; T. subnormal, P. 100. I performed laparotomy under spinal anæsthesia. 2 c.cm. of a 5 per cent. solution of stovaine Billon injected; excellent anæsthesia up to about 2 inches above nipple line. Abdomen opened through right rectus. Bowels seen to be in a state of general peritonitis; a little free fluid, not offensive; no gas. About middle of ileum a perforation about size of sixpence on antimesenteric border. Intestine at site of perforation was loosely adherent to anterior abdominal wall about midway between umbilicus and pubes. Gut at site of perforation showed no sign of previous ulceration. Perforation was sutured up in transverse direction. Abdomen was thoroughly swabbed out with cloths wrung out of hot saline and explored; no injury to any other organ. Drainage-tube placed down into pelvis; abdomen closed. During operation patient given pint of saline intravenously; after operation continuous rectal saline. Eserine sulphate, gr. 1/60, six hourly and pituitary extract 1 c.cm. administered. Marked improvement day after operation. Drainage-tube removed on fifth day. There was, at this time, profuse and offensive discharge. General condition improved until ninth day, when discharge became fæcal. He then gradually became weaker and died, 13 days after operation.

I am indebted to Captain John Morley, R.A.M.C., honorary surgeon to this hospital, for kindly giving me permission to publish this case.

[3] THE LANCET, 1917, ii., 87.