midal equilibrium rather than that of a full spasticity. (7) The extensor response comes on *simultaneously* with the incidence of pyramidal disease. The reflex is not abolished by total transverse section of the cord, and appears to be less affected than any other reflex by "nerve shock." (8) The plantar reflex may be absent in health when the foot is cold or damp; it is also frequently absent in hysteria, multiple neuritis, infantile paralysis affecting the leg, and in severe tabes. JELLIFFE.

TRAUMA AND DISEASES OF THE NERVOUS SYSTEM. Judson S. Busy (Brit. Med. Jour., April 30, 1904).

The author takes the position that when patients refer various nervous troubles to a precedent traumatism they are more often justified than the medical profession realizes. In discussing the effects of cerebral concussion, after noticing the more immediate and gross changes that may be produced, such as hemorrhage, inflammatory conditions, or laceration of brain substance, he considers the less conspicuous symptoms and changes which may develop later: the traumatic neuroses, insanity, cerebral tumor, and late apoplexy. Putting aside those cases of traumatic neurosis that are malingering or hysterical, there remains a class of cases which clinically resembles non-traumatic neurasthenia. In later stages these "bruised brain" cases can be diagnosed by their dullness and apathy; mental action instead of being acute and introspective, is carried on with difficulty; and insomnia is less common; in fact, there is often a tendency to drowsiness and stupor. Actual insanity may develop after head injuries, even though there is no hereditary predisposition. Tumors of the brain may sometimes owe their origin to nutritive changes produced by concussion. In some cases, weeks after a blow on the head the patient may have an apoplectic attack, hemorrhage from a ruptured artery, without having previously had any sign of arterial disease, and where syphilis, alcoholism or nephritis could be excluded from the case. Concussion of the spinal cord may cause immediate symptoms, or they may be delayed for several weeks. The more severe traumatism usually produce the more immediate symptoms. A subacute myelitis may not give symptoms until a month after the accident. The author suggests that other spinal cord diseases more or less degenerative in character, such as tabes, multiple sclerosis and chronic myelitis, may follow trauma, though no doubt in most cases the traumatism only co-operates with other causes, especially syphilis. Many cases of progressive muscular atrophy apparently originate from severe strain or other injury to a limb. The muscular atrophy depends on a damaged nutrition of the motor cells, this derangement being the effect of morbid impulses from the irritated nerves of the injured joint. CAMP.

JACKSONIAN EPILEPSY: OPERATION, RELIEF. H. Muir Evans (British Medical Journal, May, 4, 1904).

This case is evidently one of those early cases of epilepsy described by Gowers as originating in the first two years of life, and associated with a cortical lesion. The patient was 17 months old when she had the first convulsion. There was no history of injury or previous illness. The child was well developed and apparently healthy. The first convulsion involved only the left side, lasted six hours, and was followed by a temporary paralysis of the left side for fourteen days. There was a period of quiescence of eight months, then a recurrence of the attacks. The fits at this time were of two kinds, one a sudden dropping forward of the head, and the other a severe convulsive seizure involving the left side of the face and the left arm and leg. Between these attacks the child constantly made rapid sucking movements of the tongue, and there was paresis of the left side of the face, not affecting the eye.

There was no optic neuritis. Medical treatment, sodium bromide gr. XV t.i.d, gave no relief. The skull was trephined so as to expose the ascending frontal convolution at its junction with the third frontal. The exposed surface of the brain looked as if a mucous polyp was flattened on it. This was incised and about three drachms of clear fluid escaped. There were no signs of a blood clot. The wound was closed without drainage. No attacks occurred after the operation, which was a complete success.

CARL D. CAMP (Philadelphia).

GASTRIC DILATATION AND TETANY. John H. Cunningham, Jr. (*Annals of Surgery*, April, 1904).

The author reports a case which is instructive. The individual was a male, unmarried, 28 years of age, a collector by occupation. His previous history and his habits were negative. His father died with heart disease and his mother of cancer. The present attack dated from 1896, when he had attacks of vomiting of two weeks' duration, which occurred without known cause. During the attacks there was constant dull pain over the epigastrium. These attacks occurred to the number of twelve to twenty each year. They resulted in profound emaciation and in preventing him from carrying on his work. Early in 1897 he noticed that his eyes began to fail, and although glasses were prescribed he found that they did not entirely cure the disorder, although they helped it markedly. He very rapidly became dependent upon them, however, finding that unless he wore them in the intervals between his attack he became immediately nauseated. He went the round of a large number of hospitals, having been seen by a number of prominent physicians. In 1902 he began to vomit material which was stringy with blood. An effort was made to pass a stomach tube upon him, but this was abandoned because of its producing contractions of the fingers. The physical examinations were those usually ascribed to chronic dyspeptics. There was a slight trace of albumin in the urine. During examination the patient vomited 32 ounces, which contained the customary evidences of decomposition. Immediately after vomiting he had a typical attack of tetany which was bilateral. The thumbs were drawn in, the wrists flexed, arms flexed at elbows and rotated inward. The flexion of the lower extremity was strong. The lips were fixed and the patient was unable to speak. The patient suffered at the wrists, said he was conscious of what had happened, but was mentally confused. There was free HCl and no lactic acid. Five days after the first attack of tetany there was a second, but a milder attack. Two days later an attempt to pass a stomach tube resulted in tetany. He gradually improved under medical treatment, and refusing operation, left the hospital. Nine months later he was taken to the Emergency Hospital in profound tetanic convulsions, which lasted four days. Again he refused operation, having recovered under medical care. A posterior gastro-jejunostomy was performed at a later date, it having been found that, grossly at any rate, the viscera were in a normal condition save for a thickening at the pylorus. The recovery from the operation was uneventful. Three months after the operation he had gained twenty pounds, and was able to eat everything, including pastry. The symptom known as gastric tetany is to be differentiated from the tetanic spasms in gastro-enteritis, pregnancy, thyroidectomy, the puerperal state and many other conditions, such as epilepsy, hysteria, etc. Kussmaul in 1869 described this form: It is usually associated with benign pyloric stenosis, or it may be the result of pressure from without. The attack which is typically ushered in by a pricking of the hands usually has a direct relation to vomiting. The contractions appear in the hands and are usually characterized by flexions, although the extremities are sometimes extended. After the attack