lute upon the right side, as was the temperature sense of heat and cold. On the side of the hemiplegia there was no particularly marked hyperesthesia, save for pain on movement of the arm and leg, and extreme sensitiveness of the soles of the feet, and no genuine hyperesthetic zone. No anesthesia of left side, except small patches over left shoulder. The right knee-jerk was normal, while the left was much exaggerated and disappeared after twelve hours, and did not return for two weeks. The cremasteric, abdominal and mammillary reflexes were abolished on both sides. Retention of urine and absolute constipation for days occurred, after which involuntary evacuations of urine and stools persisted for eighteen days. There was a rise of temperature during the first week. A year after the injury the hyperesthesia on the left side had disappeared, while the anesthesia of the right side persisted, tactile sensation being normal. The left-sided, deep reflexes were exaggerated and the limbs were more or less spastic, but not atrophied.

The writer draws the following conclusions:

(1) The lack of correspondence between hemispinal section in certain animals and in man is probably due to different anatomico-physiologic conditions.

(2) The Brown-Séquard syndrome undoubtedly exists, although less an entity than a symptom.

(3) Variations from the original type occur from etiological factors and with varying extent of the cut or other lesion.

(4) The secondary temperature in the case reported was probably due to subsequent myelitis.

(5) Few vasomotor symptoms were observed.

(6) The contra-lateral anesthesia was not dissociated, but total, for all varieties of sensation, as in the original description of Brown-Séquard paralysis.

(7) It was a typical case in the bilateral lesion, the crossed and persistent monoplegia, the shallow respiration and the bladder and rectal disturbance.

(8) Spasticity, while often suggesting incomplete lesion and irritation, in that case was persistent, and suggested bilateral lesion.

(9) While a diagnosis of hemato-myelia might have been made, the clinical history, with delirium, pain in the head and neck, with fever, indicated late myelitis.

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CEREBROSPINAL MENINGITIS DUE TO FRIEDLÄNDER'S PNEUMOBACILLUS.—K. Jassinger (*Centralblatt für Bakteriologie*, 1901, July 12).

Up to the present time this bacillus has been found in only a few cases of purulent cerebrospinal meningitis. The author cites a case occurring in a patient, sixteen years of age, who was suddenly taken ill with chills, followed by a permanent rise of temperature to over 101° F. There were present uncontrollable vomiting, intense headache, general muscular pains, profuse perspiration, irregularly contracted pupils and incontinence of urine and feces. Coma supervened and death took place on the seventh day. The clinical diagnosis was cerebrospinal meningitis, and upon autopsy the characteristic pathological lesions were found. A cover-slip preparation of the meningeal pus stained by methylene blue showed both intra- and extracellularly a short, round bacillus, sometimes occurring in pairs. The twenty-four-hour bouillon growth showed a general diffuse clouding with pellicle formation. Agar cultures showed a white surface growth and gelatin plates small, round, opaque colonies. Stab cultures in gelatin after forty-eight hours' growth showed a typical development along the line of inoculation.

No coagulation was produced in milk. Glucose was fermented. Gram's stain was not retained. The organism was pathogenic for mice in twenty-four hours by intraperitoneal inoculation, capsule bacilli being obtained from their heart's blood. He therefore regards the organism found as identical with Friedländer's pneumobacillus.

HIGLEY.

MYASTHENIA GRAVIS. E. Bramwell (Scottish Medical and Surgical Journal, 1901, May).

Bramwell gives the history of the first case of myasthenia gravis reported in Scotland, and 9 such cases have come under his notice; elsewhere 80 or 90 cases have been reported. It is often mistaken for hysteria. Rapid fatigue of the muscles is the prominent symptom. Rapid exhaustion by the faradic current constitutes the myasthenic reaction. The disease may be due to a toxin of endogenous origin, or some congenital defect in construction or mode of functioning of the neuromotor apparatus. In one-quarter of the recorded cases there has been a neuropathic tendency; some have followed acute infective diseases, which probably acted simply as predisposing agents. Prognosis is uncertain; the disease is often fatal, generally from dyspnea. A favorable prognosis should be given the patient, as psychic impressions exert considerable influence, while friends should be warned of the gravity of the case. The affected muscles should be spared and if symptoms are severe, the patient should be kept in bed. The larger portion of the food should be taken early in the day before fatigue occurs, solid food should be minced, faradism avoided though galvanism may be tried, and when dyspnea occurs the tongue should be drawn forward, oxygen saline transfusion and artificial respiration tried.

JELLIFFE.

THE ACTION OF ALCOHOL ON THE NEURONES. J. Arm and Kleefeld (Journ. de physiol. et path. gen., July, 1901.)

Observations along this line are important in that modifications of the central nervous system occasion alterations in respiration, circulation and temperature range. These experimenters trephined the parietal region of a rabbit's skull under antiseptic precautions and without the use of morphine, ether or chloroform. They then closed the wound superficially and after leaving the animal at rest for two or three days injected into the jugular vein 100 c.c. of a seven per cent. aqueous alcoholic solution. After two or three minutes they raised a portion of the cerebral cortex. This was fixed in a mixture of potassium bichromate and osmic acid and submitted to the rapid manipulation of Golgi. In the second series they introduced 100 c.c. of a 15 per cent. alcoholic solution into the stomach of rabbits and made similar examination of the cerebral tissue. Here the pathological changes were increased in amount over those found in the first series. As the result of this work, they conclude that the modifications produced by alcohol are not to be considered as a form of degeneration of nerve-cells, since the alterations appear in a very few minutes after the injection of the alcohol. The moniliform (beaded) state and disappearance of the piriform appendages are seen only in the finest protoplasmic prolongations and far removed from the cell-body. As the dose of alcohol is increased this condition augments and approaches the body of the cell. This appearance is characteristic and not like that produced by chloroform, morphine or chloral hydrate.