attacks one definite spot, which may vary on different occasions, but never during the attack, the point most frequently involved being directly behind the sternum, whereas the globus hystericus is rather movable, often changing its place several times during one attack. Once the globus reaches the throat there is at once a sense of suffocation, which is never present in esophageal spasm, nor is there any vomiting in hysteria. Physiological contractions of the organs in the throat cause usually no sensation, whereas spastic contraction may sometimes be accompanied with pain; but never calls out the sensation of a foreign body. It should also be remembered that globus hystericus may first originate in the lower region of the abdomen, and thence travel upward. It thus becomes clear that we really have to deal, not with a cramp, but with the sensation simulating one. It is evidently the sympathetic nerve which may serve as the organ of irradiation, thus conveying to the patient the sense of pain in a locality different from the one where it really originates. On examining twenty hysterical patients with reference to the point the author obtained the following results: There was in all the cases a marked hyperalgesia of both the lumbar and the cervical sympathetic; this hyperalgesia was associated with real pain in the nerves that radiate from these regions; in almost half of the cases the globus hystericus could be called out by pressure on the lumbar part of the sympathetic. The globus hystericus consists of a sensation that emanates from the terminal filaments of the sympathetic with the included prevertebral branches. It thus happens that the sensation experienced by the hyperalgesic lumbar portion of the sympathetic tends to affect the hyperalgesic ganglia of the terminal nerves in such a way that the successive repetition of the stimuli determines in the ganglia themselves an activity which would otherwise remain latent. ROVINSKY.

EXHAUSTION DISEASES OF THE NERVOUS SYSTEM. L. Edinger (Deutsche med. Wochenschrift, 1904-1905).

Edinger, in an interesting series of articles, discusses once again the theory which he advanced some years ago that there are nervous diseases which develop because, under certain circumstances, the normal demands imposed by the performance of function are not met by a corresponding restoration within the tissues. The characteristic of this condition is a simple atrophy of nerve fiber. All diseases of the nervous system can be divided into focal diseases, toxic affections and exhaustion diseases. In healthy persons exhaustion is characterized anatomically within the cell by a disappearance of the tigroid bodies and perceptible changes in the medullated fibers. If the using up of tissue is too great, or the replacement is insufficient, there is more permanent and complete destruction of the cell and fiber. The glia proliferates to take the place of the degenerated nerve tissue. The process in all these diseases is identical, but the location differs. They are all progressive. These exhaustion diseases may arise on account of abnormal demands on the normal tracts, although there is normal restoration of the tissue. In this way may be explained the various atrophies from disuse and the professional neuritides. In other cases there is not a sufficient restoration to meet the demands of normal functions. These cases are usually due to some poison, such as syphilis or lead. He gives a number of interesting observations of lead paralysis to prove that the paralysis first affects the muscles upon which the greatest demands are made, and shows the excessive use of the extensors of the wrist in the use of the paint brush. He gives many instances also of the effect of over-exertion in one part or another in exciting or aggravating certain special symptoms of tabes. The cause of the exhaustion may differ according to the nature of the poison. Examples of this second class of exhaustion diseases are polyneuritis, tabes, combined systemic diseases and general paralysis. Exhaustion diseases may also develop when different nerve tracts are from the outset not sufficiently developed to be able permanently to perform their functions, but atrophy prematurely—the abiotrophy of Gowers. The hereditary nervous diseases, many of the combined scleroses, amyotrophic diseases of the cord and medulla, primary non-tabetic optic atrophy, and probably progressive nervous deafness, belong to this class. Several nerve tracts may be affected in these conditions, either simultaneously or successively, and various combinations may occur. The types are not always constant; thus, tabes and general paralysis may often co-exist, and tabes and spinal amyotrophy sometimes occur together. Combinations in the third group, however, are not observed, because here some definite tract is congenitally defective while other regions are normal, and thus this tract becomes more easily exhausted; whereas poisons exert an influence upon the entire nervous system, and that part is affected which is most used. The old theory of a selective action of poisons, such as the selective action of lead upon the peripheral motor neurones to the extensors of the wrist, is regarded as no longer tenable; those neurones are affected because they are most exhausted by the demands put upon them. The therapeutic applications of this theory are obvious. With a given predisposition, toxic or hereditary, the possibility of exhaustion must be borne in mind. Thus tabetic patients should walk little, take only such exercise as does not fatigue, urinate every hour, wear dark glasses in bright sunlight, go to bed for a few days at any exacerbation of the disease, and fear any strain. By this means of treatment Edinger states that of late, for example, he has observed no vesical paralysis in tabes. In multiple neuritis absolute rest in bed is requisite; in mononeuritis fixation of the part by splints. By careful application of this theory Edinger believes that we shall obtain greater results both in treatment and prophylaxis. (The theory thus advanced with a wealth of illustration and argument, for which space is lacking here, is interesting and suggestive, but it hardly seems absolute. In point of fact, in tabes, for example, we still know very little as to the determining cause. Syphilis is most probably the chief, perhaps the sole, predisposing cause, but we know nothing as to why one syphilitic becomes tabetic and another remains well nor, even though Edinger finds a history of exhaustion as the exciting cause in some cases of tabes, was there a greater amount of exhaustion than with other syphilitics who did not become tabetic. Or, to take one individual symptom of tabes, optic atrophy, the optic nerve is constantly stimulated during our waking hours more than any other sensory nerve of the body. Why should not optic atrophy be the first and most constant symptom in tabes, instead of being observed in only a small percentage of the cases? Why, furthermore, should exhaustion affect the sensory neurones under the influence of syphilitic toxin, and the motor neurones under the influence of lead? Even on the hypothesis that to the exhaustion is added a congenital defect of certain nerve tracts, sensory or motor, lead tabes and syphilitic amyatrophy ought to be commoner affections. Nevertheless the theory, although not fully adequate, is suggestive, and the therapeutic indications are of much promise. Certain of them, indeed, as rest in bed for tabetics and immobilization by a splint in mononeuritis, have long been recognized as beneficial.) Philip Coombs Knapp, in *Boston Medical and Surgical Journal.*