Studies on a Case of Hypo-Pituitarism. (*Amer. Journ. of Ins., October*, 1920.) *Newcomer, H. S., and Strecker, E. A.*

The patient on admission to hospital was æt. 12 years 2 months; her apparent age was 17 years. Height, 63 in.; weight, 155 lb.; circumference of head, 55 cm. Her skeletal measurements showed generalized overgrowth with a relative increase in length of the long bones. The teeth showed moderate spacing; her hair was low over the forehead and in temporal region; large amount of firm fat more or less evenly distributed, with some excess in axillæ, breasts, over abdomen, and in gluteal region; breasts well developed, but not fully so for normal woman. There was some hair on labia majora, with evidence of precocious sexual development. The urinary output was from 1,000 to 2,500 c.c.; temperature was continuously subnormal. Her metabolic quotient was above the normal. An X-ray examination showed an approximately normal sella. Her mental age was 7·5 years—a retardation of 4·5 years.

Four hundred grammes of sucrose were given without producing glycosuria, and with a 40-mgrm. fall in blood-sugar; similarly, 325 grm. of glucose gave no glycosuria and a fall of 15 mgrm. in blood-sugar.

The authors consider that there was hyperfunction of the anterior lobe, resulting in skeletal overgrowth, and the posterior lobe deficiency completes the syndrome, and accounts for the high sugar tolerance, adiposity, subnormal temperature, somnolence, and dry skin.

The child was treated with pituitary whole-gland extract up to gr. 100 *t.i.d.* without glycosuria following the injection of 200 grm. of glucose. The blood-sugar became more normal and the dose of extract was decreased; several weeks later an essentially normal sugar tolerance was reached, and the authors consider it a fair conclusion to state that the patient has had the two outstanding signs of hypopituitary disease removed, *viz.*, the weight has decreased and the sugar tolerance has become normal. The mental condition has markedly improved.

It is interesting to note that administration of thyroid extract in small doses caused symptoms of thyroidism and the drug had to be discontinued.

L. H. WOOTTON.

Trauma and other Non-Luetic Influences in Paresis. (*Journ. of Nerv. and Ment. Dis., August*, 1920.) *Osnato, N.*

Case-histories are given which tend to show that head injuries (9 cases), emotion (1 case), doubtful traumatic factor plus infection of the bladder and bed-sores (1 case), influenza with pneumonia (1 case), and prolonged etherisation (1 case) may acutely precipitate paresis in an individual already suffering from cerebral or general syphilis, or adversely affect an existing paresis. Tanzi and Lugaro believe that an endogenous or exogenous element intervenes in the cases of cerebral syphilis to produce an alteration in the permeability of the blood-vessels, allowing a continuous passage of spirochætes and their toxins into the ectodermal tissues and thus transforming syphilitics into paretics, and that the difference between the syphilitic cerebral processes and those of paresis are explained by the difference in the site of the infecting organisms. They do not agree with the theory of various French authors

that there is a specific nervous strain of spirochætes for paresis. They feel with Kraepelin that alcohol and other toxic and infectious agents do have a contributing predisposing influence in the production of paresis, and they think it possible that trauma favours a decadence of the nervous system in syphilitics. Orton found two types of lesions in the blood-vessels. One was stationary and the other showed a chronic progressive inflammation. The stationary lesions were those of healed syphilitic endarteritis with thickening of connective tissue between the intact endothelium and the elastica. He believes that these lesions are due to the varying degrees of syphilitic arteritis during the early stages of the infection, and are not characteristic of the paretic process in the vessels. This latter process consists in an active inflammatory change characterised by a lymphocytic and plasma-cell exudate in the adventitia. These processes were found also in many extra-cerebral vessels. Orton considers that these findings are sufficient to prove the hypothesis of the invasion of the brain by way of the peri-arterial lymph-spaces. The writer concludes from the pathological findings noted above and those of Adolph Meyer, Frazier and others, along with his own clinical observations, that any injury to the small blood-vessels which will allow the spirochæte to enter the brain-tissue will immediately favour the institution of the paretic process. This injury may be an actual trauma with hæmorrhage, or it may be an infective or toxic process which injures the intact endothelium and favours the entrance of the spirochæte into the adventitia and the perivascular lymph-spaces and thus into the brain tissue. Trauma of the brain may, by causing vascular injury or brain destruction, be followed later by gliosis and nerve-cell sclerosis, allowing first the spirochætal invasion and later adding to the gliosis and sclerosis of nerve-cells which are also an integral part of the paretic brain pathology. The writer thinks it possible that an excess of the adrenal content of the blood and a hypersecretion of the thyroid gland caused by emotion may produce an increased permeability of the blood-vessels or an actual damage to their structure, and so favour the passage of the spirochæte. Account should also be taken of the changes caused in the nerve-cells by fatigue and emotion (Crile).

C. W. FORSYTH.

Encephalitis Lethargica. (*State Hosp. Quart., November*, 1920.) *Montgomery, W. H., and Waldo, L. T.*

The apparent chronological relationship to influenza led to the conclusion that encephalitis lethargica was probably a sequel to influenza. Wittich and Sennert noted a comatose variety of influenza as early as 1580. In 1712 there appeared in Tübingen the first outbreak of sleeping-sickness definitely associated with influenza historically. Following the epidemic of 1889–90 in Northern Italy a condition called "nona" appeared, accompanied by fever, delirium, and coma. This did not extend to other European countries or America, in contradistinction to the wide-spread distribution of the influenza epidemic it followed. The present epidemic of encephalitis lethargica had its beginning near Vienna in the winter of 1916. Early in 1918 it was recognised in Western Europe, and about a year later had arrived in America. This history shows why encephalitis lethargica was at first