breast-fed premature infants, and in those showing alimentary disturbances, shows an anemia of chlorotic type.

Treatment: For the general and hygienic treatment of primary and secondary anemia, the suggestions made for general measures in the care of rachitis should be followed. The infant must, above all, be given the advantage of a good environment, plenty of fresh air and sunshine.

Iron therapy for the purpose of increasing the iron content of the tissues and the hemoglobin should be started early. Among the iron compounds to be recommended are ferri carbonas saccharatus, ferri et ammonii citras or ferri lactas, in doses of from 0.03 to 0.12 gm. ($\frac{1}{2}$ to 2 grains) from one to three times daily.

Small doses of liquor potassii arsenitis, from 0.03 to 0.06 c.c., may be given one or two times daily for short periods. The infant should be observed carefully for evidence of arsenic intoxication. In the presence of congenital syphilis, mercurial therapy is imperative and may be combined with the arsenic treatment to good advantage.

It is of the greatest importance to bear in mind that, as in the case of rachitis, the treatment for anemia should be started early. It is our custom to begin the prophylactic treatment of both of these conditions in the first weeks of life.

Spasmophilia.—The term spasmophilia diathesis, as applied in the clinical sense, refers to a constitutional anomaly characterized by a general hyperexcitability and irritability of the nervous system. The most frequent active manifestations are general convulsions, laryngospasms, spasmodic apnea and carpopedal spasms. Among the latent manifestations, are Chvostek's facial phenomenon and Trousseau's sign. Among the most constant findings is Erb's sign, the presence of hyperexcitability of the peripheral nerves, as evidenced by increased reaction to the galvanic current.

Spasmophilia appears earlier in premature infants than in full-term infants, and may be manifest as early as the sixth to the tenth week of life. The blood of these infants shows a marked decrease in its calcium content. Kramer, Tisdale and Howland found the calcium of the blood serum, in the presence of active manifestations, in the average of their cases to be 5.6 mg. per hundred cubic centimeters of serum, as compared with from 10 to 11 mg. in normal infants, while the inorganic phosphorus of the serum, in about half their cases, was within normal limits or slightly above normal. This is in marked contrast with cases of rickets uncomplicated by tetany, in which they found a much reduced inorganic phosphate serum content. The significance of this relatively high phosphate content in the presence of decreased calcium, in cases of rickets complicated by tetany, is as yet unexplained. The interest of the orthopedists in tetany depends to a great extent on the secondary complications, such as intracranial and intraspinal hemorrhages following convulsions, and the fractures and deformities secondary to tonic carpopedal spasms. Several cases of fracture due to prolonged and powerful contraction of the extremities have come under my care. Such deformities necessitate prompt general dietetic and medicinal treatment, reduction of the deformities and immobilization.

Scurvy.—All that has been said of the etiologic factors of rickets has an equally important bearing on the development of scurvy. Many cases of latent scurvy

undoubtedly remain unrecognized. The severer types of hemorrhage and cases complicated by pathologic fractures, epiphyseal separation and intracapsular hemorrhage need surgical care as well as dietetic treatment.

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ABSTRACT OF DISCUSSION

DR. W. McKIM MARRIOTT, St. Louis: Dr. Hess has brought out two important points; first, that a premature baby is potentially an orthopedic patient, and secondly, that realization of this fact early enough may prevent a great deal of trouble later on. It is a fact not generally recognized that birth hemorrhage occurs more frequently in premature infants than in those born at term, even though the latter may be delivered by forceps. A certain number of premature infants during the first few months of life slowly develop a condition of opisthotonos. This does not seem to be dependent on birth hemorrhage. It has been our experience that the most satisfactory way to treat these infants is to keep them on the Bradford frame continually until there is no more tendency to opisthotonos. Unless this is done, the deformity may last a long time. Such a large proportion of premature infants develop rickets that it seems safe to consider that they will all develop rickets ultimately, and to treat them accordingly. It is advisable, as a routine, to give every premature infant a small amount of cod liver oil; 5 drops, three times daily, is usually sufficient to prevent rickets in infants of this type. There is no advantage in adding phosphorus to the cod liver oil. A certain number of premature infants who develop rickets may also show evidences of spasmophilia or tetany, as Dr. Hess has mentioned, and this may secondarily lead to cerebral hemorrhage as a result of the convulsions. The specific for tetany is calcium. To control the active manifestations, large doses of calcium chlorid are required: from 10 to 15 grains, four or five times daily.

DR. JULIUS H. HESS, Chicago: After illustrating some of the pathologic conditions encountered in premature infants, I would say that they are in great part worth saving. They are deserving of the full consideration of the diagnostic group of the hospital in which they are born, and not the least important factor in proper orthopedic inspection.

SUBSEQUENT COURSE OF A CASE OF ADDISON'S DISEASE

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Under the heading, "An Autograph History of a Case of Addison's Disease," the late Dr. A. L. Muirhead¹ gave an unusually interesting description of the clinical course of his own illness. In this report we find a professor of pharmacology investigating and recording the subjective and objective results of treatment on himself, and the effects of drugs on the course of Addison's disease, from which he suffered and eventually died. I will record the course of events subsequent to his report, which covered his illness until December, 1920.

CLINICAL COURSE

Dr. Muirhead left the Mayo Clinic, Aug. 11, 1920, and for several months continued to improve. The pigmentation had almost disappeared, and he was able to resume his work as professor of pharmacology in Creighton Medical School. Improvement continued until Jan. 25, 1921, when there developed a gastric upset, loss of appetite, abdominal discomfort and weakness, accredited by Dr. Sachs, his personal

1. Muirhead, A. L.: An Autograph History of a Case of Addison's Disease, J. A. M. A. 76: 652 (March 5) 1921.

physician, to hyperacidity. The attack yielded somewhat to sodium bicarbonate, magnesium oxid and bismuth so that he was again able to return to his work. However, the weakness persisted and he was placed on digitalis. Subsequent to January 25, the salivary secretion was deficient, and he lacked appetite and could not relish food. He continued on suprarenal treatment; epinephrin solution 1:1,000, 3 minims, twice a day hypodermically; rectal injections of $\frac{3}{200}$ grain of the whole gland, and two capsules, 10 grains each, of whole gland, by mouth, at night. In February, the weakness and shortness of breath became more pronounced. He suffered from pain in the left upper portion of the chest which Dr. Sachs called "low pressure angina." The pain was relieved by heat, exertion, food and nitroglycerin.

In March, 1921, Dr. Muirhead returned to the clinic, complaining of extreme weakness which confined him to bed, and of pain in the left shoulder. The pigmentation was marked. He was extremely asthenic, and suffered almost constantly from gastro-intestinal symptoms, especially burning in the stomach. The heart was slightly enlarged and the sounds were of poor quality. An electrocardiogram disclosed a rate of 92, sinus tachycardia, notched QRS complex in all leads, arborization block, and slight right ventricular preponderance. The blood pressure was 86 systolic and 54 diastolic; the blood urea was 56 mg. for each hundred cubic centimeters, and the uric acid 2.8 mg. The hemoglobin was 68 per cent., and the erythrocytes numbered 3,820,000. The patient ate very sparingly, and toward the end of his stay developed a mild acidosis. He left the clinic, March 23, unimproved. The subsequent course of the disease is submitted in Dr. Sachs' report:

"The blood pressure just prior to his death was 84 systolic and 60 diastolic. Dr. Muirhead had a mild acidosis from time to time, for which he was given alkalis. Alkalis during the latter part of his illness distressed him greatly and were discontinued. His gastric acidity changed from a hyperacidity to a hypo-acidity. He progressively became more asthenic until it became difficult for him to swallow. He gradually lapsed into coma, and died, April 26, 1921."

REVIEW OF CASE

Clinically, Dr. Muirhead presented a typical, slowly progressing case of chronic Addison's disease with pigmentation, marked asthenia, intestinal discomfort, and low blood pressure. He gave a questionable history of renal tuberculosis; but tuberculosis was not borne out at operation, a right side nephrectomy being performed in 1918.

Subsequent to the nephrectomy, gout developed; the patient suffered from typical attacks of great severity, involving the metatarsophalangeal joints associated with high level of uric acid in the blood. Both the gout and blood uric acid responded promptly and permanently to a low purin diet. It is of interest to speculate on whether or not the removal of the kidney was a factor in the increase of the blood uric acid and in the development of gout.

The patient's improvement under treatment with epinephrin and the whole suprarenal gland during the first visit to Rochester, and after his return home, was striking objectively as well as subjectively. Rectal injections of epinephrin proved effective for a considerable period, although they were finally discontinued because of tenesmus. Glandular therapy was of unquestionable value in the relief of asthenia and of gastro-intestinal discomfort, and resulted in a temporary clearing up of pigmentation. For a time, at least, it also checked the general course of the disease.

Although the results were only temporary, they were almost miraculous. Such definite relief of symptoms in the chronic form of the disease justifies a thorough trial of such measures in other cases of Addison's disease.

Clinical Notes, Suggestions, and New Instruments

NEW ANGULAR UTERINE DRESSING AND SPONGE-HOLDING FORCEPS

ALBERT M. CRANCE, M.D., GENEVA, N. Y.

The instrument shown in the accompanying illustration was designed chiefly for use as sponge-holding forceps for the application of medication through a vaginal speculum, as well as to serve as sponge-holding forceps for employment

in gynecologic surgery. The instrument is shaped in such a way as to permit direct entrance into the speculum, and, because of the angular arrangement of the handle, the operating hand is entirely below the operator's line of vision.

There is a catch provided between the ring handles for keeping the forceps locked during the dressing or sponging manipulation.

The size of the instrument corresponds to the size of the average speculum in use. It is now in process of manufacture by the Kny-Scheerer Corporation.

Schnirel Building.

New angular uterine dressing and sponge-holding forceps.

A METHOD FOR MAINTAINING PRESSURE OVER ELBOW IN OLECRANON BURSITIS (MINER'S ELBOW), WITHOUT IMMOBILIZING THE JOINT

GUY S. VAN ALSTINE, M.D., CHICAGO

The usual treatment of a recent olecranon bursitis with nonpurulent effusion is aspiration, followed by bandage or adhesive strap pressure to favor adhesion of the bursal walls and prevent repeated filling of the sac. This I was formerly unable to accomplish successfully without immobilizing the elbow joint—and immobilization incapacitates.

The accompanying reproduction of a photograph illustrates a bandage cut from an automobile inner tube which, I find, will maintain sufficient pressure to bring about obliteration of the bursal sac without greatly inhibiting the joint action. With this device my patients have been able to continue their usual occupations while under treatment. This treatment is not suitable in old chronic cases with thickened, fibrous sacs, or in acute suppurative bursitis.

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