Diagnosis and Treatment of Growth Hormone Deficiency

Growth retardation may be secondary to a variety of diseases including growth hormone deficiency. This most commonly results from tumor or an idiopathic pituitary defect and may be associated with deficiencies of ACTH, thyroid-stimulating hormone and gonadotropins. Plasma growth hormone is measured by immunoassay techniques. Fasting growth hormone concentrations are normally very low. Therefore stimuli which are known to elicit growth hormone secretion are utilized to unveil deficiency. Subnormal growth hormone responses to hypoglycemia induced by intravenous insulin and to arginine infusion establish the diagnosis. Administration of human pituitary extracts may establish normal growth over periods of several years.

MICHAEL PERLEY, M.D.

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Kaplan SA: Human Growth Hormone. Disease-A-Month. Chicago, The Yearbook Medical Publishers Inc, Dec 1968

Treatment of Hemophilia With Newer Blood Factors

The management of the hemophilic patient has taken great strides in recent years. Fresh frozen plasma remains the agent of choice in the control of minor bleeding and hemarthroses. The introduction of partially purified protein fractions derived from human plasma has facilitated considerably the management of the severe bleeder, as well as providing specific therapy directed toward the particular type of hemophilia. Cryoprecipitates and AHG concentrates are commercially available for use in Factor VIII (AHG) deficiency. Elective operation can now be performed in cases which hitherto had a high mortality. These agents also show promise in the management of hemophiliacs with acquired resistance to plasma infusion. Recently a stable purified prothrombin complex has been introduced which has proven efficacious in the treatment of Factor IX (PTC) deficiency.

GARSON H. TISHKOFF, M.D.

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Hoag MS, Johnson FF, Robinson JA, et al: Treatment of hemophilia B with a new clotting-factor concentrate. New Eng J Med 280:581-586, 1969

Suppression of Rh Sensitization

It is now possible to prevent hemolytic disease of the newborn caused by the principal Rh factor. This prevention is accomplished by the prompt destruction of fetal red cells which normally enter the maternal circulation at delivery and stimulate the mother to produce Rh antibody. This destruction of fetal cells is accomplished by the use of a potent anti-Rh gamma globulin.

WILLIAM M. TODD, M.D.

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Use of Plasma Renin Assays In the Evaluation of Patients With Hypertension

The development of reliable methods for the determination of plasma renin has improved the reliability of diagnosis in two types of curable arterial hypertension. The normal stimulus for renin release is a decreased "effective blood volume." If renin secretion remains high despite plasma expansion with salt and water and the maintenance of bed rest, it is probable that an

obstructive reno-vascular lesion is present which creates the impression of volume depletion at the renin secretion site (the juxta-glomerular apparatus). The diagnosis is further supported if the renin in the renal vein blood from the suspect kidney is at least one and a half times as concentrated as that in the venous blood from the other kidney. On the other hand, the aldosterone-secreting tumor of the adrenal inhibits the release of renin by preventing the normal stimuli. Thus, if renin secretion remains low despite sodium depletion, potassium supplementation and exercise, the diagnosis of an aldosteronoma is supported.

RALPH GOLDMAN, M.D.

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The Clinical Use of Propranolol

Propranolol reduces rhythmicity and myocardial contractility by blocking the cardiac beta-adrenergic receptor sites. The ventricular rate has been shown to be significantly reduced in atrial tachycardias, flutter, and fibrillation by oral or parenteral administration. Hypertensive patients have demonstrated systolic and diastolic pressure reductions of approximately 10 mm (mercury) with 120 mg per day orally. Coronary artery disease patients have observed increased exercise tolerance and diminished frequency of anginal attacks with from 40 to 160 mg per day.

The side effects of severe bradycardia, gastrointestinal pain, and congestive failure have been noted in some patients at the higher dose levels.

ALLEN BOWYER, M.D.

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Dwyer EM Jr, Wiener L, Cox JW: Effects of beta-adrenergic blockade (propranolol) on left ventricular hemodynamics and the electrocardiogram during exercise-induced angina pectoris. Circulation 38:250-260, 1968

Richardson DW, Freund J, Gear AS, et al: Effect of propranolol on elevated arterial blood pressure. Circulation 37:534-542, 1968

The Pathogenic Role of the EB Virus

Studies reported over the last year have strongly implicated the EB (Epstein-Barr) virus in the pathogenesis of infectious mononucleosis. The agent is not antigenically related to any known herpes virus but has been also isolated from Burketts and other tumors, although here there is no convincing evidence for its etiologic role. Infectious mononucleosis only occurs in antibody-negative persons and after this disease antibody titers which are elevated for life, can be sensed by complement fixation and indirect fluorescent antibody assay. Antigen associated with the agent has been seen in white blood cells during infectious mononucleosis and typical symptomatology was produced in a single patient by administration of the EB virus.

THOMAS MERIGAN, M.D.

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Niederman JC, McCollum RW, Henle G, et al: Infectious mononucleosis—Clinical manifestations in relation to EB virus antibodies. JAMA 203:205-209, 1968

Amniocentesis

For the woman who has produced anti-Rh antibody a major problem is death of her offspring, either intrauterine or shortly postpartum. A major factor in outcome of the pregnancy is the estimation of the degree of affliction of the fetus. Examination of amniotic fluid has contributed greatly to the solution of this problem.

WILLIAM M. TODD, M.D.

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