ENDOCRINAL EMERGENCIES IN PEDIATRICS

AN ESSAY

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BY

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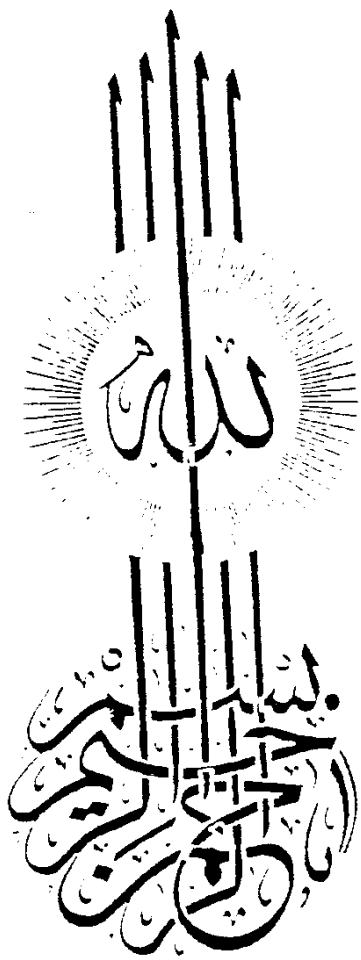

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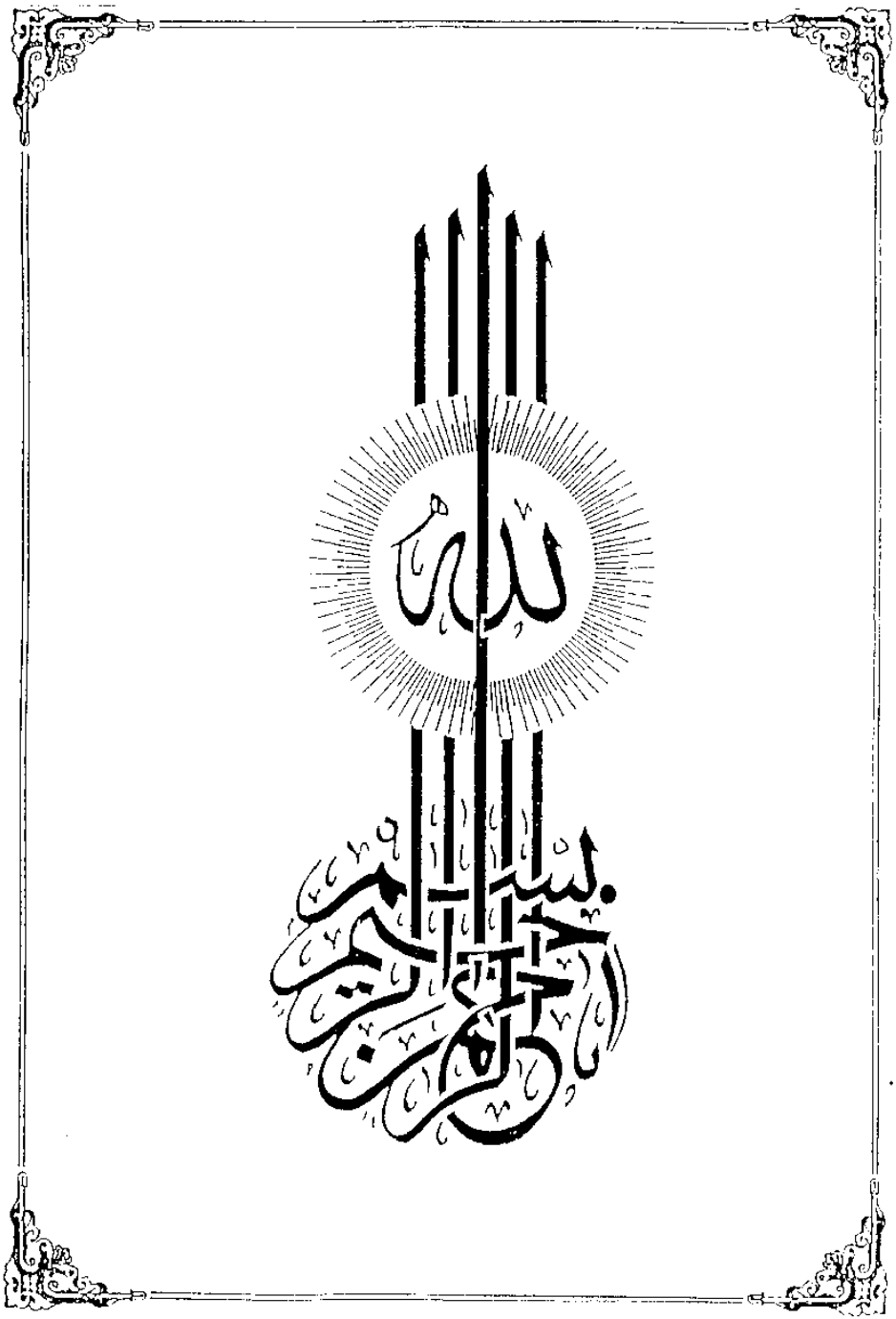

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* * *

ABBREVIATIONS

RDKA : Recurrent diabetic ketoacidosis.

2,3-DPG: 2,3 diphosphoglycerate.

LATS : Long acting thyroid stimulator.

TSAb : Thyroid stimulating antibody.

IgG :Immunoglobulins G.

TSH :Thyroid stimulating hormone.

T_4 : Tyroxine.

T_3 : Triiodothyronine.

TBG : Thyroxine binding globulin.

TRF : Thyroid hormone releasing factor.

TRH : Thyroid releasing hormone.

rT_3 : Serum reverse T_3.

Tg : Plasma Thyroglobulin.

PTU : Propylthiouracil.

ACTH : Adrenocorticotrophic hormone.

CAH : Congenital adrenal hyperplasia.

DOCA : Deoxycorticosterone.

DI : Diabetes insipidus.

DDAVP : Desamino-8-D-arginine vasopressin.

SIADH : Syndrome of inappropriate antidiuretic hormone secretion.

ADH : Antidiuretic hormone.

Ca : Calcium.

PTH : Parathyroid hormone.

$1,25[OH]_2$ D : 1,25-dihydroxycholecalciferol.

25-OHD: 25-hydroxycholecalciferol.

ECG : Electrocardiogram.

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INTRODUCTION

I N T R O D U C T I O N

Endocrinological emergencies are seen relatively infrequent in general practice with the exception of those related to diabetes mellitus. Their importance, however, outweighs their frequency [Lyen et al., 1984]. The diagnosis is often delayed missed because the symptoms of presentation are usually nonspecific. Consideration of the condition is frequently sufficient to secure the diagnosis. They may present as an immediate threat to life, yet early recognition and prompt treatment is effective, potentially life- saving and frequently returns the patient to a full measure of health [Hochman, 1984]. Most endocrinological emergencies require hospital treatment, the principal exception being hypoglycemia in insulin-treated diabetes [Black, 1979].

AIM OF THE ESSAY :

The aim of this essay is to discuss in details the clinical picture of endocrinal emergencies in pediatrics in order to pave the way for an early diagnosis of conditions. The proper treatment will be also discussed, for all these pediatric endocrine emergencies.

DISORDERS OF CARBOHYDRATE METABOLISM

DIABETIC KETOACIDOSIS

DEFINATION

Diabetic ketoacidosis is considered to be present when there is hyperglycemia with a blood glucose concentration exceeding 300 mg/dl, Ketonuria with total ketones [B-hydroxybutyrate and acetoacetate] in serum exceeding 3 mmol/L or positive at a 2:1 solution in serum or undiluted urine with sodium nitroprusside reaction, and acidosis with PH < 7.30 or serum bicarbonate less than 15 mEq/L [Hayford,1979].

Diabetic ketoacidosis can occur with blood glucose levels less than 300 mg/dl particularly if precipitated by vomiting, accompained by reduced carbohydrate intake while daily insulin adminestration is continued[Murno et al., 1973].

GENERAL CONSIDERATIONS

Ketoacidosis is a life-threatening complication of diabetes. it may be present in up to 30% of newly diagnosed insulin-dependent diabetics and still accounts for about 65% of all admissions of diabetics under 19 years of age. The mortality is small but significant , most reports

quoting a mortality rate of under 3% of children presenting with ketoacidosis, ideally, it should be zero [Lyen et al., 1984].

Precipitating factors, even for initial presentation , include stress such as trauma, infections, vomiting, and psychological disturbances. Recurrent episodes of ketoacidosis in established diabetic patients usually imply deliberate errors in recommended insulin dosage and indicate psychological disturbances or pleas to be removed from a home environment preceived to be stressful or intolerable [Sperling,1979]. Recently a program designed to prevent recurrent diabetic ketoacidosis [RDKA], based on the assumption that diabetes education in conjugation with appropriate use of and adherence to insulin therapy should eliminate all RDKA[Golden et al.,1985].

DIFFERENTIAL DIAGNOSIS

diabetic ketoacidosis in children must be distinguished from the following :

1. Hyperosmolar, hyperglycemic, nonketotic coma, which is rare in childhood and characterized by the absence of ketonemia or ketonuria. In this circumstance blood glucose concentation is usually greater than

500 mg/dl and serum osmolality exceeds 300 mOsm/kg [Holliday,1978].

2. Lactic acidosis, which is characterized by blood lactate values in excess of 7 mmol/L, values rarely encountered in pure diabetic acidosis.
3. Salicylate intoxication is characterized by severe metabolic acidosis, but usually without marked hyperglycemia. Plasma glucose is usually less than 250 mg/dl [Baun,1979].
4. Acute abdominal conditions : Abdominal pain may be sufficiently severe to suggest acute pancreatitis or acute appendicitis even when diabetic ketoacidosis is recognized, since a genuine acute abdominal condition may also be present. Unlike in the adult, acute pancreatitis is very rare in the child with diabetic ketoacidosis [Valerio,1976].

PATHOPHYSIOLOGY [Fig.1]

The genesis of diabetic ketoacidosis can now be understood in the context of hormonally mediated events in which absolute or relative insulin deficiency and excessive secretion of the counterregulatory hormones glucagon, cortisol, catecholamines and growth hormone to produce hyperglycemia, hyperlipidemia and ketonemia with acidosis.[Jordan,1983].

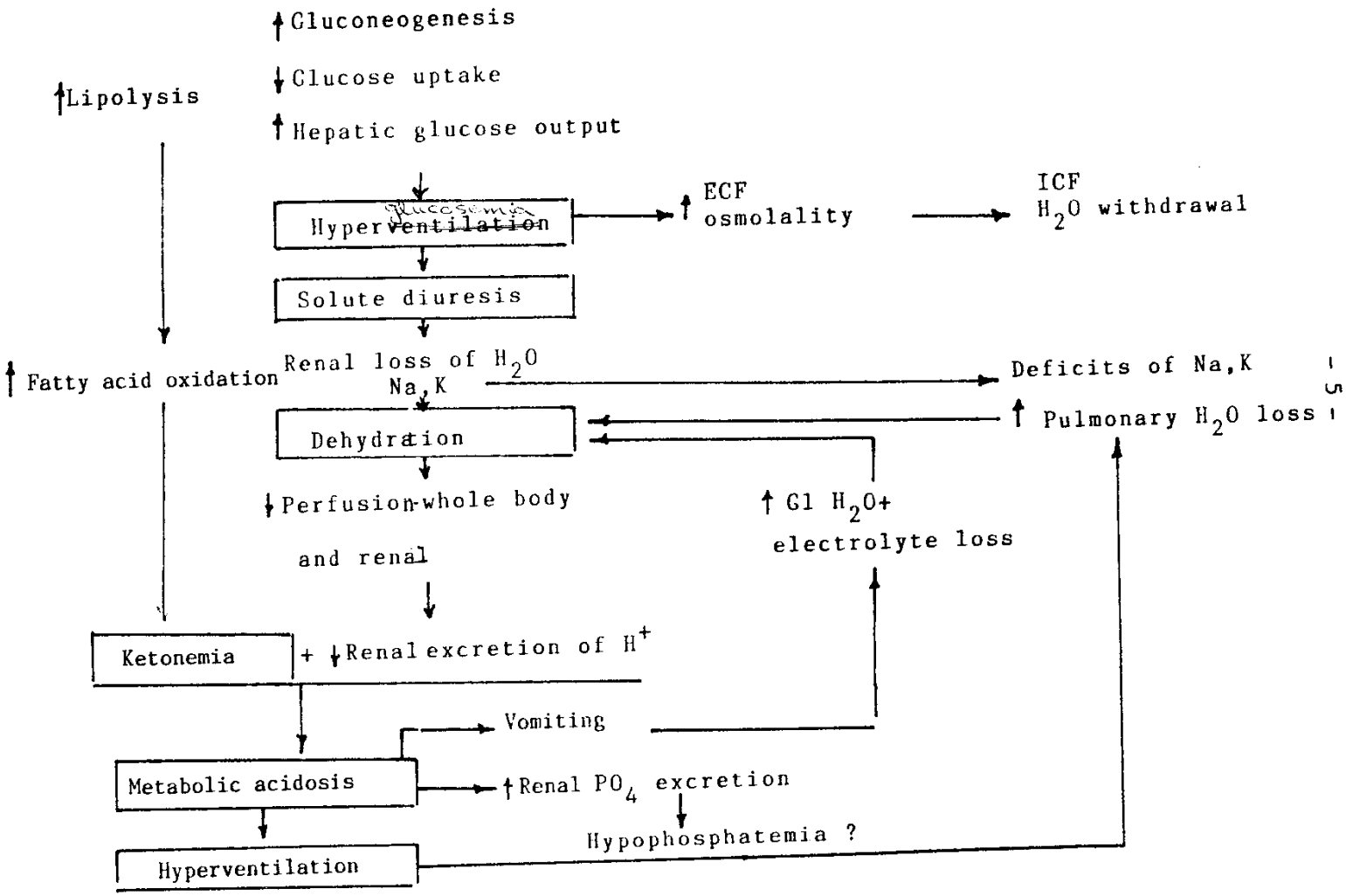

Fig.[1] Pathogenesis of diabetic acidosis.
[Kaye R. : Pediatric therapy,1980].

I. ROLE OF INSULIN

In normal physiology, insulin secretion is exquisitely fine-tuned to metabolic demands. Superimposed on a steady basal rate of insulin secretion are episodic spikes of insulin release that are coincident with food intake and proportional to the resultant glycemic excursion. This insulin stimulates anabolic processes in three major tissues, liver, muscle, and adipose tissue, to permit glucose utilization and storage of the energy from ingested foodstuffs as glycogen, protein, and fat [Cahill, 1971]. Simulataneously, the increase in insulin concentrations curtails glycogenolysis, gluconeogensis, proteolysis, lipolysis, and ketogensis. conversely, during overnight fasting when insulin concentrations are basal, these anabolic processes are curtailed while catabolic processes are activated to supply energy needs. These catabolic processes include glucose production via glycogenolysis and gluconeogenesis. Thus normal metabolism fluctuates periodically between the fed, high-insulin anabolic state and the fasted , low-insulin catabolic state [Sperling , 1983]. These events are summarized in table 1.