Volume 41, No. 1
July 1974

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

Editorial Views

Anesthesia and the Endocrine Pancreas

ANESTHESIOLOGISTS have an appreciation of the role of glucose as the sole energy source for the central nervous system. Many, however, will be tempted to bypass Greene's review of insulin activity as being relatively unimportant to the clinical practice of anesthesia. A brief survey of metabolic control mechanisms related to insulin will reveal this to be a serious misjudgment and indicate that we must pay more, rather than less, attention to this important field, in which knowledge is rapidly accumulating at the present time.

In the fasting state sufficient glucose to meet the energy needs of the central nervous system is derived from the liver by glycogen breakdown (glycogenolysis) or by glucose synthesis from lactate (gluconeogenesis). The energy demands for most other body organs are met by utilization of free fatty acids and ketone bodies derived from lipolysis of neutral fat previously stored in adipose depots. Many hormones (catecholamines, glucagon, glucosteroids, and growth hormone, for example) are capable of stimulating glycogenolysis, gluconeogenesis, and lipolysis, and therefore a variety of modulating mechanisms occur in the stimuli for breakdown of the body's energy storage compounds. Only insulin, however, stimulates the formation of these storage compounds. (See figure 1.)

In the fasting state, basal insulin secretion occurs from the larger of two pools postulated to exist in the beta cells of the pancreas.[1] Basal insulin secretion is not a simple linear function of blood sugar concentration,[2] but probably is also responsive to levels of amino acids, free fatty acids, and ketone bodies and to an adrenergic inhibitory control mechanism. The primary role of basal insulin secretion is to modulate the metabolic response to fasting.

Acute insulin secretion in response to a hyperglycemic stimulus occurs from the smaller storage pool of insulin and is conditioned in magnitude by the prior level of basal insulin secretion. It is more sensitive to inhibition by catecholamines than is basal insulin secretion.[3] (See figure 2.)

Obviously, the involved metabolic interactions are far from simple,[4] and most attempts to study anesthetic influences on insulin secretion suffer from failure to control the fasting state, adrenergic activity, or other important variables. No attempt has been made to separate basal from stimulated insulin secretion. Some studies are based on a bioassay and others on an immunoassay, which would lead to difficulties in interpretation. Nevertheless, the data suggest that some anesthetics, for example halothane, appear to inhibit insulin release from the pancreas. Could a cardiac arrhythmia in a patient awakening from halothane be attributed to hypokalemia induced by endogenous insulin secretion as the pancreas regains its sensitivity to hyperglycemia? Such patients undoubtedly are hyperglycemic from fluid infusions, and insulin does promote intracellular transport of both glucose and potassium. What are the interactions of vasopressors, neostigmine, alpha and beta adrenergic block-

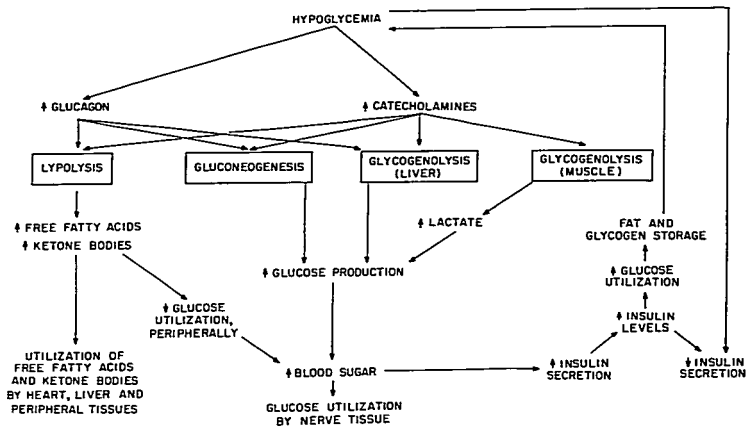

FIG. 1. The utilization of metabolic fuels in the fasting state.

ing drugs, atropine, and other commonly used anesthetic adjuvants on basal and stimulated insulin secretion and of what importance are these interactions in the anesthetized patient? Undoubtedly the answers to these and similar questions will provide information important to good patient care. The best way to be able to utilize such knowledge is to attempt to remain current as the field develops.

EDWARD A. BRUNNER, M.D., PH.D.
Department of Anesthesia
Northwestern University Medical School
Chicago, Illinois 60611

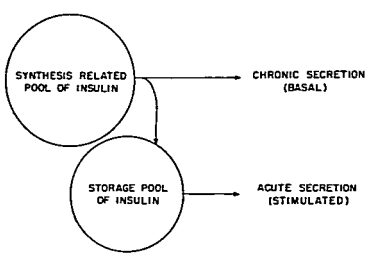

FIG. 2. Two-compartment model of insulin secretion (after Port and Bagdade[1]).

References

1. Port D Jr, Bagdade JD: Human insulin secretion: An integrated approach. Annu Rev Med 21:219–240, 1970
2. Port D Jr, Pupo AA: Insulin response to glucose: Evidence for a two pool system in man. J Clin Invest 48:2309–2319, 1969
3. Robertson RW, Port D Jr: Adrenergic control of basal insulin in man. Diabetes 20: suppl 1:322, 1971
4. Steiner DF, Freinkel N (editors): Handbook of Physiology, Section 7: Endocrinology, Volume 1. Endocrine Pancreas. American Physiological Society, Washington, D. C., 1972