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ABSTRACT BOOK

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23. INVITED

GLUCOMETABOLISAM AND DEVELOPMENT OF INSULIN RESISTANCE IN SURGICAL PATIENT;

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Anesthesiologist in their everyday practice confronts with consequences of the pain and surgical trauma on physiological and homeostatic mechanisms in patient.

This lecture incorporates the review of the present literature, the novel data and proposes how should anesthesiologist prevent insulin resistance and attenuate the stress response in order to maintain glucose regulation. However, up to day controversies still exist and opened for research.

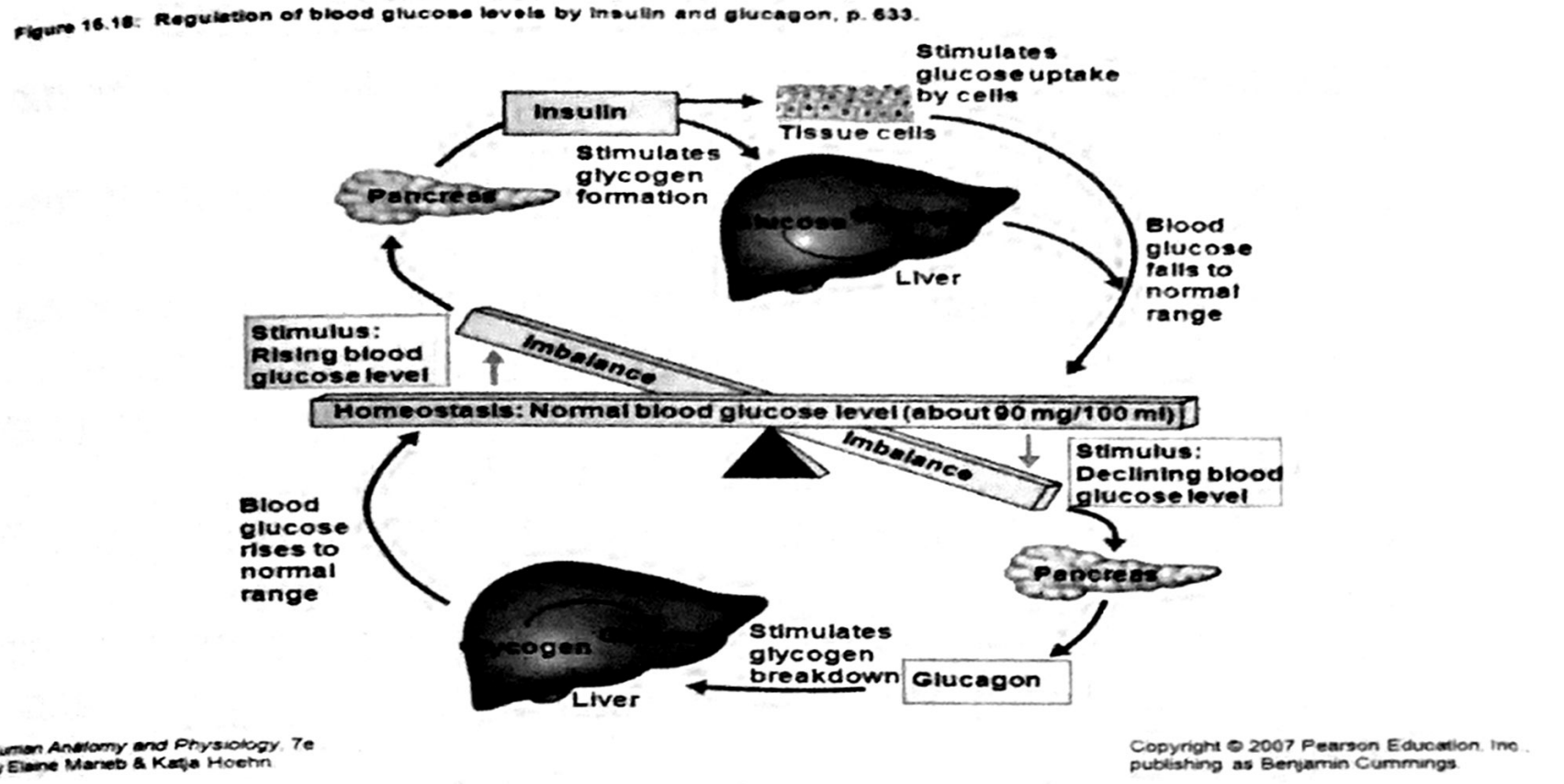
Literature review

The surgical stress response unavoidably induces activation of hypothalamus-pituitary hormonal axis and sympathetic nervous system (1, 2, 3). This increases the net catabolic action, and disturbs glucoregulation results (3, 4). Due to of increased effects of stress hormones, neoglucogenesis, glycogenolysis, glycaemia increase, while insulin secretion (4, 5) and insulin utilization are decreased (6,7). These metabolic changes are dependent of the factors like the magnitude of the surgical trauma, pain, type of anesthesia, anxiety, preoperative fasting as well as the occurrence of perioperative complications (8,9,10,11,12).

The era of evidence-based medicine refers that the impaired glucose-regulation equally affects diabetic and nondiabetic surgical patients. Strong evidence, show that disrupted glucoregulation in the perioperative and postoperative period significantly affects the final outcome and increases the morbidity and mortality in surgical patients (5).

Surgical trauma is followed by many metabolic disorders and one of them is related to the utilization of glucose by the tissues. After surgical trauma, pancreatic cells become "unfunctional" and ability to synthetize insulin is "abolished"(13). Due to this, glycaemia levels are elevated, often reaching hyperglycemic level. This metabolic state noted as "stress hyperglycemia" or "diabetes of surgery" and it influences the ability of the organism to use the glucose. This state plus the increased level of stress hormones (cortisol, catecholamines, glucagon), results in development of insulin resistance (muscle and fat tissue cell which are usually sensitive to insulin start to be insensitive)(14,15).

Image 1. Homeostasis for glucose-regulations



Taken from Nygren JO, Thorell A, Soop M, et al. Perioperative insulin and glucose infusion maintains normal insulin sensitivity after surgery. Am J Physiol 1998; 275:140-8.

However, the problem of insulin resistance is not only based on simple glucagon, insulin levels, hepatic or pancreatic involvement it involves a series of complex Inulin receptors within and out of the cells (14,15).

Literature reports that 38% of all hospitalized patient will develop hyperglycemia at any time during the hospitalization (16) and 40% of them won't have anamnesis of diabetes (18). Additionally, 51 % of nondiabetic patients undergoing surgery, even undergoing minor surgery, will develop insulin resistance and these patients have six-fold higher risk for infections and ten times higher risk of mortality (18).

Historically, the problem of glucose regulation was studied from different endocrinological, metabolic and hormonal aspects. Most of the studies were made in order to find the" ideal" way for stabilizing the glucose level in diabetic patients.

The fact that the endocrine sequels of the trauma on insulin level and glycaemia are not present just perioperative, but also postoperatively, complicates the overall picture. Additionally, the complexity of the influence of anesthesiologic actions on homeostatic mechanisms complements the complexity of glucoregulation and its interference.

Therefore, one of the key goals in anesthesiological procedures is using all available means for prevention and suppression on expected stress response in order to decrease the occurrence of glucometabolic disturbances.

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