

Hyperglycemia in Critical Illness: A Review

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Abstract

Hyperglycemia is commonplace in the critically ill patient and is associated with worse outcomes. It occurs after severe stress (e.g., infection or injury) and results from a combination of increased secretion of catabolic hormones, increased hepatic gluconeogenesis, and resistance to the peripheral and hepatic actions of insulin. The use of carbohydrate-based feeds, glucose containing solutions, and drugs such as epinephrine may exacerbate the hyperglycemia. Mechanisms by which hyperglycemia cause harm are uncertain. Deranged osmolality and blood flow, intracellular acidosis, and enhanced superoxide production have all been implicated. The net result is derangement of endothelial, immune and coagulation function and an association with neuropathy and myopathy. These changes can be prevented, at least in part, by the use of insulin to maintain normoglycemia.

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Abbreviations: (ADMA) asymmetric dimethylarginine, (eNOS) endothelial-derived nitric oxide synthase, (GAPDH) glyceraldehyde-3-phosphate dehydrogenase, (GH) growth hormone, (GLUT) glucose transporter, (ICAM-1) intercellular adhesion molecule-1, (ICU) intensive care unit, (IGF) insulin growth factor, (IGFBP) insulin-like growth factor-binding protein, (IL) interleukin, (iNOS) inducible nitric oxide synthase, (IRS) insulin receptor substrate, (JNK) c-jun N-terminal kinase, (MAPK) mitogen-activated protein kinase, (NADH) nicotinamide adenine dinucleotide, (NF) nuclear factor, (NO) nitric oxide, (NOS) nitric oxide synthase, (PARP) poly-ADP ribose polymerase, (PEPCK) phosphoenolpyruvate carboxylase, (PI-3-kinase) phosphoinositide-3-kinase, (PKC) protein kinase C, (PPAR) peroxisome proliferator-activator receptor, (TNF) tumor necrosis factor

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