Perioperative glycaemia control, Perioperative glycemic control

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Abstract: Objectives: Review perioperative glucose control in diabetic and non diabetic patients. **Data Summary:** Maintenance of blood glucose homeostasis is of great importance to the survival of the human being. Both elevated and reduced levels of blood glucose trigger hormonal responses to initiate pathways designed to restore glucose homeostasis. Hyperglycaemia is a common phenomenon in the perioperative period, linked to the preoperative metabolic state of the patient, neuroendocrine stress response, and acute perioperative insulin resistance, as well as the intraoperative management. Treatment recommendations are categorized based on the type of diabetes, nature and extent of the surgical procedure, antecedent pharmacological therapy, and state of metabolic control before surgery. **Conclusion:** Management of glucose concentrations has undergone drastic changes in the past decade, which are reflected by significant changes in recommendations for glucose management from national organizations with extensive expertise in glucose control.
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Introduction

Hyperglycaemia is a common phenomenon in the perioperative period, linked to the preoperative metabolic state of the patient, neuroendocrine stress response, and acute perioperative insulin resistance, as well as the intraoperative management.[1]

While practitioners and hospitals often have protocols and practice standards in place to manage those known to have diabetes, these previously identified patients are just a fraction of those at risk for abnormal inpatient glycaemia control. [2]

Higher IR and abnormal insulin secretion were found in RA patients in comparison with SLE patients. Hence, there is a pressing need for strategies for control of inflammation, dyslipidaemia, and evaluation of IR in patients with SLE and RA and for intervention studies for modifying IR in SLE and RA patients and whether drugs that ameliorate IR such as metformin and thiazolidinedione could be part of the treatment and regimen of these patients.[3]

Objectives:-

Review perioperative glucose control in diabetic and non diabetic patients.

Data summary:-

Data Source:-from previous Literatures, reviews and studies as well as

Medical websites (pubMed, MD consult) and Scientific Journals.

Study Selection:-selection was done by supervisors for studying new

Advancement in perioperative glycaemia control.

Data Extraction:-In this review data from published studies were manually

extracted and summarized.

Data Synthesis:-In this review the data found that several studies of

Perioperative glycaemic control were included. There are different methods for perioperative glycaemic control. We got our data from studying the different approaches of glycaemic control preoperative, intraoperative and postoperative.

Glucose metabolism

Glucose is metabolised inside the cell by the pathway of glycolysis.glucose is normally phosphorylated to form glucose- 6 – phosphate.then glucose- 6 – phosphate is converted to fructose- 6 – phosphate which after subsequent reactions produces pyruvate or lactate(or both). Aerobic tissues metabolize pyruvate to acetyl- CoA, which can enter the citric cycle for complete oxidation to CO_2 and H_2O, linked to energy production.[4]

Regulation of glucose metabolism

Maintenance of blood glucose homeostasis is of great importance to the survival of the human organism. Both elevated and reduced levels of blood glucose trigger hormonal responses to initiate pathways designed to restore glucose homeostasis. The following table summarizes the hormonal control of blood glucose levels. (Table 1).[5]

Metabolic effect of surgery and anaesthesia

Living organisms constantly undergo adjustments to adapt to ever-changing internal and external conditions. These natural alterations have

been termed *allostasis* and include diurnal variations in temperature and cortisol production, changes in metabolism due to eating and drinking, and the cardiopulmonary responses to exercise.[6]

Stress occurs when allostatic mechanisms are insufficient for maintaining homeostasis, thus causing the organism to significantly alter many of its functions to reduce and contain the stressful threat.[7]

The Hypothalamus-Pituitary Axis

Many allostatic, as well as homeostatic, changes are controlled by feedback control systems centred on the hypothalamic-pituitary axis. The response to surgical and traumatic stress is triggered by hypothalamic activation secondary to afferent neuronal input from an area of injury or emotional activity centred in the limbic system and humeral factors, such as the inflammatory cytokines tumour necrosis factor-α (TNF-α), interleukin-1 (IL-1), and IL-6. [8]

Hypothalamic activation increases the activity of the sympathetic nervous system, which increases the output of the cardiovascular system and stimulates the adrenal medulla to secrete epinephrine and the pancreas to secrete glucagon. Hypothalamic activation also stimulates or reduces the secretion of the various hypothalamic releasing factors (e.g., corticotrophin-releasing hormone [CRH], growth hormone–releasing hormone [GHRH]). The hypothalamus, the body's thermostat, also controls body temperature. Stimulation by mediators such as IL-1β (endogenous pyrogen) causes fever via a biphasic mechanism; the first phase is mediated by increased intracellular ceramide and the second by cyclooxygenase-2 (COX-2)–mediated prostaglandin E_2.[9]

Modulation of the Stress Response

The Preoperative Stress Response

Patients undergoing elective surgery often suffer from preoperative anxiety, a state of psychological stress involving low-level activation of the hypothalamic-pituitary axis and possibly cytokines.[10]

Preoperative medication is given to temper anxiety, thus attenuating the stress response. Midazolam as a premedication reduces anxiety and limits increases in intraoperative epinephrine and, possibly, nor epinephrine and cortisol.[11]

The Intraoperative Stress Response

Inhaled Anaesthetics

Volatile anaesthetics minimally suppress the stress response. There is conflicting evidence as to whether volatile agents prevent surgically induced catecholamine secretion, although, among the volatile agents, enflurane is most capable of blocking intraoperative catecholamine surges.[12]

Intravenous Anaesthetics

Compared with benzodiazepines, the α_2-adrenergic agonists clonidine and dexmedetomidine are more successful at blunting the hemodynamic and catecholamine responses to intubation and intraoperative stress. The α_2-agonists blunt the stress response by reducing norepinephrine release from central and peripheral nerve endings.[13]

Some intravenous anaesthetics partially modify the endocrine and metabolic components of the stress response. Sodium thiopental (5.0-5.1 mg/kg) does not prevent increases in catecholamine concentrations after intubation.

Opioids

Opioids are capable of blunting the stress response. Fentanyl (50 μg/kg) plus halothane and nitrous oxide abolished the elevations in cortisol, GH, and glucose normally seen during hysterectomy. Morphine (1 mg/kg) reduced GH and cortisol elevations during abdominal surgery.[14]

Neuraxial Anaesthesia

Neuraxial anaesthesia is more effective than general anaesthesia in attenuating the stress response. The ability of such anaesthesia to modify the intraoperative stress response depends on the level of the blockade, the location of the surgery, and the drugs used.[15]

Neuraxial anaesthesia for lower abdominal and lower extremity surgery reduces or abolishes the hyperglycaemic response to surgery, likely because of the lack of stimulation of hepatic glycogenolysis due to a reduced epinephrine response or blockade of hepatic sympathetic pathways or both.[16]

Thermoregulation

Another way of minimizing the stress response is maintaining perioperative normothermia.Intraoperative hypothermia followed by postoperative rewarming stimulates the response. Hypothermic patients (35.3°C core temperature) had higher postoperative plasma norepinephrine and epinephrine concentrations than their warmer counterparts. These elevated catecholamine concentrations are partially the result of postoperative rewarming with shivering and vasodilatation that leads to hypotension. Elevated catecholamine have been postulated to be among the reasons for the greater incidence of myocardial ischemia after vascular surgery in hypothermic patients.[17]

The Postoperative Period

Pain control with epidural opioids or local anaesthetics is unable to lessen many of the metabolic changes associated with such surgery. However, combined epidural opioids/local anaesthetic analgesia appears more effective in attenuating catabolism.[18]

Attempts to modulate the perioperative stress response include nonanaesthetic approaches (e.g., using β-adrenergic antagonists to block cardiovascular end-organ receptors) and α_2-adrenergic agonists to stimulate central receptors. These approaches have

met with some success. Using β-adrenergic antagonists in patients with ischemic heart disease does not reduce the perioperative neuroendocrine or endocrine stress response but just blocks the end-organ response. Such end-organ blockade also has metabolic affects. Administering propranolol to severely burned children decreased their resting energy expenditure, attenuated muscle catabolism, reduced lipolysis, preserved lean body mass, and reduced TNF and IL-1 concentrations without increasing infections.[19]

Perioperative management of diabetic patient

Diabetes mellitus (DM) is a group of metabolic disorders characterized by a chronic hyperglycaemic condition resulting from defects in insulin secretion, insulin action or both. Permanent neonatal diabetes is caused by glucokinase deficiency, and is an inborn error of the glucose-insulin signalling pathway [20]

Glucose Measurement and Monitoring

Ideally, blood glucose should be determined by the central laboratory or onsite blood-gas analyzers; as a rule, point-of-care capillary meters are less reliable, especially in hypoperfused, hypothermic, or anaemic patients. The practitioners should keep in mind that the accuracy varies with each modality and some error is allowed when accuracy of these devices is tested.[21]

Screening of an older, predominantly white, socially representative cohort of patients enrolled in population-based heart disease studies has revealed a prevalence of frank undiagnosed type 2 diabetes in this group, of around 7%. IGT had a prevalence of about 20%. [22]

Risk Evaluation

The evaluation is oriented to identifying underlying cardiac, pulmonary and renal disease, electrolyte disturbances, presence of macro vascular and micro vascular complications, as well as the assessment of antecedent glycaemia control. Adult subjects with diabetes should be considered high risk for cardiac ischemia.[23]

The risk of coronary artery disease is two to four times higher than in the corresponding general population.[24]

Risk factors for postoperative renal dysfunction include advanced age, type 1 diabetes mellitus, preoperative hyperglycaemia, a history of moderate to severe congestive heart failure, a previous coronary artery bypass graft, or pre-existing renal disease (as manifested by an elevated serum creatinine level).[25]

Perioperative Glycaemia Goals

A general theme that emerges in these guidelines is to at least maintain glucose levels 180 mg/dL perioperatively. Although it is recommended to maintain a glucose level of 150 mg/dL in cardiac surgery patients with a complicated ICU course, it should be recognized that the recommendation is not based on a high level of evidence.[26]

Approaches to Management

General Principles

Patients Treated with Diet Alone Patients whose diabetes is well controlled by a regimen of diet and physical activity may require no special preoperative intervention for diabetes.[27]

Oral agents should be discontinued one day beforesurgery. Sulfonylureas increase the risk of hypoglycaemia; in addition, a longstanding controversy exists regarding the vascularеffects of sulfonylureas in patients with cardiac and cerebral ischemia.[28]

Type 1 or Type 2 Diabetes Treated with Insulin

Minor Surgery

Most patients receiving insulin before admission can be treated with conventional subcutaneous insulin therapy. If the surgery is to be performed in the morning in a patient treated with intermediate-acting (NPH) insulin, one half of the total morning dose of NPH insulin should be administered.[29]

Major Surgery

IV infusion of insulin is the standard therapy for the perioperative management of diabetes, especially in type 1 diabetic patients and patients with type 2 diabetes undergoing major procedures. [30]

Several reports have emphasized the advantages of the insulin infusion regimen over subcutaneous delivery.[30]Institutions around the world use a variety of insulin infusion algorithms that can be implemented by nursing staff. Recently, several insulin infusion protocols have been reported in the literature.[30]

These algorithms facilitate communication between physicians and nurses, achieve correction of hyperglycemias in a timely manner, and provide a method to determine the insulin infusion rate required to maintain blood sugars within a defined target range.[31]

In most insulin infusion protocols, orders to "titrate drip" are given to achieve a target blood glucose range using an established algorithm or by the application of mathematical rules by nursing staff. [31]

Two main methods of insulin delivery have been used either combining insulin with glucose and potassium in the same bag (GIK regimen) or giving insulin separately with an infusion pump. The GIK is initiated at a rate of 100 mL/h in a solution of 500 mL of 10% dextrose, 10 mmol of potassium, and 15 U of insulin. Adjustments in the insulin dose are made in 5 U increments according to blood glucose measurements performed at least every 2 hours.[32]

In the United States, separate continuous glucose and insulin infusions are used more frequently than the glucose-potassium-insulin infusion. [30]

Summary

Management of glucose concentrations has undergone drastic changes in the past decade, which

are reflected by significant changes in recommendations for glucose management from national organizations with extensive expertise in glucose control. For example, the American Diabetes Association and the American Association of Clinical Endocrinologists currently recommend starting insulin infusions for critically ill patients with persistent hyperglycaemia (glucose greater than 180mg/dL) and aiming for a target blood glucose range of 140 – 180 mg/dL while the American College of Physicians recommends a target of 140 to 200 mg/dL for insulin therapy in critically ill patients both societies agree that adverse outcomes, including death and hypoglycaemia, are increased in patients that receive intensive insulin therapy.

Physiologic actions of insulin and insulin counter regulatory hormones.

Hormone	Function	metabolic pathways Major affected
Insulin	• promotes fuel storage after a meal • promotes growth	• Stimulate glucose storage as glycogen (muscle and liver). • Stimulate fatty acid synthesis and storage after a high carbohydrate meal. • Stimulates amino acid uptake and protein synthesis.
Glucagon	• Metabolizes fuels • Maintains blood glucose levels during fasting.	• Activates gluconeogenesis and glycogenolysis (liver) during fasting. • Activates fatty acid release from adipose tissue.
Epinephrine	• Mobilizes fuels during acute stress	• Stimulates glucose production from glycogen (muscle and liver). • Stimulates fatty acid release from adipose tissue.
Cortisol	• Provides for changing requirements over the long term.	• Stimulates amino acid mobilization from muscle protein • Stimulates gluconeogenesis. • Stimulates fatty acid release from adipose tissue.

Short running head:
Perioperative glycemic control.

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