HYPERMETABOLIC STATE IN ICU

An essay
Submitted For Partial Fulfillment of Master Degree
In
Intensive Care Medicine

By
Ahmed Fawzy Mohammed Attiya
M.B.B.CH – Tanta University

Supervised By

Prof.Dr. / Soheir Abbas Mohammed
Professor of Anesthiology & Intensive Care
Faculty of Medicine, Ain Shams University

Prof.Dr. / Ahmed Aly Fawaz
Professor of Anesthiology & Intensive Care
Faculty of Medicine, Ain Shams University

Dr. / Amal Hamed Rabie
Lecturer of Anesthiology & Intensive Care
Faculty of Medicine, Ain Shams University

Department of Anesthiology and Intensive care

(2013)

حالات زيادة الأيض بالعناية المركزة

رسالة
مقدمه من

أحمد فوزى محمد عطيه
بكالوريوس الطب والجراحة – جامعةطنطا

توطئه للحصول على درجة الماجستير فى
العناية المركزة

تحت اشراف

الأستاذ الدكتور/ سهير عباس محمد
أستاذ التخدير والعناية المركزة
كلية الطب – جامعة عين شمس

الأستاذ الدكتور/ أحمد على فواز
أستاذ التخدير والعناية المركزة
كلية الطب – جامعة عين شمس

الدكتور/ أمل حامد ربيع
مدرس التخدير والعناية المركزة
كلية الطب – جامعة عين شمس

قسم التخدير والعناية المركزة

(٢٠١٣)

Contents

Abbreviation

CRHs	Counter-regulatory hormones
ICU	Intensive care unit
BMR	Basal metabolic rate
BEE	Basal energy expenditure
REE	Resting energy expenditure
kcal	Kilocalories
TBSA	Total body surface area
IL-1 AND 6	Interleukin 1 and 6
TNF	Tumor necrosis factor
TBI	Traumatic brain injury
FFAs	Free fatty acids
HPA	Hypothalamo-pituitary-adrenal axis
ACTH	Adrinocorticotropin hormone
IL-4	Interleukin 4
IL-10	Interleukin 10
GH	Growth hormone
GHBP	Growth hormone binding protein
IGF-1	Insulin like growth factor 1
IGFBP-1	Insulin like growth factor binding protein 1
TSH	Thyroid stimulating hormone
T3	Tri-iodotyrosine
T4	Tetra-iodotyrosine
TRH	Thyrotropin releasing hormone
PRL	prolactin
LH	Luteinizing hormone
TH1	T-helper lymphocyte type 1
TNF-α	Tumor necrosis factor-α
IL-2	Interleukin 2
IFN-γ	Interferon-γ
TH2	T-helper lymphocyte type 2
IR	Insulin receptor
IL-6	Interleukin 6
IL-8	Interleukin 8
IL-12	Interleukin 12
IL-18	Interleukin 18
HMGB1	High mobility group box 1
ASPEN	American society for parenteral and enteral nutrition
BMI	Body mass index
WT	Weight
HT	Height
M^2	Squired meter
MAC	Midarm circumference
TSF	Triceps skin fold thickness

MUST	Malnutrition Universal Screening Tool
BABEN	British Association for Parenteral and Enteral Nutrition
UK	United Kingdom
NRS	Nutritional Risk Screening
ESPEN	European society for parenteral and enteral nutrition
SNAQ	Short Nutritional Assessment Questionnaire
NB	Nitrogen balance
I	Intake
U	Urine
F	Faeces
S	Dermal loss
V O2	Oxygen consumption
V CO2	Carbon dioxide excretion
UUN	Urine urea nitrogen
FIO2	Fraction of inspired oxygen
PEEP	Positive end expiratory pressure
PUFA	polyunsaturated fatty acid
EN	Enteral nutrition
PEG	Percutaneous endoscopic gastrostomy
PN	Parenteral nutrition
TPN	Total parenteral nutrition
PPN	Peripheral parenteral nutrition
CRBSI	Catheter-related blood-stream infection
CVC	Central venous catheter
PICC	peripherally inserted central catheter
CPP	Cerebral perfusion pressure
SBI	Secondary brain insults
BCAA	Branched chain amino acids
ARDS	Acute respiratory distress syndrome
Co2	Carbon dioxide
ATP	Adenosine triphosphate
NO	Nitric oxide
rHGH	Recombinant human growth hormone
NOS	Nitric oxide synthase
eNOS	Endothelial nitric oxide
nNOS	Neuronal nitric oxide
iNOS	Inducible nitric oxide
DNA	Deoxyribonucleic acid
PGs	Prostaglandin
LTs	Leukotrienes
EPA	Eicosapentaenoic acid
ROS	Reactive oxidative species
SIRS	Systemic inflammatory response syndrome

List of Figures

List of Tables

Aim of the essay

The aim of this essay is to discuss briefly the hypermetabolic state in the ICU considering its causes, pathophysiology, diagnosis and management.

Acknowledgement

First and foremost, thanks to **ALLAH**, the most beneficial and merciful.

In few grateful words, I would like to express my greatest thanks to all my professors who helped me throughout this work.

I wish particularly to express my deepest gratitude and appreciation for unfailing support; valuable advice and generous help and patience rendered me by **Prof.Dr. / Soheir Abbas Mohammed**, professor of anesthesiology and intensive care, Ain Shams University. She sacrificed a great deal of her precious time and effort guiding me throughout the preparation of this essay.

Also, I want to thank **Prof.Dr. / Ahmed AlyFawaz**, professor of anesthesiology and intensive care, Ain Shams University for his great help and advice.

I am indeed immensely indebted and deeply grateful to **Dr. / AmalHamedRabie**, lecturer of anesthesiology and intensive care, Ain Shams University, for her sincere care, untiring effort and her great assistance during every step and every detail in this essay.

Finally, I give all the thanks and grateful feeling and gratitude to my family for their unlimited support and help they offered me throughout my life.

Ahmed Fawzy Mohammed

2013

Introduction

Introduction

Injury and infection evoke in the host a hypermetabolic inflammatory response and a compensatory hypometabolichypoimmune response. The magnitude of the response is proportional to the extent of injury **(Russell, 2006).**

The hypermetabolic response in critically ill patients is characterized by a hyperdynamic circulatory response with massive protein and lipid catabolism, total body protein loss, muscle wasting, peripheral insulin resistance, increased energy expenditure, increased body temperature, increased infection risks, and stimulated synthesis of acute phase proteins located in the liver and intestinal mucosa **(Atiyeh et al ., 2008).**

These responses occur in all traumas, surgical or critically ill patients, but the severity, duration and magnitude is uniquely severe for burn patients (**Herndon and Tompkins, 2004).**

Metabolic changes after trauma occur in two different phases, termed the *"ebb" phase* and the *"flow" phase*. The "*ebb" phase* is initiated within minutes after trauma and persists for several hours after the initial insult. It is characterized by a decline in body temperature and oxygen consumption, aimed at reducing posttraumatic energy depletion. However, the brief duration of this phase limits its clinical relevance. The *"flow" phase*, which occurs after compensation of the state of traumatic-hemorrhagic shock, is associated with an increased metabolic turnover, activation of the innate immune system and induction of the hepatic acute-phase response **(Keel and Trentz, 2005).**

The posttraumatic catabolic state requires an adjusted energetic balance with early protein substitution and hypercaloric nutrition **(Slone, 2004).**

The specialized nutritional support for severely injured patients includes the administration of "immune nutrient cocktails" which have been shown to improve the survival of septic patients during the intensive care period **(Griffiths, 2003).**

Definition and causes of hypermetabolic state

Definition and causes of hypermetabolic state

Critically ill patients are characterized by wide variations in their carbohydrate, lipid and amino acid (protein) metabolism. Such variations can lead to increase in their energy requirement with accelerated protein catabolism and ultimately alterations of their immune and gastrointestinal systems. In the normal weight person, the metabolic response to injury causes an increase in protein and energy requirements. As a result, endogenous substrates serve as fuel sources and as precursors for protein synthesis. This response is mediated by counter-regulatory hormones (CRHs) such as epinephrine, glucagon, cortisol, and growth hormone, which regulate the flow of endogenous substrates between the various organs and tissues. In addition, cytokines such as tumor necrosis factor-α and interleukin-1β may play an important role in this systemic response inducing hyperglycemia ***(Biolo et al., 2002).***

Critically ill patients exhibit a characteristic metabolic response to severe illness or traumatic injury independent of the cause. Both types of inciting events activate the immune system and induce a coordinated systemic inflammatory response syndrome aimed at limiting the extent of injury and restoring normal physiologic processes. The specific nature and extent of this response can vary widely based on the causative insult. When physiologically controlled, this response can facilitate recovery. When uncontrolled, it can impair host responses to critical illness ***(Martindale et al., 2002).***

Several studies have shown that metabolically stressed and malnourished patients have more negative outcomes and higher health-care costs. Patients with continuous energy deficits have a higher ventilator-dependence rate, longer intensive care unit (ICU) stay and higher mortality***(Renee and Roschelle, 2009).***