Introduction

The human gastrointestinal tract is colonized by a dense population of microorganisms, referred to as the bacterial flora. Although the gut provides a functional barrier between these organisms and the host, bacterial translocation is a common event in the healthy person. However, in critically ill patients, with various underlying diseases, this bacterial translocation may lead to infections and consequently to a further reduction in general health status.

The mechanism of bacterial translocation is widely, and somehow controversially investigated in vitro and in animal models. In human studies, several diseases have been associated with bacterial translocation.

However, methodological shortcomings, insufficient populations and conflicting results leave many open questions. This is also reflected in the various published therapeutic strategies. To overcome this problem more investigations in humans are needed, especially in techniques for detecting bacterial translocation.

Several methods have been used to identify bacterial translocation, including direct and indirect methods. The identification of intestinal bacteria in normally sterile MLN is considered direct evidence of bacterial translocation.

The human response to severe stress, SIRS, is characterized by massive cytokine release, endothelial cell damage tissue edema, increased tissue permeability, activation of the coagulation system platelet aggregation, local tissue hypoxia with shunting and a hypermetabolic state.

Some studies have demonstrated that BT from the gut to MLN is not a rare event, occurring in 4-59% of patients having various clinical conditions, especially when intestinal obstruction or Crohn's disease is present. This phenomena is observed in 15% of a large series of patients undergoing laparotomy. It is also found a significantly increase in postoperative sepsis in patients with evidence of BT (45%) compared with those with negative MLN cultures (19%) O'Boyle et al. Furthermore, the organisms responsible for clinical infections were similar to those isolated in the MLN. Conversely, most of the patients with evidence of BT to MLN had no clinical infectious complications, supporting the hypothesis that it could be a natural event in some situations

and not clinically significant in the presence of a fully functional immune system. In systemic infection, isolation of the bacteria responsible for the disease is sometimes not possible in spite of routine bacterial cultures. The knowledge that inflammatory compounds are responsible for clinical symptoms, and not necessarily the bacteria itself, advanced the understanding of SIRS. When the pathogenic bacteria is isolated the patient is considered to have sepsis, when it cannot be identified the diagnosis is of SIRS and antibiotic treatment is administrated either way. This concept that inflammation causes clinical symptoms was transposed to the translocation theory.

The Criteria of Critically Ill Patient

Recognizing Critical Illness:

Critically ill patients are identified by review of the history, by examination and investigations. Higher risks are associated with extremes of age, with significant co morbidities or with serious presenting conditions. Outcome is often related to and can be predicted by abnormal physiology.

Many studies show patients have abnormal physiology for hours and sometimes days before critical events such as cardiopulmonary arrest. However, measuring and recording of vital signs on general wards are often inadequate **(Gunning and Rowan, 1999**).

A physiologically based system for identifying critical illness should have certain attributes. For early recognition of hospital patients to be effective:

- Abnormal physiological values or biochemistry or other patient factors should enable identification.

- There must be enough time to identify the patients – and to obtain expert assistance.

- Early intervention should be beneficial.

1- Abnormal physiology and adverse outcome

There is an association between abnormal physiology and adverse outcome. Critical care severity scoring systems such as Acute Physiology, Age and Chronic Health Evaluation II (APACHE II) are based on this relationship. Patients who suffer cardiopulmonary arrest or who die in hospital generally have abnormal physiological values recorded in the preceding period, as do patients requiring transfer to the critical care unit. It therefore follows that vital signs can predict many adverse events.

1-1 Acute Physiology and Chronic Health Evaluation Score (APACHE II)

This system became the most widely used of the general outcome prediction systems, and today it is still used in a large number of ICUs. APACHE II was developed based on data registered between 1979 and 1982 in 13 hospitals of the USA **(Spillman and Lubitz, 2000).**

The choice of variables and their weights was selected by a group of experts, using clinical judgment and physiological relationships as documented in the literature. The model uses the most deranged value from the first 24 hours in the ICU. A main reason for ICU admission has to be chosen from a list of 50 operative and non-operative diagnoses, in order to transform the APACHE II score into a probability of death (in the hospital). The APACHE II score varies from 0 to 71 points: up to 60 for physiological variables, up to 6 for age and up to 5 for previous health status.

APACHE III

The APACHE III system was developed in 1988-89 based on a sample of critically ill patients from 40 hospitals in the USA. Patients with an ICU length of stay less than 4 hours, age < 16 years or an admission diagnosis of burn injury, acute myocardial infarction or coronary artery bypass surgery, were excluded from the cohort. The model consists of the Acute Physiology Score (APS), age, and chronic health status. The equation uses the APACHE III score and reference data from the main diagnostic categories, the surgical status, and the location of the patient before ICU admission to estimate the vital status at hospital discharge **(Rubenfeld et al., 1999).**

The APACHE III scores vary between 0 and 299 points, including up to 252 points for the 18 physiological variables, up to 24 points for age, and up to 23 points for the chronic health status. All the physiological variables are evaluated as the most deranged values from the first 24 hours in the ICU. This strategy was chosen by the authors to minimize the amount of missing data and to increase the explanatory power of the model, but eventually there are pitfalls when the model is used to evaluate the performance of the ICU A specific software for the calculation of hospital mortality has thus to be purchased from the developers.

The APACHE III calculator is not available to put in print as it is expensive software, which becomes a major limitation to its clinical utility as well

1-2 Mortality Prediction Model (MPM II)

The MPM II was described by Stanley Lemeshow et al in 1993 **(Kvale and Flaatten, 1997)**. It is based on the same database that was used for the development of the SAPS II, with same exclusion criteria. In the MPM II models, the final result is expressed as a probability of hospital mortality and not a score. The actual version includes models to predict mortality

at hospital discharge based on data from admission (MPM II 0) and after the first 24 hours in the ICU (MPM II 24). Later, the same authors developed additional models based on data from 48 hours (MPM II 48) and 72 hours after admission to the ICU (MPM II 72) **(Mehrez and Gafni, 1999).**

The MPM II 0 model uses 15 variables. All of them are evaluated based on data collected in the first hour before and after ICU admission. The MPM II 24 is based on 13 variables. The physiological variables are based on the most deranged values during the first 24 hours in the ICU. The MPM II 48 and the MPM II 72 use the same variables as MPM II 24, with different weights to compute the probabilities of death and are based on the most deranged values of the preceding 24 hours.

These principles have been incorporated into a number of early-warning scoring (EWS) systems table (1). The systems incorporate different combinations of physiological parameters, a range of approaches to scoring and various trigger thresholds. In the UK these methods are often called track and trigger warning systems. They can be broadly summarized as single-parameter systems, multiple-parameter systems, aggregate weighted scoring systems or combinations **(Subbe et al., 2001).**

Table (1): Identification of Patients with Potential Critical Illness Using the Early Warning Score.

Score	3	2	1	0	1	2	3
Respiratory rate (/min)		<8	8–11	12–20	21–25	26–30	>30
Arterial oxygen saturation (%)	<85	86–89	90–94	>95			
Heart rate (/min)		<40	41–50	51–100	101–110	111–130	>130
Systolic blood pressure (mmHg)	<70	71–80	81–100	101–179	180–199	200–220	>220
Temperature (°C)		<35	35.1–36.5	36.6–37.4	>37.5		
Neurological status			New confusion	Alert	Responds to voice	Responds to pain	Unresponsive

A score of 3 or more suggests potential critical illness and requires immediate assessment **(Brown, 2010).**

Emergency physicians (EPs) diagnose and manage critically ill patients on a daily basis table (2). Published reports from nearly a decade ago indicate that EPs provide approximately 15% of the total critical care a patient receives during their hospital admission. With the persistent problem of hospital overcrowding and emergency department (ED) "boarding," it is likely that EPs are actually providing substantially more critical care than previously reported. For many patients, the EP is, in fact, the first "intensivist" encountered on arrival at the hospital**(Brown, 2010).**.

Fundamental to the care of critically ill patients in the ED is the use of non invasive and/or invasive monitoring devices to detect early cardiovascular compromise, initiate treatment, and monitor response to therapy. With the goal of optimizing tissue perfusion and maintaining adequate oxygen delivery, EPs may use these devices to monitor oxygenation, ventilation, arterial pressure, intravascular volume, cardiac output, and markers of global and regional perfusion. Because time is crucial for detection of hemodynamic compromise, it is important that the EP understand the utility and limitations of these commonly used modalities for monitoring the critically ill, table (2). **(Andrews and Nolan, 2006),**

Table (2): Systematic examination of the critically ill patient.

Site	Check list
Central nervous system	Conscious level and mental state
	Pupils: size, symmetry, response to light
	Fundi
	Lateralized weakness?
	Tendon reflexes and plantar responses
Head and neck	Neck stiffness?
	Jaundice/pallor?
	Jugular venous pressure
	Central venous cannula?
	Mouth, teeth and sinuses
	Lymphadenopathy?
Chest	Focal lung crackles/bronchial breathing?
	Pleural/pericardial rub?
	Heart murmur?
	Prosthetic heart valve?
	Pacemaker/ICD?
Abdomen and pelvis	Vomiting/diarrhea?
	Distension?
	Ascites?
	Tenderness/guarding?
	Bladder catheter?
	Perineal/perianal absces?
Limbs	Acute arthritis?
	Prosthetic joint?
	Abscess?

ICD, implantable cardioverter-fibrillator **(Gunning and Rowan, 1999).**

Table (3): Nine key monitoring in suspected critical illness.

Observation	Signs of critical illness	Action
1. Airway	Evidence of upper airway obstruction	Airway Management and Upper Airway Obstructiion for management of the airway
2. Respiratory rate	Respiratory rate <8 or >30/min	Give oxygen (initially 60–100%)
		Check arterial oxygen saturation and blood gases
		management of respiratory failure
3. Arterial oxygen saturation	Arterial oxygen saturation <90%	Give oxygen (initially 60–100% if there are other signs of critical illness)
		Check arterial blood gases
4. Heart rate	Heart rate <40 or >130 bpm	Give oxygen 60–100%
		Connect an ECG monitor and obtain
		IV access
		management of cardiac arrhythmia
5. Blood pressure	Systolic BP <90 mmHg, or fall in systolic BP by more than 40 mmHg with signs of impaired perfusion	Give oxygen 60–100%
		Connect an ECG monitor and obtain
		IV access
		management of hypotension/impaired perfusion
6. Perfusion	Signs of reduced organ perfusion: cool/mottled	Give oxygen 60–100%
		Connect an ECG monitor

	skin with capillary refill time >2 s; agitation/reduced conscious level; oliguria (urine output <30 ml/h)	and obtain IV access
		management of hypotension/impaired perfusion
7. Conscious level	Reduced conscious level (unresponsive to voice)	Stabilize airway, breathing and circulation
		Endotracheal intubation if GCS 8 or less
		Exclude/correct hypoglycemia
		Give naloxone if opioid poisoning is possible (respiratory rate <12/min, pinpoint pupils)
		management of the unconscious patient
8. Temperature	Core temperature <36 or >38°C, with hypotension, hypoxemia, oliguria or confusional state	management of sepsis
9. Blood glucose	Blood glucose <3.5 mmol/l, with signs of hypoglycemia (sweating, tachycardia, abnormal behavior, reduced conscious level or fits)	Give 50 ml of 50% glucose IV via a large vein (or 500 ml of 5% glucose
		IV over 15–30 min) or glucagon 1 mg IV/IM/SC
		Recheck blood glucose after 5 min and again after 30 min.

GCS, Glasgow Coma Scale score **(Arts et al., 2005).**

1-3 Investigation of the critically ill patient include:

1-3-1 Immediate

Arterial blood gases, ECG, blood glucose, sodium, potassium and creatinine and full blood count.

1-3-2 Urgent

Chest x ray, cranial CT if reduced conscious level or focal signs, coagulation screen if low platlet count, suspected coagulation disorder, jaundice or purpura, Biochemical profile, amylase if abdominal pain or tenderness, c-reactive protein, blood cultures if suspected sepsis, urine stick test and toxicology screen (serum 10 ml & urine 50 ml) if suspected poisoning **(Arts et al., 2005).**

2- Severity of illness and likely outcome from critical illness

At present scoring systems are not sufficiently accurate to make outcome predictions for individual patients. Clinical assessment of severity of illness is an essential component of medical practice. It influences the need and speed for

supportive and specific therapy. Initial acuty may also indicate likely prognosis when other factors such as comorbidity and organisational aspects of critical care delivery are considered. It is intuitive to consider whether patterns and severity of physiological disturbance can predict patient outcome from an episode of critical illness. Perhaps the earliest reference to grading illness was an Egyptian papyrus which classified head injury by severity.

More recently it has been the constellation of physiological disturbances for specific conditions which popularised an approach which linked physiological disturbance to outcome in the critically ill.

2-1 Purposes of scoring systems

There are five major purposes of severity of illness scoring systems. First, scoring systems have been used in randomized controlled trials (RCT) and other clinical investigations **(Mehrez and Gafni, 1999).** The second purpose of severity-of-illness scoring systems is to quantify severity of illness for hospital and health care system administrative decisions such as resource allocation. The third purpose of these scoring systems is to assess ICU performance and compare the