EVALUATION OF ENERGY EXPENDITURE IN THE ACUTE CARE OF SEVERE HEAD INJURY PATIENTS : INDIRECT CALORIMETER VERSUS HARRIS BENEDICT FORMULA

By

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Dedication

To my dearest wife :

Siti Noor Elisa Binti Selamat

And

My Children :

Nur Husna Binti Saiful Razman

Nur Syuhada Binti Saiful Razman

Nur Aula Binti Saiful Razman

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Contents

ABSTRACT

Introduction

Management of comatose patients especially severely head-injured patients is very crucial due to the fact that secondary insult such as brain edema, hypo/hypertension, anemia and hypoxemia may develop during this period of time. Such patients are managed in intensive care units and usually need ventilatory support and further neurosurgical interventions.

Apart from the medical and surgical aspects of management, nutritional support plays an important role in patient's recovery. There are few literatures on head-injured patients' energy requirement or expenditure in the acute setting (Clifton et al., 1986, Hadley et al., 1986, Michele et al., 2003 and Foley et al., 2008). Adequate energy supply for head-injured patients is an important part of intensive care management (ICU) in order to achieve optimal care and to avoid complications of hypo/hyper caloric feeding. It is believed that patients with different grades of head injury have different energy requirements and thus nutritional support both in the acute and chronic setting. By mean of an indirect calorimeter, we managed to measure the energy expenditure of severe head injury patients in an acute setting in the Neuro Intensive Care Unit, Hospital Universiti Sains Malaysia and compared the results to the predictive values obtained via Harris-Benedict Formula. We also managed to compare the difference of energy expenditure in the different grades of severity of head injury based on Marshall's classification system and compared the energy expenditure values amongst major operation, minor operation and conservative management groups. In this study, we also managed to determine the sensitivity and specificity of Harris-Benedict Formula as compared to the indirect calorimeter. Special interest was given to the analysis of twenty-four hour blood glucose levels and its association with severe head injury. This study is another step forward for better understanding in the management of critically ill patients in a tertiary neurosurgical center in Malaysia.

Patient and Method

This was a prospective observational study of whom severe head injury patients admitted to Neuro ICU in Hospital Universiti Sains Malaysia were selected for the measurement of energy expenditure by indirect calorimetry in an acute setting. A total of 31 severely head injured patients in Kelantan, Malaysia were selected for this study from January 2009 to March 2010 after fulfilling the inclusion criteria. The indirect calorimeter (Deltatrac II) was connected to these patient's ventilator and the measurements of energy expenditure were measured for 24 hours. The values of the measured energy expenditure (MEE) of each patient were compared to the predicted values from the Harris Benedict equation.

A total of 31 patients who were involved in this observational study were divided into four groups of severity determined by Marshall's CT gradings (Marshall's grade 1 – 4). The energy expenditure of these patients in each groups were compared and analysed to see whether or not there were differences between these groups.

Of all the 31 patients involved, the energy expenditure of operated (major or minor operation) and non-operated (conservative) patients were documented and analysis were made between these groups using specific statistical tests.

Results

The lowest energy expenditure measured in this study was 740 kcal/day and the highest was 2060 kcal/day with mean energy expenditure in this study population was 1498 ± 297 kcal/day. The comparable predicted value by Harris Benedict equation was 755 kcal/day (lowest) and 2170 kcal/day (highest) with the mean of 1543 ± 268 kcal/day. By using Pearson correlation test, there was strong positive correlation of measured energy expenditure values, MEE (by indirect calorimeter) and the predicted energy expenditure values, BEE (by Harris Benedict equation), Pearson correlation value was 0.789 (p-value <0.001).

The mean energy expenditure in each groups of Marshall's grading 1,2,3 and 4 were 1440 ± 42 kcal/day, 1484 ± 349 kcal/day, 1358 ± 308 kcal/day and 1595 ± 277 kcal/day respectively. By using Kruskal-Wallis test, there was no significant difference of energy expenditure between these groups in the acute setting (p value = 0.343).
The mean energy expenditure in major operation group was 1535 ± 265 kcal/day whereas the mean energy expenditure in minor and conservative groups were 1113 ± 365 kcal/day and 1565 ± 305 kcal/day respectively. By using the one-way ANOVA test, there was no significant difference of energy expenditure between the major, minor or conservative groups in this study in the acute setting (p value = 0.055).

The lowest blood glucose level was 3.6 mmol/L and the highest was 9.2 mmol/L in this study. The mean blood glucose level was 6.4 ± 1.4 mmo/L. Pearson correlation showed no association of blood glucose levels to MEE (Pearson correlation equals to 0.013 with p-value = 0.943). The Kruskal-Wallis test and one-way ANOVA test showed that there were no significant difference of blood glucose levels between Marshall's gradings (p-value = 0.432) and between major operation, minor operation and conservative groups (0.830) respectively.

Conclusion

Predictive formula (Harris Benedict equation) can be used to determine the energy expenditure thus the energy requirement of severe head injury patients which are fully sedated and ventilated in order to achieve energy equilibrium.

This preliminary study showed that there was no difference in energy expenditure amongst groups in the severely head injured patients which were fully sedated and ventilated regardless of their CT brain findings and of whether they were operated or conservatively managed in the first 24 hours of management.

ABSTRAK

Pendahuluan

Pengendalian pesakit yang koma terutamanya yang mengalami kecederaan kepala yang teruk adalah sangat kritikal disebabkan oleh beberapa faktor dan masalah yang terjadi susulan kecederaan utama. Ini termasuklah kejadian seperti kebengkakan otak, ketidakstabilan tekanan darah, kekurangan sel darah merah dan kekurangan oksigen dalam darah yang biasa terjadi semasa pesakit dalam keadaan koma akibat kecederaan kepala yang teruk. Pesakit-pesakit sebegini biasanya memerlukan sokongan alat bantuan pernafasan di unit rawatan rapi dan rawatan atau pembedahan neuro.

Selain daripada rawatan pembedahan, sokongan nutrisi juga memainkan peranan penting dalam proses pemulihan pesakit ini. Terdapat hanya beberapa kajian terdahulu yang menumpukan kepada penyukatan pembebasan tenaga dalam pesakit-pesakit yang mengalami kecederaan kepala yang teruk dalam tempoh akut (Clifton et al.,1986, Hadley et al.,1986, Michele et al., 2003 and Foley et al., 2008). Hingga kini, kekurangan pengetahuan dan pengalaman dalam keperluan tenaga pasakit yang mengalami kecederaan kepala yang teruk, menyebabkan ketidaktepatan dalam pemberian bekalan nutrisi kepada golongan ini. Bekalan tenaga yang cukup amat penting dalam memastikan pencapaian rawatan yang memuaskan dan mengelakkan komplikasi kekurangan atau lebihan bekalan nutrisi. Adalah dipercayai setiap pesakit ini berbeza dari segi keperluan tenaga mereka berdasarkan tahap kecederaan kepala mereka dan dengan itu berbeza juga sokongan nutrisi yang diberikan. Melalui mesin "indirect calorimeter", kita dapat mengukur tenaga yang dibebaskan oleh pesakit ini dalam tempoh akut dan dengan itu gantian jumlah tenaga yang sama dapat diberikan melalui bekalan nutrisi yang tersedia. Melalui kajian ini juga, kami dapat membandingkan jumlah tenaga yang dibebaskan oleh pesakit-pesakit daripada gred kecederaan kepala yang berlainan berdasarkan klasifikasi Marshall dan membandingkan jumlah tenaga di antara kumpulan pembedahan besar, kecil

dan kumpulan yang tiada pembedahan. Menerusi kajian ini, kami juga dapat menentukan sensitiviti dan spesifisiti Formula Harris-Benedict berbanding “indirect calorimeter”. Tumpuan istimewa telah diberikan kepada analisa kandungan gula dalam darah semasa dua puluh empat jam selepas kecederaan dan hubungkaitnya dengan kecederaan kepala yang teruk. Kajian ini adalah merupakan satu langkah ke hadapan dalam usaha memahami dengan lebih baik perawatan pesakit-pesakit kritikal di pusat rujukan saraf di Malaysia.

Pesakit dan Metodologi

Kajian ini adalah secara pemerhatian prospektif di mana pesakit yang dimasukkan ke unit rawatan rapi neurosurgeri Hospital Universiti Sains Malaysia dipilih untuk kajian penghasilan/pembebasan tenaga semasa rawatan dengan menggunakan mesin “indirect calorimeter” dalam tempoh akut. Sejumlah 31 orang pesakit terpilih untuk kajian di negeri Kelantan, Malaysia, ini yang berlangsung sepanjang bulan Januari 2009 hingga Mac 2010 setelah memenuhi kriteria tertentu. Mesin “indirect calorimeter (Deltatrac II)” disambungkan kepada mesin bantuan pernafasan pesakit dan pengukuran pengeluaran tenaga dilakukan sepanjang 24 jam. Nilai tenaga yang diperoleh oleh mesin penyukat tenaga itu dibandingkan dengan nilai tenaga yang dijangkakan melalui formula Harris Benedict.

Daripada keseluruhan 31 pesakit yang terlibat dalam kajian ini, mereka dibahagikan pula kepada kumpulan berdasarkan keterukan gambar skan CT kepala mereka mengikut gred Marshall dari gred 1 hingga gred 4. Jumlah tenaga yang dibebaskan daripada pesakit dari setiap kumpulan dicatit dan dibandingkan untuk mengetahui sama ada terdapat perbezaan dalam pembebasan tenaga di antara mereka.

Jumlah tenaga yang dihasilkan daripada pesakit dalam kumpulan pembedahan major atau minor dan daripada pesakit dalam kumpulan yang tidak dibedah juga dicatit dan dibandingkan menggunakan pengiraan statistik.

Keputusan

Nilai tenaga yang paling kurang yang dicatit dalam kajian ini ialah sebanyak 740 kcal/sehari dan yang paling tinggi ialah sebanyak 2060 kcal/sehari dengan purata nilai tenaga sebanyak 1498 ± 297 kcal/sehari. Nilai yang setara yang diperoleh melalui kaedah jangkaan(Formula Harris Benedict) pula ialah 755 kcal/sehari (terendah) dan 2170 kcal/sehari (tertinggi) dengan purata nilai sebanyak 1543 ± 268 kcal/sehari. Melalui ujian statistik Pearson correlation, kedapatan kesesuaian/kesetaraan yang kuat di antara nilai tenaga yang disukat oleh mesin penyukat dengan nilai yang diperoleh melalui formula jangkaan Harris Benedict, nilai Pearson correlation ialah 0.789 (p-value < 0.001).

Jumlah purata tenaga dalam kumpulan-kumpulan gred Marshall 1,2,3 dan 4 pula ialah 1440 ± 42 kcal/sehari, 1484 ± 349 kcal/sehari, 1358 ± 308 kcal/sehari, 1595 ± 277 kcal.sehari masing-masing. Melalui kaedah statistik Kruskal-Wallis, didapati tiada perbezaan ketara jumlah tenaga yang dihasilkan di antara kumpulan-kumpulan gred Marshall itu dalam tempoh akut (nilai p = 0.343).

Jumlah purata tenaga yang dihasilkan dalam kumpulan pembedahan major ialah 1535 ± 265 kcal sehari manakala tenaga yang dihasilkan dalam kumpulan pembedahan minor dan kumpulan konservatif pula ialah 1113 ± 365 kcal sehari dan 1565 ± 305 kcal sehari masing-masing. Melalui kaedah statistik "one-way ANOVA", didapati tiada perbezaan ketara di antara jumlah tenaga yang dibebaskan oleh pesakit-pesakit dalam kumpulan pembedahan major, minor mahupun konservatif dalam tempoh akut tersebut (nilai p = 0.055).

Kandungan gula dalam darah paling rendah ialah 3.6 mmol/L and yang paling tinggi ialah 9.2 mmol/L. Purata kandungan gula dalam darah pesakit-pesakit tersebut ialah 6.4 ± 1.4 mmo/L.

Tiada perhubungan didapati di antara kandungan gula dalam darah dengan tenaga yang dibebaskan (Pearson correlation sama dengan 0.013, p-value = 0.943). Melalui kaedah Kruskal-Wallis dan one-way ANOVA, didapati tiada perbezaan ketara antara kandungan gula dalam darah dengan gred Marshall's (p-value = 0.432) dan tiada perbezaan ketara juga di antara kandungan gula dalam darah dengan kumpulan pembedahan major, pembedahan minor and kumpulan konservatif (p-value = 0.830) masing-masing.

Kesimpulan

Formula jangkaan Harris Benedict boleh digunakan untuk menentukan jumlah tenaga yang dibebaskan oleh pesakit-pesakit yang koma disebabkan kecederaan kepala yang teruk di mana pesakit-pesakit menerima bantuan mesin pernafasan dan direhatkan atau disedasikan sepenuhnya.

Dengan mengetahui jumlah tenaga yang dibebaskan oleh pesakit, maka nutrisi dengan jumlah kandungan tenaga yang sama dapat diberikan kepada pesakit untuk mencapai keseimbangan tenaga.

Dalam kajian awal ini, tiada perbezaan dalam pembebasan tenaga di kalangan pesakit-pesakit koma yang mengalami kecederaan kepala yang teruk di mana mereka direhatkan/disedasikan sepenuhnya dan dibawah bantuan mesin pernafasan, tidak kira apa jua gambaran skan CT mereka dan tidak kira sama ada mereka dibedah atau tidak dibedah.

1. INTRODUCTION

Management of comatose patients especially severely head-injured patients is very crucial due to the fact that secondary insult such as brain edema, hypo/hypertension, anemia and hypoxemia may develop during this period of time. Such patients are managed in intensive care units and usually need ventilatory support and further neurosurgical interventions.

Apart from the medical and surgical aspects of management, nutritional support plays an important role in patient's recovery. There are few literatures on head-injured patients' energy requirement or expenditure (eg. Clifton et al., 1986, Hadley et al., 1986, Brader et al., 1998, Michele et al., 2003 and Foley et al., 2008). Adequate energy supply for head-injured patients is an important part of intensive care management (ICU) in order to achieve optimal care and to avoid complications of hypo/hyper caloric feeding. It is believed that patients with different grades of head injury have different energy requirements and thus nutritional support.

Many studies have shown that energy expenditure are elevated in post traumatic patients especially severe head injury patients ranging from 20% - 45% above estimated normal resting values (Robertson CS et al.,1984, Young B et al., 1985, Clifton et al., 1986, Fried RC et al., 1989, Borzotta AP et al., 1994). These studies done helping us to discover the actual energy requirement for the head injured patients in general.

Most of previous studies compared the energy expenditure of the polytraumatic to non traumatic patients, the diseased patients to healthy patients and the obese to non-obese patients (eg. Frankenfield et al., 1994, AM Schols et al., 1991,and AM Prentice et al., 1986).

None of these studies compared the energy expenditure of different grades of severe head injury or between surgically operated and non-operated groups of head injury.

It is believed that patients with different grades of head injury have different energy requirement and thus the specific nutritional support targetted to the different grades of severe head injury. We were looking forward to measure the energy expenditure of different grades of severity of head injury patients, whether they were subjected for major or minor operations or managed conservatively. .

2. LITERATURE REVIEW

2.1 Epidemiolgy and classification of severe head injury

Head injury is the leading cause of mortality and morbidity after trauma. In some series almost half of all trauma deaths result from head injury. Most head injuries requiring hospitalization occur in young person between the ages of 15 and 24 (Cooper, 1982, Jennett, 1996). Thus, the patients at greatest risk of sustaining a fatal or disabling head injury are individuals just beginning their productive years. The direct and indirect financial losses to society are staggering (Robert et al., 1998).

Malaysia is one of the countries that have seen a decline in infectious disease with an increase in mortality and disability from injuries and non-communicable disease over the past decade (Ministry of Health Malaysia, 1997). Injuries are a leading cause of mortality in men under the age of 40, and the majority are attributed by road traffic injuries (RTIs), particularly motorcycle crashes (Ministry of Health Malaysia, 1997).

Head injury is classified as mild, moderate or severe, depending on the Glasgow Coma Score (GCS) on admission (Rimel et al.,1981, Rimel et al., 1982, Jennet et al., 1974). There is general consensus that mild head injury patients are those that have a GCS of 13-15 (Rimel et al., 1982). Moderate head injury have an admission GCS of 9-12, and severe head injury an admission GCS of 8 or less after resuscitation (Jennet et al., 1974).

Based on CT brain finding of severe head injury patients, they can be further classified into grade I, II, III or IV depending on Marshall's CT classification, Table 1 (Marshall et al., 1991).

Table 1 : Marshall's CT Brain Classification/Grading

Diffuse Injury Grade	**CT appearance**
I	Normal CT scan
II	Cisterns present. Shift < 5mm, no high or mixed density lesion > 25cc
III	Cisterns compressed/absent. Shift < 5mm, no high or mixed density lesion > 25cc
IV	Shift > 5mm, no high or mixed density lesion > 25cc
Evacuated mass lesion	Any lesion surgically evacuated
Non-evacuated lesion	High or mixed density lesion > 25cc, not surgically evacuated

CT : computed tomography

2.2 Metabolic response to severe head injury

The metabolic response to severe head injury is characterised by hypermetabolism and typically hypercatabolism (Wilson and Tyburski, 1998 and Yanagawa et al., 2002). These responses reflects on the energy expenditure of the injured patients.

The resting energy expenditure (REE) in patients with severe head injury tends to be increased by an average of about 40%. Clifton et al found the REE of their non-sedated head injury patients to be elevated by 38% +/- 37% compared to the normal basal energy expenditure (BEE) predicted by the Harris-Benedict equation (Rosendahl et al.,1937, Clifton et al., 1986) :

$$\text{BEE (kcal/day)} = 66.47 + 13.75 \times W + 5.O \times H - 6.76 \times A \text{ for males ,}$$

$$65.51 + 9.56 \times W + 1.85 \times H - 4.68 \times A \text{ for females and}$$

$$22.1 + 31.05 \times W + 1.16 \times H \text{ for infants ;}$$

where W = weight in kilograms, H = height in centimeters, and A = age in years.

Other studies have also reported increases in BEE ranging from 20% - 200% above predicted values (Long et al., 1979 and Haider et al., 1975).

Many variables may affect energy expenditure in these severe head injury patients. These include the severity of injury, medications, nutritional support, presence of sepsis and body temperature (Foley et al., 2008, Michele et al., 2003 and Bruder et al.,1998). It is generally accepted that this group is in hypermetabolic and hypercatabolic in the initial few weeks after injury. However in many patients, the use of sedatives, analgesics and neuromuscular blockade can significantly lower energy expenditure (Bruder et al., 1998 and Michele et al., 2003).

Robert and James, (1998) suggested the use of indirect calorimetry to guide nutritional therapy in these patients since they found that predictive formula in patients with severe head injury are extremely unreliable.

Protein catabolism is a well known feature in severe traumatic patients and an essential process in body's defence mechanism. This leads to availability of esential amino acids for the production of immune cells, plasma protein and enzymes in order to repair damaged tissues.The drive behind the catabolic response involves the activation of the pituitary gland and the sympathetic nervous system after trauma within the first 24 hours. This leads to an increased catecholamines in the body (Clifton et al., 1981) as well as dopamine, glucagon and cortisol (Feldman et al.,1993).

From a nutritional perspective, the so-called " counter-regulatory" hormones help create an environment of substrate availability at the expense of body stores. The result is increased protein catabolism, primarily from skeletal muscle, with the released amino acids providing substrate for new protein synthesis of acute phase reactants or carbon skeletons for gluconeogenesis especially after 24 hours post trauma. Alanine and glutamine play a predominant role, accounting for 50-60% of the amino acids released. The counter-regulatory hormones also cause both accelerated gluconeogenesis in the liver and accelerated uptake of glucose by peripheral tissues. Hyperglycaemia will often persist despite elevated insulin levels, a metabolic picture often referred to as the insulin resistence of injury (Souba and Wilmore, 1994)

2.3 Body temperature and sepsis

Body temperature and sepsis have significant effect in energy expenditure. Fever developes in response to the actions of either endogenously produced pyrogens, for example, Interleukin 1 (IL-1) and Tumor Necrosis Factor (TNF) or exogenous pyrogens such as toxins and microbes. Immune cells of the body producing IL-1 upon stimulation of bacteria, viruses or tissue injury. The IL-1 then effects the hypothalamus via prostaglandin and catecholamine production to induce fever (Dinarello, 1988). It is well established that fever increases metabolic rate. Dubois in 1921 concluded that metabolic heat production increases approximately 13% per degree centigrade in humans. Other reserchers have reported higher increases in energy expenditure due to fever. Buskirk et al., (1964) found an increment of 27-50% per degree centigrade in humans injected with endotoxin, with substantial variability among subjects. This particular information is relevant to the studies that last longer than a few days in ICU patients in which the fever developed after this period usually related to infection. The increase of metabolism from a fever of 38°C could be misinterpreted as an effect of hypermetabolism from the trauma causing a rise of 30% in energy expenditure. Many studies make no mention or attempt to correlate body temperature to the measured energy expenditure (Young et al.,1985, Fruin et al., 1986 and Jeevandum et al.,1992), while others exclude patients with fever in their studies (Raurich and Ibanez, 1994). In our study however, we excluded patients with fever (temperature 38 degree Celcius and above).

A later study done by Bruder et al., (1998) showed correlation of energy expenditure with the body temperature. In their study, there were 24 patients recruited and these patients were divided into 4 groups according to the anesthetic agents used for sedation: fentanyl and midazolam (group FM); fentanyl, midazolam, and curarization (group C); thiopental (group T); and no sedation (group NS). They found that in sedated patients, body temperature was the main determinant of energy expenditure; the anesthetic agent used had little influence on the level of energy expenditure and sepsis increased energy expenditure independently of fever, probably through hormonal changes.

2.4 Thermogenic effect of nutrition

Energy expenditure is increased by 5-10% for 3 - 6 hours after a meal, which varies depending on the quantity and composition of the meal (Heymsfield et al.,1987). This phenomenon is due to digestion, absorption, metabolism and storage of the nutrients (Horton, 1983). The effect of continuous delivery of nutrition on metabolic rate depends on the amount of energy delivered. Study by Heymsfield et al., (1987) showed that continuous enteral feedings provided at maintenance levels in healthy subjects (ie for energy balance), caused no increase in resting metabolic rate when compared to fasting metabolic rate. However, once the quantity of enteral feeding was increased to provide 2.6 – 3.0 times the fasting resting energy expenditure, measured energy expenditure (MEE) increased by 11.7%. Thus, overfeeding can cause the metabolic rate higher, not only due to the cost of metabolising the nutrients but also due to the energetic cost of lipogenesis (Michele McCall 1997). For example, the cost of storing glucose directly into glycogen versus into fat showed an increment of 5% and 19% respectively (Simonson and DeFronzo, 1990).

In our study however, we kept the patients fasted throughout the energy expenditure measurement. Thus, in such a controlled condition, the thermogenic effect of nutrition was excluded.

2.5 Effect of body and limbs movement

A study by Fruin et al., (1986) showed muscular activities causes significant increase in mean energy expenditure (MEE). His group measured energy expenditure on day 1, 4, 9 and 15 in 15 head injured patients with admission GCS of seven or less. The amount of movement was specifically categorised into four levels : no movement or muscle tone, normal muscle tone with rare spontaneous activity, random normal motor activity, and abnormal motor activity (ie. posturing, muscle rigidity or tremors). The MEE increased steadily from 108% of predicted basal metabolic rate (PBMR) to 114%, 142%, and 192% respectively. The rise above PBMR could have been mainly due to movement rather than hypermetabolism from the head injury.

2.6 Nutritional support for severe head injury patients

Traumatic brain injury leads to an elevated energy expenditure, or hypermetabolic response in many patients. The magnitude of this response is directly proportional to the degree of injury. Along with hypercatabolism of body protein, significant wasting of skeletal muscles occur in patients with severe head injury.

Many researchers believe that appropriate nutritional support could decrease the amount of muscle catabolised and improve the outcome of these patients. The goal of nutritional therapy is the achievement of weight maintenance and zero or positive nitrogen balance. It is believed that protein could be spared if adequate energy and protein could be delivered, as was seen in models of unstressed starvation (Michele McCall, 1997).

Interestingly, positive nitrogen balance was very difficult if not impossible to achieve in many patients and often led to complications such as hyperglycaemia and gastrointestinal intolerance. This was the finding of eight trials completed in 1980's that prospectively measured nitrogen balance, and/or energy requirements in head injured patients (Clifton et al., 1984, Clifton et al., 1985, Robertson et al.,1985, Young et al., 1985, Twyman et al., 1985, Bivins et al., 1986, Hadley et al., 1986 and Grahm et al.,1989).

Clifton et al. in 1984 studied fourteen severely head injured patients for 28 days post injury. The average energy expenditure (MEE) measured by indirect calorimetry was 138%±37% of predicted calculated values for basal metabolic rate (PBMR). Predicted basal metabolic rate was calculated using Harris Benedict Equation (1919). Only half of the patients were able to achieve nitrogen balance, which required an intake of 161 – 240% of MEE. Although this first study did not recommend a specific energy goal, subsequent work by Clifton led to the recommendation of 50 kcal/kg/day (Clifton et al., 1985).

Other studies by Young and Twyman (Young et al.,1985, Twyman et al.,1985, Young et al., 1987) in head injury patients also used high energy intakes of 75% above the PBMR in an attempt to achieve positive nitrogen balance in the acute phase post injury. They were unsuccessful unless a simultaneous protein intake of 2.2g/kg/day could be delivered. Young et al., (1985), measured energy expenditure and nitrogen balance in 16 severely head injured patients for 18 days post admission. The mean MEE was 140% of PBMR (35 kcal/kg/day), and nitrogen balance was not achieved without energy intakes of 20-60% above the MEE and protein intakes of 1.9 –2.6 g/kg/day. The group recommended goal intakes of 40 - 50 non-protein kcal/kg/day and 2.0 – 2.5 g/kg/day of protein for severely head injured patients.