# Changes in weight after traumatic brain injury in adult patients: A longitudinal study.

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**Key words**: traumatic brain injury, weight change, behavioral dysexecutive syndrome, eating disorders

**Abbreviations**: TBI: traumatic brain injury. BMI: body mass index. WHO: World Health Organization.

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**Abstract**

**Background & Aims**: Although changes in weight have been reported after traumatic brain injury (TBI), their frequency and underlying factors are little known. Our aim was to determine the prevalence of weight changes and the associated factors during the recovery phase after TBI.

**Methods**: Longitudinal follow-up of adults with TBI. Multivariate analysis was carried out on weight change, demographic data, dysexecutive syndrome, eating behavior, physical activity, therapeutic classes and metabolic complications.

**Results**: 107 patients (81 males/26 females), age 36±13 yrs, baseline BMI 23.3±3.9, followed for 38 (8–66) months, were included. In intensive care, patients lost a mean 11±6 kg. End of follow-up, mean BMI was not different to pre-TBI BMI, but patients could be categorized in 3 groups: stable (30%), loss (28%, −8±7 kg) and gain (42%, +9±6 kg). Sex, age, severity of TBI, intensive care weight loss, physical activity, therapeutic classes and the occurrence of metabolic syndrome did not differ between the groups. Factors related to weight gain were hyperphagia, OR 4.5 (IC95%, 1.6–12.1) and presence of a dysexecutive syndrome, OR 2.5 (IC95%, 1.03-6.3). Factors related to weight loss were hypophagia, OR 4.1 (IC95%, 1.5–10.9) and higher pre-TBI BMI, OR 4.9 (IC95%, 1.7–14.0).

**Conclusions**: Over a median period of 38 months, 42% of TBI patients gained and 28% lost weight. Factors associated with these changes were the presence of a behavioral dysexecutive syndrome for weight gain, oral food intake and initial BMI, which were inversely associated with weight at end of follow-up. These findings highlight the importance of evaluating the time course of weight changes and providing specific nutritional care.

## Introduction

Obese passengers are more likely to suffer a more severe head injury after frontal motor-vehicle crashes[1]. After traumatic brain injury (TBI), changes in weight and (or) food behavior have been reported in short studies [2,3] and various clinical case-reports [4,5]. Some reports have described hyperphagia and reduction of satiety [6]. Anorexia following TBI has also been reported [7]. A recent longitudinal study in 39 children with TBI showed that 15% were overweight 1 year after the TBI [8]. However, the frequency of weight changes and the underlying factors are currently unknown in adult patients. In a study of 20 TBI patients, the presence of other persons during meals, or the social factor, was a significant predictor of meal size for healthy control subjects, but not for brain-injured patients [2] indicating probable central involvement. In addition, a low prevalence (5.4%) of hypopituitarism, including GH deficiency and hypogonadism, which can contribute to obesity, was reported in a study of 112 adult TBI patients [9]. The same trend was observed in 39 children, with a prevalence of 2.5%[8]. A higher prevalence -15%- was found in a cross-sectional study of 104 adult patients 13 months after TBI [10]. Modification of food behavior – and thus of weight – can be expected to occur after TBI, due to the presence of a dysexecutive syndrome and because the hypothalamus is the main brain center involved in food intake in both animal experimental models and humans [11]. Cognition and behavior can also be impaired in moderately severe TBI, and is related to a "post-concussion syndrome" [12].

Changes in nutritional intake after TBI appear to occur in two phases: a constant, prolonged and significant hypermetabolism during the stay in neurosurgical units and intensive care [13,14], with a risk of severe undernutrition despite nutritional support, followed by a recovery phase which may follow different patterns. To determine the prevalence of changes in weight after TBI in the recovery phase, and the associated factors, we performed a

longitudinal study in a cohort of adult TBI patients followed in a specialized hospital for neurocognitive rehabilitation.

## Patients and methods

*Patients*

This was a single centre longitudinal study of a cohort of adult TBI patients. All the patients were initially cared for in different intensive care units and were then admitted to our physical medicine and rehabilitation (PMR) center between 2004 and 2009. The study protocol was approved by the local Ethics committee. Informed consent was obtained from the patients or their relations in compliance with the French regulations for observational clinical research. Inclusion criteria were: age 18–70 years at the time of the TBI, isolated TBI assessed with the cerebral Marshall CT-scan classification[15] and the Glasgow coma scale (GCS) [mild (score 13–15), moderate (9–12), or severe (<9)] with no associated spinal cord injury or polytraumatic lesions of the viscera which required surgery. In addition, a follow-up of at least 6 months after the intensive care period and from the beginning of rehabilitation was mandatory.

We excluded patients below the age of 18 or above the age of 70, pregnant women, patients with paraplegia, tetraplegia and those in a persistent vegetative state, patients with no indications for rehabilitation and patients with previous surgery to the digestive system, in order to eliminate possible interference with the regulation of food intake.

Analyses of hypothalamic and pituitary hormones were carried out in patients for whom there was a clinical or biological suspicion of deficiency, such as polyuria or hypernatremia.

Enteral nutrition adapted to their needs in terms of energy and protein (1500 to 1800 kcal/d, 56 to 67 g of proteins) was given to each patient in intensive care units by a nasogastric tube or gastrostomy and was continued during the transfer to PMR. Reduction and weaning from enteral nutrition was carried out under the supervision of the dietician during the stay in PMR (4 months in average) when the patient regained the ability to swallow without difficulty. A mixed feeding program was put into place, with a nocturnal enteral intake until oral intake

100 became sufficient, with an energy intake goal of 1600 to 1800 kcal/d for patients who were
101 overweight before the TBI and 1800 to 2000 kcal/d for those who were not.
102
103 *Data collection*
104 Weight was noted at different time points: before the TBI (W1 i.e. usual weight recorded in
105 previous medical files or noted during interviews with the patients or their relatives), at
106 admission to, (W2) and discharge from (W3) the PMR center, and at the end (W4) of the
107 follow-up period. BMI (body mass index) was calculated at each of these time points. The
108 following potential explanatory factors were noted: age, sex, initial GCS score, duration of
109 coma and stay in intensive care, level of physical activity according to the WHO (World
110 health organization) criteria[16] and therapeutic classes of drugs taken by the patients.
111 Quantitative oral food intake was noted according to three categories which were relative to
112 pre-TBI levels: lower, i.e. hypophagia (<1500 kcal/d), similar, and higher, i.e. hyperphagia
113 (>2500 kcal/d). This was based on a dietary inquiry, verbal or visual analogue scales and
114 eating behavior (number of meals per day, eating between meals, taste preferences (sweet or
115 savory), binge eating and nocturnal eating) during the stay in PMR and following return
116 home. No patients took topiramate [17] as an antiepileptic drug or were treated for a binge
117 eating disorder. Oral food intake and eating behavior were determined during dietary inquiries
118 with the patients and their families, at least twice in a 2-month period during a medical
119 consultation or a phone call by a dietician and a physician. In addition, alcohol and tobacco
120 abuse, presence or absence of addictions since TBI and before TBI, were recorded. The
121 presence of a behavioral dysexecutive syndrome was scored dichotomously (yes/no) during
122 the stay in PMR by an experienced neuropsychologist, based on the patient's performance on
123 a standardized test [18]. Metabolic complications which were present before the TBI and any

occurring during the follow-up after TBI were recorded: arterial hypertension, diabetes mellitus or glucose intolerance and dyslipidemia.

*Statistics*

Data from all the patients included were used in the initial analysis. For this analysis, three classes of BMI were used: below 20 (underweight), 20 to 25 (normal), and above 25 (overweight). Data were evaluated at four time points: W1: weight prior to TBI, W2: admission to PMR, W3: discharge from PMR and W4: end of the follow-up period. In a posthoc analysis, patients were categorized in three groups according to the amount of weight change at the end of the follow-up compared with pre-TBI weight: group 1 (weight loss), group 2 (weight stabilization: ±3%) or group 3 (weight gain). Student *t* tests and ANOVAs were used for quantitative data comparisons, and chi-squared tests for qualitative data. To study independent associated factors in relation to weight change, a logistic regression was used. SPSS software version 11.5 was used for the statistical analysis. Statistical significance was set at $p < 0.05$.

## Results

During the period from 2004 to 2009, among the 280 patients admitted to our PMR center and classified as post-TBI, 107 patients met the inclusion criteria. Median duration of follow-up was 38 (8–66) months from the end of the intensive care period.

### *Baseline*

Patient characteristics relating to the severity of TBI are listed in Tables 1 and 2. Neurosurgical interventions were carried out in 28 patients (26%), mostly for compressive intracranial hematoma. A diffuse cerebal injury on the initial cerebral X-ray or MRI according to Marshall classification was found in 79 patients (74%) and was associated with a prefrontal lesion in 74 (69%) patients.

### *End of follow up*

At the end of follow-up, 104 patients (97%) were discharged home and 3 (3%) were admitted to a specialized institution for patients with severe cognitive disorders. Fifty-three TBI patients (49%) had resumed work or school activities at the end of follow-up whereas 51 (48%) were not able to return to a normal socio-professional life. Sixty eight patients (64%) were found to have a behavioral dysexecutive syndrome following assessment in the PMR department, including 12 (11%) with severe disinhibition. Only three patients had significant hypopituitarism, two had diabetes insipidus and took vasopressin (DDVAP) and one had a partial adenohypophysis deficiency that required hormonal supplementation.

### *Weight changes*

W1 to W2:

All the patients received enteral nutrition (including 18 by gastrostomy) during intensive care and (or) neurosurgery, but still lost a mean 11 ± 6 kg (IC 95%, 7.9–12) in 38 (7–364) days: W1 = 71 ± 13.5 kg *vs.* W2 = 60 ± 12.7 kg ($P < 0.001$).

W2 to W4:

All the patients were weaned from enteral nutrition, and from the gastrostomy, during their stay in PMR, once they recovered the ability to eat by mouth without any swallowing impairments. During the stay in PMR (W2 to W3), mean weight gain was 8.8 ± 7.8 kg (IC 95 % = 6.5–11.1, $P < 0.001$). W3 was 68.8 ± 11.5 kg and W2 was 60 ± 12.7 kg, corresponding to a mean gain of 80% of weight lost during intensive care. This change occurred over 3.9 ± 3.4 months, the mean duration of stay in our PMR unit. Between the end of PMR (W3) and the end of follow-up (W4) there was a mean weight increase of 3 ± 6.6 kg (IC 95% = 1.2–4.8) over a 32 ± 16.6 month period.

W1 to W4:

Mean BMI at W4 (23.8 ± 4.1) was not different to W1 (pre-TBI) (23.3 ± 3.9) ($P = 0.08$). However, we found that the patients could be categorized in three different groups at the end of the follow up: group 1, weight loss ($n = 30$, 28%: −8 ± 7 kg), group 2, weight stabilization ($n = 32$, 30%), and group 3, weight gain ($n = 45$, 42%: +9 ± 6 kg).

*Factors associated with changes in weight*

Changes in weight and BMI showed that patients with a lower initial BMI tended to become more overweight at the end of follow-up than patients with a higher initial BMI (Figure 1). There was a statistically significant relationship between weight change at W4 and pre-TBI BMI class for each of the three groups ($P = 0.015$): 54% of pre-TBI patients with a BMI > 25 lost weight during the follow-up compared to 14% with a BMI < 20. In addition, 59% of pre-TBI patients with a BMI < 20, and 25% of patients with a BMI > 25 gained weight during the

follow-up. Thus the weight loss during intensive care reversed for all patients on entry to PMR (W2) and the weight time courses of each group crossed over during the stay in PMR (W2-W3) (see Figure 1).

There was a significant relationship between the presence of hypophagia or hyperphagia and W4 weight group ($P = 0.001$). Patients in group 3 ate more than patients in the other two groups, with a 40% prevalence of hyperphagia compared with 16% and 10% in groups 1 and 2 respectively. In contrast, hypophagia was observed in 50% of group 1 compared with 31% and 11% in groups 2 and 3 respectively. There was, however, no statistically significant difference between the three groups for eating behavior (Table 3). The prevalence of a behavioral dysexecutive syndrome was, however, more frequent in the weight gain group (Table 3). There was no significant difference between the severity of TBI, assessed by the initial type of cerebral lesions, the GCS and the duration of coma or intensive care, for any group. There were no significant differences in the number of drug prescriptions at discharge from PMR and during the follow-up between the three groups. Neither did the level of physical activity, sex, age, weight loss during intensive care, level of physical activity, or modification of alcohol or tobacco addiction differ significantly between the three groups.

The multivariate analysis included categories of oral food intake, behavioral dysexecutive syndrome and classes of initial BMI in the model. The results showed that the factors which were independently related to weight gain were hyperphagic food intake ($P$=0.003, OR 4.5 (IC 95%, 1.6–12.1) and the presence of a behavioral dysexecutive syndrome ($P$=0.04, OR 2.5 (IC 95%, 1.03-6.3). Two factors ($P < 0.01$) were related to weight loss, hypophagic food intake (OR 4.1 (IC 95%, 1.5–10.9) and higher pre-TBI BMI (OR 4.9 (IC 95%, 1.7–14.0).

*Metabolic complications*

Before the TBI, 15 patients (15%) had a pre-existing metabolic disease: 3 had type 2 diabetes, 5 had arterial hypertension and 7 had dyslipidemias. The occurrence of new metabolic complications was 16% with no significant differences between the three weight change groups. During the post-TBI follow-up, 4 patients (4%) developed arterial hypertension, 3 type 2 diabetes (3 %) and 9 (8 %) a dyslipidemia. In contrast, type 2 diabetes associated with hyperlipidemia resolved in one patient with a normalization of previous obesity.

## Discussion

The results of this study showed that weight change during the rehabilitation period after TBI was correlated with level of oral food intake. Higher intakes (hyperphagia), and the presence of a behavioral dysexecutive syndrome, were associated with a higher weight gain than a low level of food intake (hypophagia), which was associated with lower weight gain or weight loss. The second, more paradoxical result, was that the patients with the lowest pre-TBI BMI had the highest BMI at the end of follow-up, while patients with the highest pre-TBI BMI had the lowest BMI at the end of follow-up. Patients could be categorized in three groups of weight change at the end of follow-up –loss, stabilization and gain. Only 3 independent factors were associated with the time course of weight change: nutritional state before TBI, based on the BMI, level of food intake, which significantly changed in more than one half (57 out of 107) of the patients after TBI, and dysexecutive syndrome for the weight gain group. In contrast, type of medication, duration of intensive care, age, sex and physical activity were not related to the type of weight change. All patients lost weight during intensive care and gained weight during the stay in PMR. These findings suggest that the TBI is responsible for changes in weight through the modification of food intake such as hyperphagia and hypophagia, probably as a result of disruption of the appetite control network following the brain lesions. However, it is difficult to establish the direct impact of TBI on feeding behavior and weight. No significant association was found between the type of cerebral injury according to the initial CT scan, and changes in nutritional status and eating behavior at the subacute/chronic stage. However, this is not a surprising finding, as standard CT and/or MRI assessment at the acute stage lack sensitivity to detect diffuse traumatic axonal injury [19]. It was therefore very unlikely that subtle functional or structural lesions involving the hypothalamus, which could explain changes in eating habits, would be detected with these techniques. A more detailed exploration of morphological lesions could be useful

to gain a better understanding of behavioral changes, including interest in food after a brain injury. More sophisticated imaging techniques, such as functional magnetic resonance imaging or Diffusion Tensor Imaging may in the future help to understand the relationship between brain injuries, morphological lesions and eating behavior following TBI [20].

It is well accepted that the brain, notably the hypothalamus and its complex network, plays a crucial role in the regulation of food intake [11]. It can therefore be expected that TBI will disrupt this regulation by altering the structures involved. Our study was the largest ever conducted in this field in a cohort of TBI patients of varying severity, with initial management in intensive care and secondary care in a specialized university center for TBI rehabilitation. To limit bias, we included only patients who were not in a neurovegetative state and who had not undergone previous digestive surgery that could disturb normal digestive physiology. We also controlled for potential associated factors, such as medication or physical activity in order to evaluate the specific effect of TBI on weight change. Given the low prevalence of hypopituarism: 3% in our study, 5% in a multicenter study [9] and 2.5% in a cohort of children[8], it is unlikely that this pathology plays a major role in weight change. It must, however, be noted that we did not carry out systematic hormonal examinations, as is considered to be optimal practice [21]. In addition, the results showed that there was no significant relationship between weight gain and the various drugs prescribed for these patients. Data regarding food intake and eating behavior were recorded at least twice in a 2-month period, through patient inquiries and confirmation by the family and relatives in the case of cognitive sequelae. No differences were found between the two inquiries, similarly to previous studies of alcohol consumption [22]. Most of the patients had recovered sufficient cognitive capacity to respond to the inquiry: for example 49% had resumed normal work or school activities by the end of the follow-up.

Weight change pattern after TBI was noteworthy: our results confirmed a constant significant weight loss in the intensive care unit of a mean 11 kg, despite the use of enteral nutrition. This is a well-known effect of severe post-traumatic aggression during the acute phase [23]. The goal of treatment is to maintain nitrogen balance but, more particularly, to control stress and reduce malnutrition through nutritional assistance. During their stay in PMR, patients regained an average of 8.8 kg, almost all of the weight lost, in an average time of 3.9 ± 3.4 months, which is twice that spent in intensive care. This weight regain was comparable to that found by Brooke [3], who evaluated the effect of nutritional status on functional outcome in 53 TBI patients. Their study describes a significant weight gain in patients during hospitalization in a rehabilitation unit, even exceeding their pre-morbid weight such that 60% of patients left PMR significantly overweight. Various factors probably contributed to the weight change during PMR. First of all, “intrinsic” factors related to the TBI *per se,* notably a dysexecutive syndrome which was present in 68 patients during the PMR hospitalization and was characterized by major cognitive disorders, including reduced inhibition which can lead to increased food intake [24]. This phenomenon is clinically similar to the genetic Prader-Willi syndrome [25]. Secondly, an “extrinsic” factor related to feeding management may have been involved. Because of the undernutrition during intensive care and the related potential complications such as bedsores, all the patients received hyperenergetic and hyperproteic diets, however no metabolic evaluations (for example with indirect calorimetry) were carried out to adapt the nutrition to each patient. Families also appear to have played a role in weight gain by overfeeding despite instructions from the dietician [2]. It appears that seeing their loved ones regain the capacity to eat and gain weight after a long period of artificial feeding and, for some, "wasting", could be seen by relatives as part of the recovery process. These factors probably maintained and worsened disorders which were directly related to the TBI. This illustrates the complexity of nutritional

management after brain injury. A diet adapted to the metabolic level of each patient could help to improve the nutritional changes. To do this, the systematic assessment of metabolic and morphological clinico-biological status is necessary at admission to PMR. Resting energy expenditure and body composition could be evaluated by bioelectric impedance or DEXA according to available resources, for example. Unfortunately, this was not done in the present study. Indeed, the changes we observed in weight could be related to fat deposition in the phase of regaining weight.

This study has important clinical implications. TBI is an important cause of eating disorders, particularly in the subacute phase, i.e. during rehabilitation. The correlation of weight change with hyperphagia or hypophagia and dysexecutive syndrome, suggests that TBI strongly disturbs the central areas which control food intake and weight set-points. It is therefore necessary to forestall these disturbances through a better assessment of nutritional needs, tailored to each patient, depending on available resources and technical feasibility. It is important to keep a watchful eye throughout the medium- and long-term follow-up, since we found that 42% of patients significantly gained weight and 28% significantly lost weight over a median of 38 months. The metabolic consequences of this weight gain in the medium term were demonstrated in 16% of patients who developed arterial hypertension, dyslipidemia or type 2 diabetes, although their average age was only 36 years. This could be a significant problem. Monitoring in the longer term might reveal many more metabolic complications, and therefore the extended follow-up of these patients should include weight and metabolic management. The weight loss found in 28% of patients could also have significant somatic consequences, since chronic undernutrition can impair functional recovery [26].

## Conclusion

The results of this study showed that over 42% of adult TBI patients gained weight and 28% lost weight over a period of 38 months. The amount of weight change was related to the level of food intake and was strongly determined by behavioral changes. Initial weight was inversely related to weight at the end of follow up and the time course of weight change was independent from the amount of weight loss during intensive care. The results of this study therefore suggest that TBI strongly disturbs quantitative eating behavior and body weight set-points. A study of the long-term metabolic consequences appears justified since in this 38-month study, 16% of patients developed a metabolic syndrome. Future studies should evaluate the related changes in body composition, mainly fat free mass and fat mass.

## Conflict of interest

The authors declared no potential conflicts of interest with respect to the authorship and/or publication of this article.

## Statement of authorship

PC, SH, JCM and PA contributed to the study design, analysis and interpretation. SH, ABM and MH collected the data. The paper was written by PC, SH and PA and all the authors read and approved the final manuscript.

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**Table 1. Baseline characteristics of patients (*n* = 107) before traumatic brain injury**

| | |
|---|---|
| Sex ratio: M/F | 81/26 |
| Age (mean ± SD) | 36 ± 13 |
| Weight (mean ± SD) | 71 ± 13.5 |
| BMI (mean ± SD) | 23.3 ± 3.9 |
| BMI classes: | |
| BMI < 20 | 22 (21%) |
| BMI between 20 and 25 | 61 (57%) |
| BMI > 25 | 24 (22%) |
| Metabolic disorders (*n*) | 15 (15%) |

**Table 2. Criteria of severity of traumatic brain injury (*n* = 107)**

| | **Mean ± SD or number (%)** | **Min** | **Max** |
|---|---|---|---|
| Initial Glasgow coma scale score (GCS)* | 6.8 ± 3.1 | 2 | 15 |
| Mild TBI (GCS 13–15) | 8 (9) | 13 | 15 |
| Moderate TBI (GCS 9–12) | 10 (10%) | 9 | 12 |
| Severe TBI (GCS < 9) | 76 (81%) | 3 | 8 |
| Coma duration (days) | 15 ± 10 | 0 | 68 |
| ICU duration (days) | 38 ± 40 | 2 | 364 |
| Post-traumatic amnesia (days) | 72 ± 89 | 2 | 510 |
| Cerebral lesions** | Diffuse injury | n=79 | |
| | Evacuated mass lesion | n=28 | |

* For 94 patients in whom the GCS was documented
** according to Marshall CT-scan classification
TBI: traumatic brain injury

**Table 3. Characteristics of patients (*n* = 107) according to weight change groups at the end of follow-up [38(8-66) months after traumatic brain injury]**

| | Total | Weight loss | Weight stabilization | Weight gain | *p* (between the 3 groups) |
|---|---|---|---|---|---|
| Patients (*n*) | 107 | 30 | 32 | 45 | |
| Sex ratio: M/F | 81/26 | 20/10 | 26/6 | 35/10 | NS |
| Age (yr) | 36 | 38 | 37 | 33 | NS |
| T1 BMI (pre-TBI) | 23.3 ± 3.9 | 25.0 ± 4.4 | 22.5 ± 2.5 | 22.6 ± 4.1 | *P* = 0.014 |
| Intensive care duration (d) | 38 ± 40 | 31 ± 18 | 30 ± 17 | 46 ± 56 | NS |
| Coma duration (d) | 15 ± 10 | 15 ± 10 | 16 ± 11 | 14 ± 11 | NS |
| Δ BMI in intensive care | −3.2 ± 1.5 | −3.3 ± 1.6 | −2.9 ± 1.4 | −2.8 ± 1.4 | NS |
| T4 BMI (end of follow-up) | 23.8 ± 4.1 | 22.4 ± 4.0 | 22.7 ± 2.6 | 25.4 ± 4.5 | *P* = 0.01 |
| Behavioral dysexecutive syndrome (*n*) | 68 | 16 | 18 | 34 | *P* = 0.04 |
| Hyperphagia (*n*)* | 26 | 5 | 3 | 18 | *P* = 0.004 |
| Hypophagia (*n*)** | 31 | 15 | 10 | 6 | *P* = 0.003 |
| Number of meals > 3/d (*n*) | 15 | 3 | 3 | 9 | *P* = 0.09 |
| Eating between meals (*n*) | 35 | 10 | 8 | 17 | NS |
| Taste preferences sweet (*n*) | 30 | 11 | 6 | 13 | NS |
| Taste preferences savoury (*n*) | 36 | 12 | 9 | 15 | NS |
| Physical activity (*n*) *** | 36 | 8 | 13 | 15 | NS |
| Drugs (*n*) | | | | | |
| neuroleptics | 19 | 9 | 3 | 7 | *P* = 0.09 |
| antidepressants | 38 | 13 | 9 | 16 | NS |
| analgesics | 45 | 15 | 11 | 19 | NS |
| anticonvulsants | 46 | 13 | 13 | 20 | NS |
| Addictions: alcohol/tobacco (*n*) | 4/17 | 2/5 | 0/4 | 2/8 | NS |
| Onset of metabolic disorders (including dyslipidemia) (*n*) | 16(9) | 6(2) | 2(2) | 8(5) | NS |

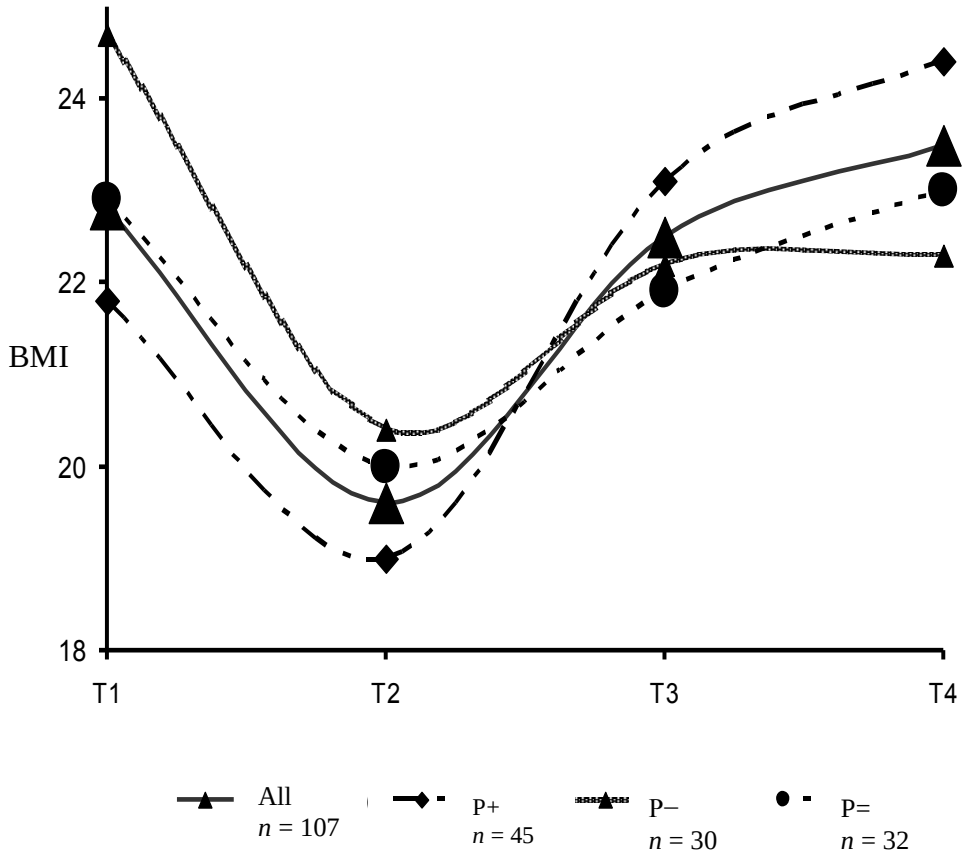
* defined by an increase of usual food intake compared with pre-TBI (T1); ** defined by a decrease in usual food ingestion compared with pre-TBI (T1)

*** According to WHO (World Health Organization)

TBI: traumatic brain injury

**Figure 1: Time course of body mass index in 107 adult traumatic brain injured patients**

P+: weight gain group. P−: weight loss group. P=: weight stabilization group.
T1: before traumatic brain injury. T2: end of intensive care. T3: discharge from rehabilitation.
T4: end of follow-up.
BMI = body mass index


24
22
20
18
BMI
T1
T2
T3
T4
All
n = 107
P+
n = 45
P–
n = 30
P=
n = 32