Technical and Research Department

Adult Malnutrition in Emergencies

An Overview of Diagnosis and Treatment

Field Guidelines

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I. Introduction:

Most field guidelines on malnutrition only make reference to children. When adults are considered, it is usually in a brief and uncritical manner. In fact, when emergency nutritionists talk of "malnutrition" they often understand "child malnutrition". This is not surprising, as children are the main victims of malnutrition in developing countries. Indeed, most of our current knowledge on malnutrition arises from research carried out specifically in children. Current protocols to treat malnutrition in adults are simple adaptations of the protocols developed for children.

Unfortunately, experience from the field has shown that these adaptations are not always satisfactory. The diagnosis and treatment of malnutrition in adults has specificities that go beyond adapting the amounts of energy necessary for weight gain. Field observations of adult malnutrition seem to contradict some of the diagnostic recommendations found in field guidelines and medical textbooks. This misfit between guidelines and reality triggered the implementation of several research projects by ACF and other organisations at the end of the nineties. The results of these investigations are now being translated into new guidelines.

In these guidelines, we present current thinking on the diagnosis and treatment of malnutrition in adults during emergencies. They are specific for adult patients. It is inevitable that they build on knowledge of child malnutrition, but they also present and critique recent research carried out specifically for malnourished adults in emergency situations. In this way, they will address the problems of identifying adult patients in need of nutrition rehabilitation, and the main features of their medical and nutritional management. In addition, the text will consider some strategic considerations that need to be taken into account when assisting a population where adult malnutrition is prevalent, and the means to evaluate if this is the case. These guidelines are based on those originally written for children (ACF and WHO guidelines[1]). We will refer to the original guideline whenever the treatment of adult and child malnutrition is similar.

The primary source of new information contributing to these guidelines is the work developed by the author as part of a PhD programme supported by ACF and the University of Aberdeen. Benefit is also drawn from the author's clinical experience in the field and, in particular, from the experience of several field teams of expatriate and local ACF workers (in particular those of South Sudan, Burundi, Congo, Sierra Leone and DRC). Our acknowledgement and sincere thanks goes to all of them.

Despite this large amount of work, malnutrition in adults still poses more questions than we have answers. I will present some of these in the text, with specific mention to those subjects that are still under debate, those that need validation, and other new developments that are still to be tested. These guidelines will meet their objective if they aid field teams in the task of improving the quality of nutrition programmes addressing adults, helping to save valuable lives and reduce poverty. In addition, we expect to stimulate critical thinking and promote new developments to improve the guidelines themselves.

[1] See Bibliography for details.

Importance of adult malnutrition in emergencies.

The majority of subjects presenting with Severe Acute Malnutrition in developing countries and during emergencies are children. However, this is not always the case: in some emergencies, adults are the group most affected by malnutrition[2], and NGOs need to adapt their treatment protocols and intervention strategies accordingly. Even in emergencies where the focus of the intervention is children, some adults will be identified as being potentially malnourished and in need of treatment. This is more often the case since the pandemic of HIV/AIDS, often present alongside malnutrition, seriously affects more and more populations in developing countries. Therefore, NGOs and other agencies working in the fields of nutrition and health need to be ready to answer the specific problems raised by the diagnosis and treatment of adult patients who are underweight or suffering from malnutrition as a result of famine, poverty or disease.

At the local and household level, the impact of the loss of an adult cannot be underestimated. From a social and economic point of view, the loss of an adult is more dramatic than that of a child (though both are equally regrettable). Adults are the main source of income and food for the rest of the group, they are the caretakers of the younger and older members of the group, and they are often the only means for the family to be represented in social structures. Indeed, assessments of vulnerability often consider the lack of the "head of household" (usually the mother or the father) among the key criteria to identify families at a particular risk of suffering the effects of the emergency (food shortages, malnutrition, and many other). The effect of the loss of one (or both) of the parents for the family and the social group have been recently demonstrated in the context of the HIV pandemic in southern Africa (Wilson 2001).

Most of the deaths related to malnutrition in adults can be avoided, in the same way that they are avoided in children. Avoiding adult deaths will reduce the burden of any emergency in the social group in individual, social and economic terms, for example, preventing an increase in numbers of orphans. It will also preserve the health and the lives of the main actors of post-crisis reconstruction: an invaluable asset.

[2] For example, in 1992 two thirds of the deaths registered in Baidoa (Somalia) were adolescents or adults (Concern Worldwide). The following year, 75 % of the deaths registered in the town of Melange (Angola) were in persons above 10 years old (ACF). In the 1998 famine in South Sudan 56 % of deaths reported were among adults older than 20 years (Epicentre). More than 60 % of ACF admissions to TFCs in Burundi (1999-2000), Congo (2000) and DRC (2001) were adults.

When do we observe adult malnutrition in the field?

It is unclear why high numbers of adults appear to be malnourished in some crises, while not in others. The duration of the crisis is often invoked as an explanation, with some crises lasting long enough to affect all levels of population and not only children. This explanation, though based on field observations[3] has never been proved, and is sometimes contradicted by field practice[4].

Whether adults are affected by the crisis or not may depend on the coping strategies taken at household level to share limited quantities of food. In some populations, adults may prefer to eat less in order to feed their children. Other reasons that may affect the distribution of malnutrition between age groups include: the severity of the food shortage (complete starvation for short periods vs. semi-starvation for long periods), the previous nutritional status of the population (i.e. the presence of large numbers of thin adults in the population before the crisis), the prevalence of chronic diseases (in particular tuberculosis and HIV), the severity of Public Health disruption, and so on.

Malnutrition in adults also occurs in situations of individual dependency. This is often the case for elderly people, those with mental illnesses or a severe physical handicap and prisoners.

In any case, it is highly suspected that malnutrition among adults is more prevalent during emergencies than is reflected by the numbers of patients assisted in nutrition programmes in the past. The concentration of activities on children, including initial assessments and nutrition surveys, and the standard implementation of programmes tailored for children, have probably hidden the true extent of malnutrition among adults in recent emergency interventions.

[3] This is based on observations made in hospitals during the siege of the Warsaw ghetto during the Second World War (Winick 1979).

[4] For example, in Congo, 2000 adults were affected since the beginning of the humanitarian intervention at the same time as the children. In other protracted emergencies adults do not seem to be affected in large numbers.

II. What is different in adults?

The main physiological changes and principles of management of malnutrition in adults are the same as those in children. There are, however, several differences between children and adults that need to be taken into account from the start. These affect the biology and physiology of the individual organism, but there are also other less obvious factors to consider which relate to their psychology and their role in society. These will all have an effect on the protocols and the general organisation of programmes provided. They are summarized in the following paragraphs[5].

Physiology:

The maintenance energy requirements for a child are about 95 kcal/kg/day (400 kJ/kg/day), while those for an adult are only about 38 kcal/kg/day (160 kJ/kg/day). The protein requirements of an adult for maintenance of body weight are about the same as a child (0.6g/kg/day). Therefore, an adult must have a diet which supplies 6 % of its energy as protein to maintain body weight, whereas a child only requires about 2.4 % energy from protein if he or she is not gaining weight[6]. These differences are related to the higher percentage of tissues with low energy consumption in adults compared to children (peripheral protein, fat), and are further increased by the need for children to maintain growth.

Adults, in general, tolerate a loss of a higher proportion of their body mass than children do. Consequently, adults will be less vulnerable to nutrient deficiencies and will require relatively less calories per kilo of body mass during rehabilitation from malnutrition.

On the other hand, children can protect themselves against a reduction in nutrient intake by simply slowing growth, reducing nutrient and energy needs. This may prevent the development of Acute Malnutrition – wasting- to the detriment of producing Chronic Malnutrition – stunting. Therefore, we can make the difference between Chronic and Acute Malnutrition in children by examining Weight-for-Height and Height-for-Age anthropometric indicators. In the absence of growth, adults cannot use this "defence" mechanism. A chronic reduction of nutrients and energy in adults will therefore only produce thinness, which will not be different, from the anthropometric point of view, from that produced by an acute and severe reduction of intake. The difference between **Acute Malnutrition** (producing metabolic distress and endangering the life of the patient in the short term) and simple long-standing thinness (with relative preservation of metabolic function and not threatening life in the short term) will need other diagnostic procedures that we will discuss in the following chapters (we will refer to the second as **Stable Malnutrition**, to avoid the confusion produced by the alternative term "Chronic Malnutrition").

[5] This section presents background information on adult malnutrition. From sections IV onwards, more practical advice on diagnosis and treatment is given.
[6] The amounts needed for rehabilitation of malnutrition are higher (see Treatment) .

Medical:

Malnutrition in adults is commonly associated with other diseases. These include chronic and acute infections, intestinal malabsorption, liver and endocrine disease, alcoholism and other addictions as well as cancer and AIDS. Even in times of famine, these conditions may present as the first cause of malnutrition. However, they may have been exacerbated by weakness, immune depression and weight loss in a malnourished patient, or be directly due to primary malnutrition itself. In all cases, both the underlying disease and malnutrition must be treated.

In addition, there are many diseases among adults, and particularly in older adults, that can present with weight loss and/or oedema as part of the clinical syndrome. The identification of malnutrition oedema in adults will require a careful differential diagnosis (unlike in children, for whom this differential diagnosis is quite straightforward).

Owing to differences in metabolism and body composition, dosages for most drugs are different in children than in adults.

Pathophysiology of malnutrition:

The physiological changes arriving during primary Acute Malnutrition in the adult patient have not been studied in detail. The few physiological studies performed during and after the Second World War (Keys 1950, McCance 1951, Winick 1979) seem to indicate that the main processes involved are the same as those described for children, including, for example the mechanisms for development of oedema and the pathogenesis of cardiac failure and pulmonary oedema. This is supported by clinical observations and the recovery of patients during treatment in TFCs with the protocols that were originally intended for children.

Psychosocial factors:

As in children, psychological factors may play a major role in the development and evolution of Acute Malnutrition in adults. Psychological trauma related to war and crisis, and the extraordinary hardship of life concomitant to any humanitarian crisis may take the form of depression (producing both anorexia and the loss of will and motivation to provide food) and several forms of reaction to stress. These are of particular importance if there has been loss of a close relative, loss of important family assets, displacement or violence (often, Post-Traumatic Stress Disorder). These can also be expressed in the form of changes in alimentary habits that may lead to malnutrition.

On the other hand, adults may adapt to difficult situations in ways that children cannot. Some adaptations may be beneficial to prevent the development of malnutrition in the individuals and their dependants.

This ability of the adult patient to take decisions concerning his or her dietary needs should be respected, rather than treating them "as children" for the simple fact that they are suffering from malnutrition or are severely ill. This involvement of the patient in decisions related to his or her treatment may include deciding the type of treatment most suitable for the patient (for example, when deciding between in-patient or home-based treatment).

Often, adults do not relate their thinness and oedema to their diet. In these cases, they may be reluctant to accept the change in the diet that the treatment involves, particularly when this means taking monotonous milk meals, which are often not part of the normal adult diet. Careful explanation of the condition of the patient should be given, including description of the milk diet as medicine. In addition, alternatives such as home-based treatment should be sought whenever possible.

Suffering from malnutrition may be perceived by adults in some societies as shameful, particularly among men, reducing the likelihood that the patient will present spontaneously for screening. In other situations, the fear of being identified by political enemies, or the fear of having to leave home and family behind without protection, contribute to the fact that many adult patients do not accept being admitted to nutrition centres. Again, new home-based strategies will be an acceptable alternative for most patients. In all cases, active screening of patients will be an integral part of any nutrition programme addressing adults.

Main effects of Acute Malnutrition in adults:

Among others, Severe Acute Malnutrition in adults produces:

- Impaired immune responses, with increased risks of infection.
- Reduced muscle strength and fatigue.
- Reduced respiratory muscle function, resulting in increased difficulties in breathing and expectoration. These can in turn increase the risk of chest infection and respiratory failure.
- Impaired thermoregulation with predisposition to hypothermia, particularly during infections.
- Impaired wound healing and delayed recovery from illness.
- Apathy, depression and self-neglect. Poor interactions with environment and with other persons.
- Poor libido, impotence and infertility. Poor pregnancy outcomes and poor mother - child interactions.

III. Brief review of methods to assess nutritional status in adults.

This section presents a brief overview of the main criteria that have been proposed in the past for the assessment of nutritional status of adults patients, both in developing and developed countries. It is intended as background information and, as such, it is not necessary for the reading of the following sections. From section IV we will concentrate on those practical guidelines and procedures more appropriate for the emergency setting. This section will also present a new approach, based on recent analysis of ACF experience in Congo and Burundi (Navarro-Colorado 2005). This new approach is still in development and needs to be validated in a wider variety of field situations.

As in children, Acute Malnutrition in adults can present as two distinct syndromes: Marasmus and Oedematous Malnutrition (the term Kwashiorkor is not usually employed for adults). The pathology of each of them is different, as well as the consequences on individual's health and the means of diagnosis. For this reasons, it is appropriate to study and present them separately.

1. Adult Marasmus:

BMI

The use of BMI to identify malnutrition in adults was proposed in 1986 by the IDECG[7]. This group of experts defined "Chronic Energy Deficiency" (CED) "…*not as a prolonged continuing loss of body energy, but as a steady state at which a person is in energy balance although at a 'cost' either in terms of risk to health or as an impairment of functions and health*" (James *et al*. 1988). They proposed a classification of chronic malnutrition based on Body Mass Index (BMI) (James *et al*. 1988) that was later simplified using the classification in the following table (Ferro-Luzzi et al 1992).

The simple classification of adult Chronic Energy Deficiency .

CED grade	**III** Severe thinness	**II** Moderate thinness	**I** Mild thinness	Normal
BMI (kg/m^2)	< 16.0	16.0 – 16.9	17.0 – 18.4	> 18.5

This classification was originally intended to identify *populations* with low mean levels of energy intake (the direct measurement of mean energy intake was discarded for being too complex and unreliable). The objective was to assess the need for intervention programmes and to prioritise them. However, the same classification was soon proposed to identify *individuals* suffering from malnutrition and in need of

[7] International Dietary Energy Consultation Group.

treatment, despite the fact that the classification was never validated with individual outcome data. It is important to note that the "deficiency" to which this classification refers is not a characteristic of the individual being measured, but a comparison of the mean "estimated" energy intake with that considered "ideal" in a "normal" population.

The IDECG also defined "Acute Energy Deficiency" (AED) as *"... a state of negative energy balance, i.e. a progressive loss of body energy"* (James *et al*. 1988), but did not explore the specific means to diagnose it. Instead, the classification of CED was later extended to cover lower ranges of BMI (CED level IV or "severe wasting", below BMI 13 kg/m^2 and CED level V or "extreme wasting" below a BMI of 10 kg/m^2), and recommended that all patients with BMI below 16 kg/m^2 should be treated during emergencies (Ferro-Luzzi & James 1996).

Underpinning these definitions there is the assumption that the so-called AED is no more than an aggravation of CED, and not a fundamentally different process, as their definitions implied (with CED representing stable thinness and AED a progressive weight loss with negative metabolic balance and dysfunction). Nevertheless, this classification has been reproduced in all guidelines until today, even though its application in the field was less than satisfactory[8].

MUAC

MUAC is recognised as a useful indicator to identify children with Acute Malnutrition. In particular, it is considered as the best predictor of risk of mortality in children under treatment. Its use for adults was originally proposed as a proxy for BMI, based on the observed correlation between both indicators in some populations. In this way, a MUAC cut-off point was estimated for each BMI cut-off point in the CED classification. As a result, the new classification was easier to use in the field, but had all the problems mentioned for BMI, plus those related to the fact that the correlation between both indicators is not perfect (around 60 to 65 % in most studies).

Pursuing this idea, new studies made during emergencies suggested that the correlation between BMI and MUAC waned below a MUAC of 185 mm (corresponding to a BMI of 13 kg/m^2 in that study). This cut-off was interpreted as the physiological point at which the patient had consumed all peripheral reserves and started consuming proteins from central (visceral) organs, and therefore the individual is at high risk of death (Collins 1996). They discarded the use of BMI because it was considered difficult to use in the field, and because it can be affected by the presence of oedema (but they did not consider developing separate criteria for patients with and without oedema, as is done for children). The same group, in a different study, observed that the use of clinical symptoms could identify patients with Severe Acute Malnutrition at least as well as BMI (Collins & Myatt 2000). Based on a combination of these observations they proposed the following classification.

[8] These guidelines will use the concepts developed by the ICEDG, but the terms "Chronic Energy Deficiency" and "Acute Energy Deficiency" are not used, as they imply a specific cause of malnutrition, while others are possible.

CHANCES model for assessing adult undernutrition in the field (Collins & Duffield 2000)

Therapeutic Feeding Centres

MUAC < 160 mm alone
MUAC 161 – 185 mm plus one of the following*:
- Oedema
- Inability to stand
- Apparent dehydration

Famine Oedema (Beatte grade 3 or worse) alone as assessed by a clinician to exclude other causes

* Additional social factors can be included in the model, such as: access to food, distance from centres, absence of carers, shelter, or dependants, lack of cooking utensils.

Supplementary Feeding Centres

MUAC 161 – 185 mm and no relevant clinical signs or few relevant social criteria.

Unfortunately, this classification has never been validated as such in any population. It is derived from research conducted in Nilotic populations, and in most of the studies which it is based upon, the outcome of the patients under treatment was not known. Recent analysis of ACF data from adults from Burundi and Congo has failed to validate it as a *diagnostic* tool. However, the use of MUAC as a *prognostic* tool can be recommended in the light of the observations presented by Collins and those done by ACF in Burundi and Congo (i.e. to identify patients with the highest risk of death among those already in TFC treatment).

More importantly, as with BMI, MUAC cannot differentiate between patients that are actively loosing weight and those that are stable (the requirement to differentiate between patients that may need urgent treatment and those that do not)[9].

Other tools to measure malnutrition in adults:

Weight loss:

The calculation of the actual weight loss is considered the best means by which to identify patients with Acute Malnutrition. Unfortunately, this requires accurate knowledge of the "usual" weight of the individual, which is rarely available in the context of emergencies. In clinical settings, unintentional loss of 10 % of body weight

[9] Practical advice on the use of BMI and MUAC, and their cut-off points, is given in the following chapters.

has been shown to be a good predictor of clinical outcomes. A combination of percentage weight loss and BMI, completed with a basic evaluation of presence of acute disease, is used to decide upon the need for nutrition support in UK hospitals (NICE 2006). This approach could be used in the exceptional situations in the field where patients know their "usual" weight (that is the case, for example, in Bosnia and in many urban centres in Africa). In many other situations patients do not know their "usual" weight, and different diagnostic procedures will be necessary (see next chapter). For patients who require a long follow up, as for those receiving treatment for a chronic disease (AIDS, tuberculosis, etc.), the first contact with the patient can provide a "baseline" weight that can be used to evaluate weight changes in the following visits, and hence to potentially diagnose Acute Malnutrition if the situation of the patient worsens.

Other anthropometric measurements:

The calculation of other anthropometric indices such as Ponderal Index (weight over the cube of height) or Weight divided by Height has not provided a better identification of patients with Acute Malnutrition than those obtained for BMI. The measurement of Skinfold Thicknesses (tricipital, subscapular, etc.), even when added to the measurements of MUAC or BMI, also fails to provide a better diagnosis. In addition, these measurements are difficult to obtain in a reliable and reproducible way. These measurements are of interest only in a research setting, to understand the proportion of fat and lean tissue in malnourished patients. The measurement of body composition through isotope dilution techniques, bioimpedance analysis, image techniques or other is only interesting for research purposes.

Laboratory tests:

Many laboratory tests have been proposed in western clinical settings to diagnose Acute Malnutrition in adults, including: the Creatinin-Height index (as an estimation of muscle mass); serum albumin or prealbumin (as a measurement of protein deficit and turnover); serum cholesterol and the estimation of immune competence (as a proxy for nutrition status, by delayed cutaneous hypersensitivity). Although these tests tend to be altered in malnourished patients, their normality does not exclude the diagnosis of malnutrition. More importantly, they can all be affected by concurrent disease, such as infections, and their values are altered by the presence of renal or liver disease, situations often found in patients with and without malnutrition during emergencies. As a consequence their usefulness in the clinical setting is very limited.

Subjective Global Assessment (SGA):

This approach defines malnourished patients as those that are "at increased risk of medical complications" (i.e. those that presumably will benefit from nutritional treatment). It takes into account the history of weight change and dietary change, as well as subjective assessment of gastrointestinal symptoms, functional capacity, presence of metabolic stress and general physical status of the patient. With this information, the patient is ranked as well-nourished, moderate or severely malnourished. This tool was developed to assess the need of nutritional support in surgical patients and individuals with systemic diseases in Western hospitals (usually malnutrition secondary to disease). It has never been validated in patients with disease

as a consequence of semi-starvation or in the emergency setting. In addition, it requires estimation of the actual weight loss of the patient. Appendix 1 presents the features of SGA, as they could be used to aid clinical interviews in the field.

Clinical assessment of nutritional status and dietary history:

Clinical assessment of the patient, including history of disease and diet, complete anamnesis and patient examination remains the best means to assess the need for treatment in individual patients. This can be done with the help of assessment tools like the SGA, and using experience and clinical judgement. The inconvenience of this method is that it takes more time and requires some expertise. In addition, it cannot be used for population assessment during surveys (this would require developing a simpler case definition). The following sections introduce the key features that need to be explored in order to diagnose Acute Malnutrition from a clinical point of view.

Functional tests of malnutrition:

Functional changes in Acute Malnutrition were first studied by examining changes in immune function, ability to perform work and changes in heart rate during intensive exercise. These tests are difficult to perform in sick patients and depend on the individual's previous exercise status. Other studies, also conducted in western hospitals, showed that handgrip strength was predictive of the development of post-operative complications. Jeejeebhoy's team has led a series of studies that show that hypocaloric feeding results in a fall of the membrane potential and in the concentration of intracellular potassium. They showed that Acute Malnutrition affects first cell energetics, before affecting changes in mass, resulting in measurable changes in muscle function, among others. As malnourished patients are often too weak to perform exercise tests, they explored methods that stimulate muscle function without involvement of voluntary effort, and that would not be affected by sepsis, drugs, trauma, surgical intervention or anaesthesia. These methods study fatigue of muscle contraction and relaxation rate after stimulating specific muscles with increasing frequencies. They showed that in malnourished muscle, relaxation is markedly slow. Handgrip strength (force of maximum contraction) was also shown to correlate with nutritional status. These altered processes would depend directly on the availability of chemical energy to induce contraction and relaxation, altered in malnourished patients. They also described the intracellular reactions explaining these effects. In other studies, muscle function (including handgrip strength, respiratory muscle strength and muscular relaxation rate) predicted surgical complications better than weight loss. In addition, they showed that, during treatment, muscle function is restored before body composition, and even before a raise in body protein content (for a summary of these studies, Jeejeebhoy 1998).

It is clear from these studies that there is good evidence that muscle function is an index of nutritional change and of the risk of complications in sick people, with the added advantage that it can be identified earlier than changes in weight or body mass. Its measurement can be used to identify recovery with treatment as well. There is still a need to develop a practical tool for clinical use. In Western hospitals, this can be the use of electrical stimulation (evoqued potentials), and in particular the measurement of the relaxation rate. In less sophisticated environments, the measurement of handgrip strength remains a promising tool that needs to be explored further.

Recent developments for the diagnosis of adult marasmus in complex emergencies (ACF study):

A recent study completed in ACF centres in Burundi and Congo-Brazzaville collected extensive information on malnourished adult patients (2409 treated in SFCs and 2500 treated in TFCs, all between 18 and 50 years). The anthropometric and clinical characteristics of these patients were compared by outcome of treatment. Separate analysis was done for patients under SFC weekly food supplementation and those in TFC intensive medical and nutritional treatment. The main interest of this study resides in the fact that many patients treated in SFC presented a BMI below 16 kg/m^2 (743 patients, representing what was considered at the time "severe acute malnutrition").

In this study, BMI was not able to predict outcome of treatment of the patients in SFC in a satisfactory way. MUAC prediction ability in SFC was even lower. Several combinations of clinical symptoms and anthropometry successfully predicted the outcome of patient treatment and could be used as indicators of patients in need of intensive treatment. However, logistic regression analysis identified the presence of three homogeneous sub-groups of patients in the sample[10]. These groups were identified by the presence of specific features in the clinical examination and history of the disease of the patient, as follows:

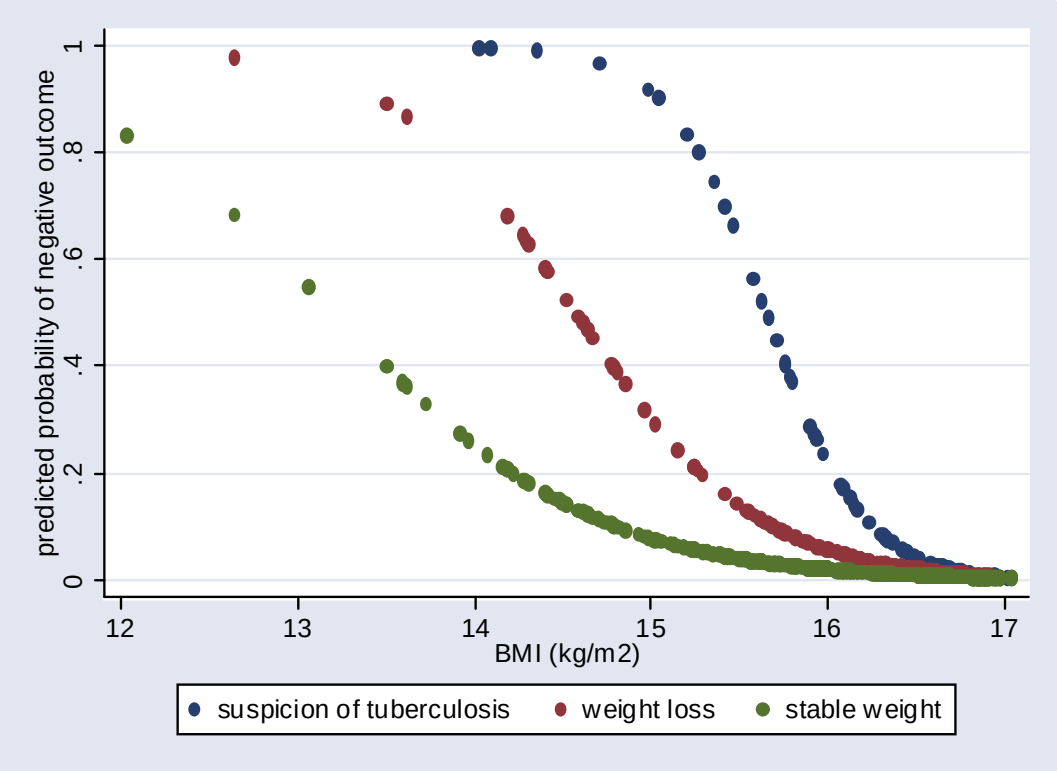
- patients with a history of recent weight loss before admission[11]
- patients with a cough present for at least 4 weeks before admission (representing malnutrition secondary or associated to chronic disease)
- patients with no history of weight loss (declared stable weight in recent weeks).

Once the patients were separated in this way, BMI showed a much more satisfactory ability to predict outcome of treatment in SFC within each of the groups. However, the predictive ability of MUAC in these groups remained low. The level of BMI associated with an increase in the risk of a negative outcome of treatment in SFC was not the same for each group. The Figures on the next page show the differences for BMI and for MUAC.

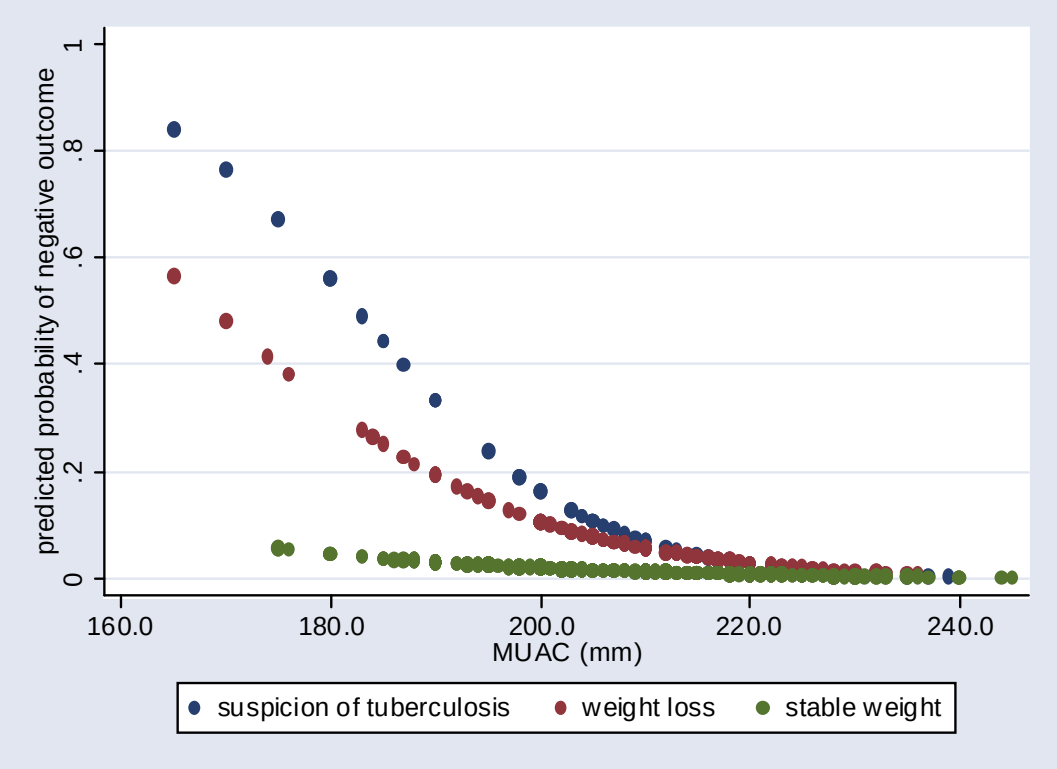
In summary, the graphs show that the BMI at which the risk of negative outcomes of treatment (death, transfer to hospital or failure of treatment) reaches a critical point is higher for patients whose thinness is secondary, or associated with chronic disease. This is followed by that of patients who had primary malnutrition and presented with active weight loss in recent weeks. The risk of negative outcome in patients with stable weight was raised as well, but it was only relevant in extremely low values of BMI.

[10] Subgroups identified through the presence of statistical interactions in the development of logistic regression models of anthropometry and symptoms in the outcome of treatment of the subjects.
[11] The exact duration or amount of weight loss was not considered.

BMI prediction of negative outcome: Comparison of patients with suspicion of tuberculosis, weight loss and stable weight.



MUAC prediction of negative outcome: Comparison patients with suspicion of tuberculosis, weight loss and stable weight.



The ability of MUAC to identify patients who would have a negative outcome of treatment was low in the three groups (and almost negligible in the group of patients with stable weight). However, due to the presence of a smaller sample size for MUAC than for BMI, this study could not exclude the interest of the use of MUAC for diagnosing Acute Malnutrition in these patients[12].

The analysis of patients treated in TFC showed a better (though not optimal) prognostic ability both for BMI and MUAC. The latter better identified patients who presented with secondary malnutrition or malnutrition associated to infection (chronic or acute). Clinical symptoms such as the inability of the patient to stand and walk unaided, muscular hypotony, extreme clinical thinness (the presence of symptoms resembling dehydration or "apparent dehydration"), skin lesions typical of malnutrition (thin desquamated skin sometimes evolving towards ulceration), anaemia on admission or oral candidiasis also contributed to the bad prognosis of the patient in TFC treatment.

As in SFC, the presence of weight loss in the weeks preceding the admission and the presence of chronic cough were the most important determinants of the evolution of the patient in the TFC. It is also interesting to note that the presence of weight loss before admission and that of muscular hypotony (as a proxy for "weakness") were highly correlated, and in fact, one could almost replace the other in the development of models for predicting outcome of treatment in the TFC. This observation reinforces those presented above on the importance of functional assessment to identify Acute Malnutrition.

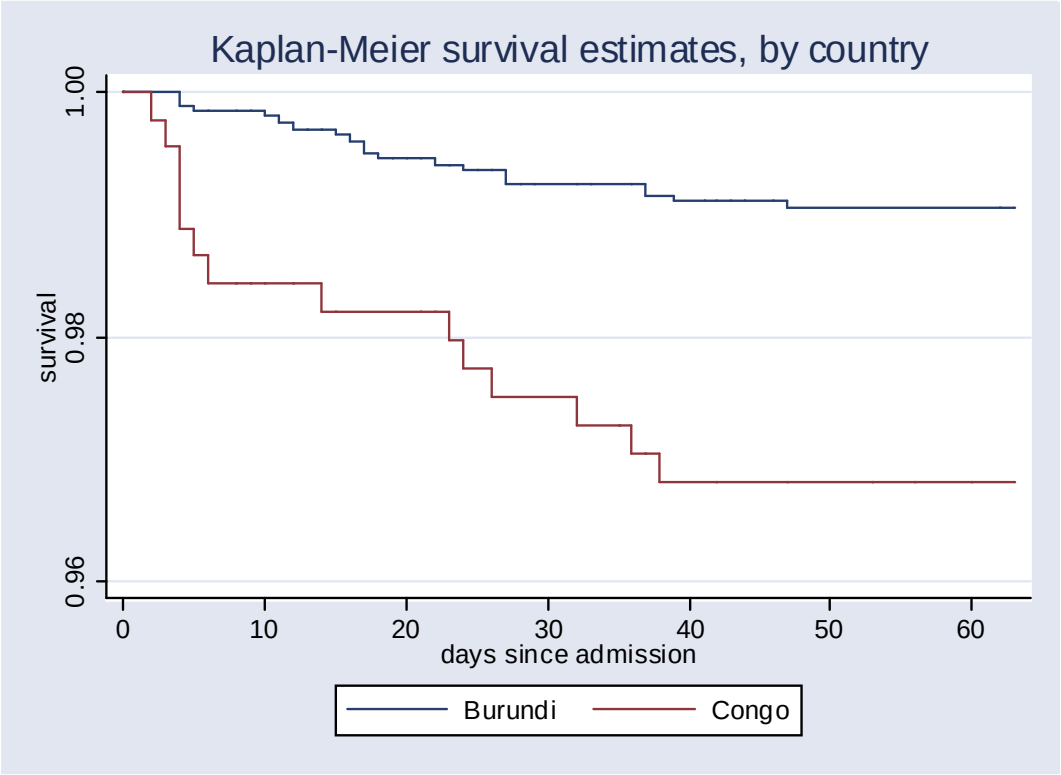
In addition, the study showed that for the same levels of BMI and MUAC, the patients treated in TFC in Congo (an acute humanitarian crisis) presented with a more severe clinical presentation than those in Burundi (chronic food shortages aggravated by an acute crisis). Patients in Congo presented more often with acute weight loss before admission, and their outcome of treatment was the worst overall, despite the fact that the same protocols were used in both interventions. This is shown in the following Figure by higher mortality in the first few days of treatment in Congo, which then stabilises over time, thanks to the effectiveness of TFC protocols. In the equivalent curve for Burundi, mortality is extremely low from initial treatment, and is not affected by the evolution of the treatment, implying that most patients were not at high risk of death when they were first admitted.

The study concluded that, in order to assess malnutrition correctly, we first need to be able to identify the patients that have a stable weight, despite their thinness, and separate them from those who are thin and are loosing weight actively. It is usually the second that present with severe weakness and higher risk of death. Once this division is made, BMI and clinical symptoms are useful to identify patients that need intensive treatment (in a TFP), while the others can recover with only food supplements of the type of those offered in supplementary programmes[13]. The study

[12] Further research in the role of MUAC in the diagnosis of malnutrition in adults is therefore necessary.

[13] In children, Weight-for-Age does not differentiate between children with Acute Malnutrition –wasting- and those with Chronic Malnutrition –stunting-. When only children with Acute Malnutrition are selected, the results obtained with Weight-for-Age and Weight-for-Height are equivalent. A similar

Death during treatment in TFC for patients in Burundi and Congo*.



* Y-axes truncated at a probability of survival of 0.96.

also concluded that patients with malnutrition secondary to acute or chronic infection have an increased risk of negative outcomes at higher levels of BMI, and therefore need earlier treatment (using different criteria of admission).

It is interesting to note that none of the systems to classify malnutrition presented in the previous section takes into account this important difference between long-standing thinness and active weight loss[14]. In order to present the protocols of diagnosis of malnutrition in adults these groups will be referred to as **Stable Malnutrition** (for thin patients that have a stable weight, a functioning metabolism and a low risk of death in the short term), and **Acute Malnutrition** (for thin patients with active weight loss and an increased risk of death in the short term).

thing happens for BMI in adults. Given the lack of growth in adults, the difference between acute and "chronic" needs to be made through other means: clinical or functional assessment of the patient.

[14] The previous classification systems were derived from studies done in homogeneous groups of patients, none of them mixing acute weight loss and stable thinness in the same sample.

2. Oedematous Malnutrition:

Acute Malnutrition in adults often presents with oedema. According to some observations (Winick 1979, Keys 1950) most adults with Acute Malnutrition will end up developing oedema if the condition is left to evolve long enough (and if the patient has not already died).

There has been extensive debate on the causes and physiopathology of oedema in malnourished patients. The most common explanation found in the literature, is that oedema of malnourished patients is due to hypoalbuminemia. This is based on the concepts developed by Starling in the 19th Century. Under this explanation, oedema would be the consequence of low oncotic pressure of plasma proteins, following a low protein diet. However, experimental studies in adults (Wuppertal 1951 among others) have shown that a low protein diet is not necessarily followed by a lowered plasma protein concentration, and patients with Acute Malnutrition are often observed with normal plasma protein concentrations.

The same observations have been made in paediatric Kwashiorkor, in particular through the experiments developed in Jamaica by Waterlow, Golden and colleagues. They showed that the main mechanism involved in the development of some of the symptoms of Kwashiorkor was an increase in oxidative stress with production of free radicals that would damage cell membranes. This process would be started by specific micronutrient deficiencies, and not only by the simple lack of proteins in the diet, though the exact mechanism and responsible nutrients are still not known. Free radical damage to cell membranes, and perhaps to other associated structures in the interstitial space, would be the final link that could explain the leak of fluids to the interstitial space and clinical oedema.

As noted by Golden, these observations coincide with those made in adults in the studies done in the mid 20th Century (quoted above). Although the debate is still open, it seems today that the processes leading to Kwashiorkor in children and to nutritional oedema in adults are the same.

IV. Acute Malnutrition and Stable Malnutrition: Definitions and General Description.

Definitions:

The key to the diagnosis of malnutrition in thin adults resides in the correct differentiation of patients suffering from active weight loss from those with a stable weight. Only the first are considered to have Acute Malnutrition. The others may be extremely thin without an increased risk of death in the short term, and we will refer to them as Stable Malnutrition[15]. These are the main definitions that we will use to classify these patients[16]:

Acute Malnutrition:

Acute Malnutrition represents a state at which the person is in negative nutritional balance, with progressive loss of body weight and nutrients. Acute Malnutrition associates thinness to other symptoms of weakness and specific clinical features related to nutritional deficiencies and progressive loss of function of specific organs. It is often complicated by associated disease. If left untreated the patient will deteriorate until death, often in the short term.

Stable Malnutrition:

Stable Malnutrition (also referred to in the literature on Chronic Energy Deficiency[17]) represents a state at which the person is in nutritional balance, despite being markedly thin. If the patient's metabolism remains in equilibrium, the risk of death is not increased in the short term (i.e. the patient doesn't need emergency treatment). However, the state of the patient is not "normal" and a return to a more appropriate body weight and nutritional status is desirable. The "cost" of Stable Malnutrition for the patient can be measured in terms of lower work capacity and performance, and a higher vulnerability to disease.

Acute Malnutrition is not an aggravation of Stable Malnutrition, but a different process. However, Acute Malnutrition can superimpose in patients that have Stable Malnutrition if their nutritional situation worsens.

[15] These guidelines propose "Stable Malnutrition" for this condition, to underline the non-progressive characteristic of these patients. This term is new and not present in the literature. Other names could be used and adopted, as the debate evolves.
[16] These definitions are made for practical purposes. They do not represent a formal classification.
[17] This nomenclature will not be used for two reasons : a) it implies that energy is the main problem leading to malnutrition, though other mechanisms are known today, and b) the use of the word "chronic malnutrition" in adults is often misinterpreted as referring to past stunting during childhood.

Principles of diagnosis and decision making:

The main objective of a medico-nutritional intervention during an emergency is to save lives. From this point of view, the purpose of this diagnosis is to identify the patients that need intensive treatment to save their lives and those that may benefit from simple interventions. Once a patient has been identified as suffering from Acute Malnutrition, we still need to decide which type of treatment is more appropriate for the patient, depending on the level of severity and complications.

For this purpose, patients with Acute Malnutrition will be further divided into those with Severe Acute Malnutrition and those with Moderate Acute Malnutrition. In **Severe Acute Malnutrition**, the risk of death in the short term is higher, and the patient's metabolism is not in a measure to utilise nutrients in order to gain weight and re-establish function. These patients need intensive treatment, including at least metabolic stabilisation, before entering the phase of weight gain. **Moderate Acute Malnutrition** refers to patients whose metabolism remains responsive, despite the active weight loss, such that they can recover with food and without need of medical stabilisation. All patients with clinical nutritional oedema are considered, *a priori,* as Severe Acute Malnutrition.

The mechanisms that gave place to malnutrition will also affect the characteristics of the strategy used in the field to address the problem and, in some cases, the type of treatment recommended for an individual patient. We refer to malnutrition as **Primary** when its main cause is a deficit in nutrients in the diet, or a deficit in the diet itself, and **Secondary** when it appears as a consequence of disease. The diseases producing Secondary Malnutrition are most often chronic (HIV, TB…) but can be acute as well (as is the case with children). The difference between Primary and Secondary Acute Malnutrition in an individual is not easy to note in the context of a humanitarian emergency, where severe food deficits are associated with public health disruptions, outbreaks of chronic and acute infection diseases, and often occurring in environments with a high prevalence of chronic and acute disease. In addition, malnutrition itself may be the cause of acute infectious disease, or promote the expression of latent chronic infections (e.g. tuberculosis), making the differential diagnosis between Primary and Secondary Malnutrition more difficult.

The major interest of making this difference is to understand the dynamics of malnutrition in a particular context, at the population level. When it comes to the treatment of the individual patient, the main objective is to identify the diseases present, and treat them, even if it is not possible to tell if they are the cause of the malnutrition or its consequence!

Finally, in a clinical context we can refer to malnutrition as **Complicated**, when it is associated with other diseases (coincidentally or as a consequence of malnutrition itself), and **Non-complicated** when it is not. Severe Acute Malnutrition almost systematically presents with complications during emergencies. Patients with Stable Malnutrition may present complications as well, often similar to those prevalent in well nourished patients. The complications associated with Acute Malnutrition will largely define the need for specific treatment in each particular patient.

V. Some Typical Medical Histories of Malnutrition:

Stable Malnutrition:

Stable Malnutrition can be the consequence of several processes.

a. Stable Malnutrition can be the consequence of chronic disease. This can be infectious (tuberculosis, malabsorption) or non-infectious (some cancers, hyperthyroidism). If the disease is severe, it will be more likely to result in Acute Malnutrition.

b. Acute disease can also be the cause of long term thinness. When an individual suffers from an acute disease (e.g. infectious diarrhoea), anorexia can develop, producing weight loss. In normal circumstances, the recovery from acute disease is usually accompanied by an increase in appetite and reactivation of anabolism, which translates in weight gain and recovery of previous weight. In times of hardship or famine, this recovery of weight may be hindered by the lack of food availability at the household. As a result, the weight after the acute disease is lower. Repetition of this process over time, particularly during frequent displacement and outbreaks, can end in Stable Malnutrition. During protracted emergencies, it is not rare to find thin individuals with a stable weight, who can precisely state the time at which they lost weight, some years before.

c. Long-term semi-starvation, if not severe, can produce Stable Malnutrition. Although no metabolic mechanism of adaptation has ever been described, some patients report very light loss of weight over long periods of time that do not translate into symptoms and signs of Acute Malnutrition.

d. In poor areas, and in populations suffering from durable and protracted emergencies, many young adults that appear to be thin may not have lost weight at all. This could be the case for individuals that completed their growth (including adolescence) during times of hardship, while suffering food shortages. Consequently, they never arrived at a "normal" BMI and the thinness observed is not related to weight loss.

e. A similar situation could be "normal" for populations with "tall and thin" phenotype, like the Dinkas and Nuer of South Sudan, the Samburu and Massai of Kenya, and others. The estimation of population average BMI in these populations needs standardisation in order to be compared with other populations (see below).

When Stable Malnutrition is the consequence of a disease that is active, this disease needs to be treated. Except for the last two examples, weight gain is desirable, as it will reduce the vulnerability of the individual to disease and further nutritional deterioration. However, intensive treatment, such as in a TFC, is not necessary for patients with Stable Malnutrition (see below).

Acute Malnutrition:

Acute Malnutrition always entails a complex picture of medical and nutritional disturbances, whatever the origin of the condition (primarily nutritional or secondary to a disease).

Even in hunger strikers, probably the simplest case of "pure" Acute Malnutrition (often taking place in a "healthy" environment, without the added risks of infectious diseases, etc.), serious metabolic disturbances appear after three or four weeks of refusal of feeding, leading to serious medical problems and death in 6 to 10 weeks, if no treatment is provided.

Anorexia Nervosa is another type of Severe Acute Malnutrition often observed in western countries. These patients tend to attain important weight loss before metabolic disturbances or associated symptoms (other than thinness) can be observed. This is in part explained by the high content of micronutrients of the usual diet of anorexic patients (particularly in relation to the small amounts of energy absorbed) and the "aseptic" environments in which these individuals tend to live. However, in bulimic patients, when the quality of the diet is disrupted by large amounts of foods of less nutritional quality and vomit is provoked by the patient, metabolic and medical disturbances appear rapidly, together with signs of Acute Malnutrition, increasing the risk to the life of the patient.

Anorexia of psychogenic origin can be found in Africa, often as a consequence of Post Stress Traumatic Disorder in violent environments. In these situations, the picture is expected to be more complex than in patients of Anorexia Nervosa in western countries, due to the limited micronutrients in the diet and the Public Health disruptions and associated exposure to infectious diseases. If identified it should be treated in the same way as any other Acute Malnutrition, with the addition of the referral of the patient to the appropriate Mental Health Services.

Acute Malnutrition during emergency conditions or during famines is usually due to semi-starvation. This can take several forms:

- several short periods of complete starvation (while hiding in the bush, for example), alternated with periods of "normal" feeding,

- a prolonged period of food deficit, producing partial starvation (the patient then usually reports that they only eat "once a day", or "not every day"),

- more often, it can take the form of changes in the composition of diet, which concentrates on the less food-dense products and is deprived of micronutrients and vitamins (while sometimes preserving the total amount of energy consumed).

In any of these cases of Primary Acute Malnutrition, soon after the patient starts loosing weight there will be associated symptoms of weakness (often described as "vertigo", though it is not such). The patient is soon unable to perform usual tasks at work, in the fields, or at home, and tries to rest most of the day, if there is the possibility of doing so.

At the same time, associated diseases will appear that can be the consequence of malnutrition or simply be associated with it. These associated diseases can often be interpreted by the patient (and the physician) as being the cause of the malnutrition. In fact, it is not always easy to distinguish which was first.

In emergency and non-emergency situations, Acute Malnutrition can be the consequence of chronic disease. The most common causes of Acute Malnutrition secondary to chronic disease are infectious (e.g. HIV/AIDS, Tuberculosis, Kala-Azar, etc.), but non-infectious causes can be observed in any setting among adults (e.g. cancer, endocrine diseases).

Adult Acute Malnutrition can also be the consequence of acute infections. Many infections cause associated anorexia and weight loss. If the patient does not recover weight after the clinical infectious episode, he/she can develop Acute Malnutrition or Stable Malnutrition, depending on the availability of food for the patient at the time among other factors. When treating these patients, it will be important to assess if the original infection that began the malnutrition is still present and remains to be treated.

In other situations, a succession of acute episodes of infections, not allowing complete recovery of the patient, is the cause of malnutrition (as an extreme example, malnutrition has been observed in the field as a possible result of frequent pregnancies, in an environment of food shortages).

VI. Protocols for the diagnosis of malnutrition in adults.

Marasmus:

In the absence of a definitive single objective indicator to identify malnourished adults that need treatment, the diagnosis will be based on a combination of objective measurements (anthropometry), history taking and clinical examination of the patient. The process for the diagnosis of marasmus in adults is summarized in the form of a flow chart at the end of this section. It is important to keep in mind that these recommendations represent a proposition that will need to be validated and refined as it is used in field programmes[18].

These recommendations are specifically for adults (18 to 50 years). See the text in the boxes for recommendations for adolescents (14 to 18 years), elders (above 50 years) and pregnant or lactating women.

After collection of the patient's history and physical examination, you should be able to answer the following questions:

- **Does the patient present Acute Malnutrition or Stable Malnutrition?**
- **If acute, does the patient need intensive treatment (stabilisation in TFC)?**
- **Is malnutrition complicated or associated to other diseases? Do they need to be treated now? If yes, where (in TFC, at home, in hospital)?**

1. Selection of thin patients for evaluation:

1.1. BMI:

The first step for diagnosis is to identify the patients that need to be evaluated further. This is done by selecting patients that present objective thinness, using Body Mass Index (BMI). BMI is calculated as the weight of the patient *in kilograms* divided by the square of the height *in metres*:

$$\text{BMI} = \text{weight} / (\text{height})^2$$

The tables in Appendix 4 can be referred to obtain the BMI of an individual without making the calculation. See Appendix 2 and 3 for recommendations on the measurement of Weight and Height. Normal BMI extends from 18.5 to 25 kg/m^2.

In the context of an emergency in a developing country, use a BMI below 17 kg/m^2 to select thin patients for further evaluation.

[18] In this sense, your experience may be useful to improve these guidelines. Contact headquarters with comments !

In some instances, a person with a BMI between 17 and 18.5 kg/m^2 may be actively loosing weight. In this case, the need for specific treatment (medical or nutritional support) will be assessed after a clinical evaluation (see below). However, in the context of an emergency it is preferable to select patients as explained above, as they are the most likely to need treatment.

Alternatively, MUAC can be used instead of BMI. Patients with a MUAC below 210 mm can be considered thin, and selected for further evaluation.

Notice that a BMI of 17 kg/m^2 or a MUAC of 210 mm are suggested here only for screening of the patients that should be examined in detail. They are not admission criteria, nor criteria for malnutrition. The identification of Acute Malnutrition depends on the following criteria:

2. Diagnosis of Acute Malnutrition vs. Stable Malnutrition:

2.1. Weight loss:

Ideally, the combination of a low BMI and a 10 % unintentional weight loss during the last 3 – 6 months is enough criteria to declare Acute Malnutrition and decide on the necessary treatment.

Unfortunately, the amount of weight loss can seldom be estimated in a reliable way from the information provided by the patient. In the absence of this information, Acute Malnutrition can be suspected in the presence of the following:

2.2. Physical strength / Muscular tonus:

The presence of **muscular weakness** should be evaluated both during clinical examination, and on the verbal opinion of the patient.

During physical examination:

- Observe patient's gait and posture. Explore strength by the means of a handshake. Explore muscles in limbs for signs of flaccidity.
- Can the patient move around without help, or do they need help to stand up and walk? Inability to stand and walk unaided is one of the most important signs of severity of Acute Malnutrition, carrying the highest risk of death.

Subjective information from the patient:

- Ask the patient: are you able to carry out normal daily activities (including washing, moving around, eating), or do you need help from others to accomplish them. In this case, when did the patient start needing help from others? Is the patient becoming dependant from others due to weakness?
- Ask for the patient's usual occupation. Ask: Can you still work as usual? When did you start feeling that you were not strong enough to complete the work? When did you have to stop?
- Ask the patient directly about feelings of muscular weakness or loss of strength (some patients describe it as "vertigo", though it is not the case!).

If the answers to the previous questions suggest the presence of significant weakness, discard other possible causes different from malnutrition (muscular or neural lesions, for example).

Progressive Weakness is a key symptom of Acute Malnutrition. If the patient presents normal strength, it is unlikely that he/she has Acute Malnutrition.

2.3. Subjective weight change:

Weight change in recent weeks is a key indicator to help differentiate between those with Acute Malnutrition and those with Stable Malnutrition.

Ask patients what their normal status was (weight, cloths size, etc.), and if they think that they have lost weight. Ask them to estimate the amount of weight lost, if possible.

Recent weight loss is the main sign of Acute Malnutrition. In the absence of an objective assessment of the amount lost (see above), consider the subjective estimation from the patient in the light of the other information provided.

2.4. Changes in diet and appetite:

Ask the patient to broadly describe what and how much they are currently eating (how many times a day and main products). Ask if this is the usual diet, or if it was different before the problems started. Try to assess what has changed in terms of quantity of food, frequency and quality, and estimate when these changes took place.

Ask the patient if he/she has their normal appetite. Ask if the patient is still hungry after the main meal of the day, and how often they go to sleep at night feeling hungry.

Some patients stop eating due to anorexia while there is still enough food for the family. Find out now if this is the case. This is often due to the trauma suffered during the conflict, bereavement, or due to infections and associated diseases. Malnutrition itself may be a cause of anorexia, but it can also be the consequence of associated disease. The increase in appetite observed during the first stages of starvation only lasts some days. Conversely, the presence of good appetite (normal or increased) reveals a good functioning metabolism, the absence of severe infection or hepatic disease and a proper functioning of regulatory feedback systems.

A patient who **has not eaten** at all during the last 5 – 7 days is at high risk of Severe Acute Malnutrition. These patients should be admitted to a therapeutic programme regardless of the other indicators (including BMI). Once in the programme, their status should be evaluated to identify the reason for not eating. Refer for treatment (if due to acute disease) and observe the effect of initial re-feeding. Most patients will be in a situation to be discharged home (or transferred to OTP) after a few days.

2.5. Mental and psychological changes:

Feelings of depression, apathy and reductions in intellectual capacity are classical signs of Acute Malnutrition, particularly if these have progressed in recent weeks, and in parallel with signs of weakness or weight loss. These are usually obvious to the clinician during the examination and may coincide with lack of collaboration from the patient. Talking to the family of the patient can help identify recent mood changes.

No matter how thin, a patient that looks happy and optimistic, and with good mental and intellectual reactions is unlikely to have Severe Acute Malnutrition.

Depression, apathy and slow intellectual reactions are key symptoms of Severe Acute Malnutrition in need of intensive treatment.

2.6. Is the patient loosing nutrients?

In some occasions, the presence of active loss of nutrients by the patient is obvious during the first examination. Note the presence of diarrhoea, vomiting or other possible sources of nutrient loss. These symptoms are not necessary for the diagnosis of Severe Acute Malnutrition, but their presence increases the chances that the patient may need intensive treatment. If possible, identify the cause of the symptom and treat it.

3. Symptoms and signs of Acute Malnutrition (Marasmus):

In addition to the symptoms and signs explored in the previous section, a profound physical examination of the patient will be necessary to identify the characteristics of Acute Malnutrition and the presence of other diseases. In order to better understand the clinical picture of the patient, it is helpful to distinguish the symptoms of malnutrition from those produced by its complications and those from diseases that may accompany or be the cause of the malnutrition.

Apart from weakness, weight loss and mood changes, other symptoms that can point to the presence of Acute Malnutrition include:

- Muscle wasting (obvious in the temporal areas of the head, with evidence of zygomatic bone, and in extremities and thorax, with prominent spine and ribs),

- Loss of subcutaneous fat (often revealed as apparent surplus skin, more obvious in the buttocks –"baggy pants"- and face –sunken eyes-),

- General appearance of dehydration, even though the patient is well hydrated (review the differential diagnosis of dehydration and Acute Malnutrition in ACF-guidelines). Normally, it is safe to assume that there is no dehydration if the symptoms have evolved over a period of days, rather than hours, and if there is no suspicion of important liquid and electrolyte losses (no diarrhoea or vomiting),

- Hair changes: hair appears thin, sparse and easy to pull-out (weak roots). Decolouration of hair may be present, but it is most typical of oedematous malnutrition,

- Skin changes: usually present as thin, dry, flaky skin, particularly in extensor areas of limbs. It can evolve towards infection and ulcers, more often in the legs. A black subcutaneous deposit is often described, particularly in legs and in the back (parallel and in both sides of the vertebral spin) but this sign is difficult to see in black skin or with inadequate light. Decolouration of the skin is often seen in patients with oedematous malnutrition,

- Amenorrhea in women and sexual impotence in men. Infertility and loss of libido, usually associated with apathy and depression,

- Associated infections (see below).

As noted above, there are no definitive laboratory tests to help on the diagnosis of malnutrition in adults.

4. Wrapping up the information: Acute Malnutrition or Long-standing Thinness?

With the help of the flow chart presented at the end of this chapter, consider the information collected through the five previous steps to decide if the patient has Severe Acute Malnutrition. A patient that presents with weakness and apathy, particularly of recent progression, and reports changes in weight and dietary habits or anorexia is most probably Acutely Malnourished. For the patients where weight loss or physical strength cannot be evaluated (or the results are not conclusive), a weighted evaluation of the other characteristics is necessary. This is a subjective decision based on the clinical presentation of the patient and their history.

Evaluate the progression of the condition, by comparing the time at which each of the previous symptoms started. Do they seem to be related?

Conversely, a patient who has preserved strength, or whose strength has only waned slightly or over a long period of time, presenting with good intellectual reflexes and a positive mood and who does not report change in weight or diet, is likely to be exhibiting Long-standing Thinness. In these cases, intensive treatment is not necessary, but it is interesting to continue the clinical evaluation to understand the reason for Stable Malnutrition and to provide appropriate action if possible (see following chapters).

Assessment of Acute Malnutrition (Marasmus) in Older patients (>50 years)[19]:

Older persons in developing countries and poor societies tend to be thinner. There is a progressive loss of fat content with age (opposite to what is often observed in wealthy societies). For this reason, it is common practice to reduce the threshold of BMI or MUAC used to consider an older individual as thin. In addition, this age group is most commonly affected by degenerative diseases that will be almost systematically present from a certain age. Consequently, the differential diagnosis of malnutrition and its associated diseases is more difficult in this age group.

Individuals with a BMI below 16 (or a MUAC below 180 mm) should be selected for evaluation of their nutritional status. As with other adults, this does not imply a diagnosis of Acute Malnutrition: the diagnosis is given by the combination of weakness, weight loss, and the clinical presentation of the patient. This threshold can be modified (i.e. lowered) when resources are scarce, the prevalence of Stable Malnutrition is high or if the capacity of the centres is already overwhelmed.

The measurement of height can be complicated by the presence of kyphosis or in patients that cannot stand. See Appendix 2 for alternatives ways to measure height in these patients.

Social factors may play an important role on the development of malnutrition, particularly in this group. Admit to TFC malnourished patients who do not receive support at home (and those living on their own), patients that are physically or mentally disabled, not strong enough to cook for themselves or those who are psychologically traumatised (loss of home or family members, for example). If the patient arrives at the TFC without a caregiver, make arrangements to find one in the family or in the centre, but start treatment even if one is not available.

Older adults may have problems eating some of the foods proposed during treatment (RUTF paste, porridge, family plate…). Check that the patient is able to eat them and if not, consider changing the therapy (by diluting the food, or changing it to other more palatable or easier to accept options for the patient).

Pregnant and Lactating women:

See general ACF guidelines for specific diagnosis and treatment of Acute Malnutrition in these groups.

[19] In many developing countries, adults 50 years old or above are classified as "Older adults". Use this to classify patients as "old" unless there is a different local threshold. Consider that there may be local preferences and perceptions of who is old. From a social point of view the definition of old may be made by the community itself.

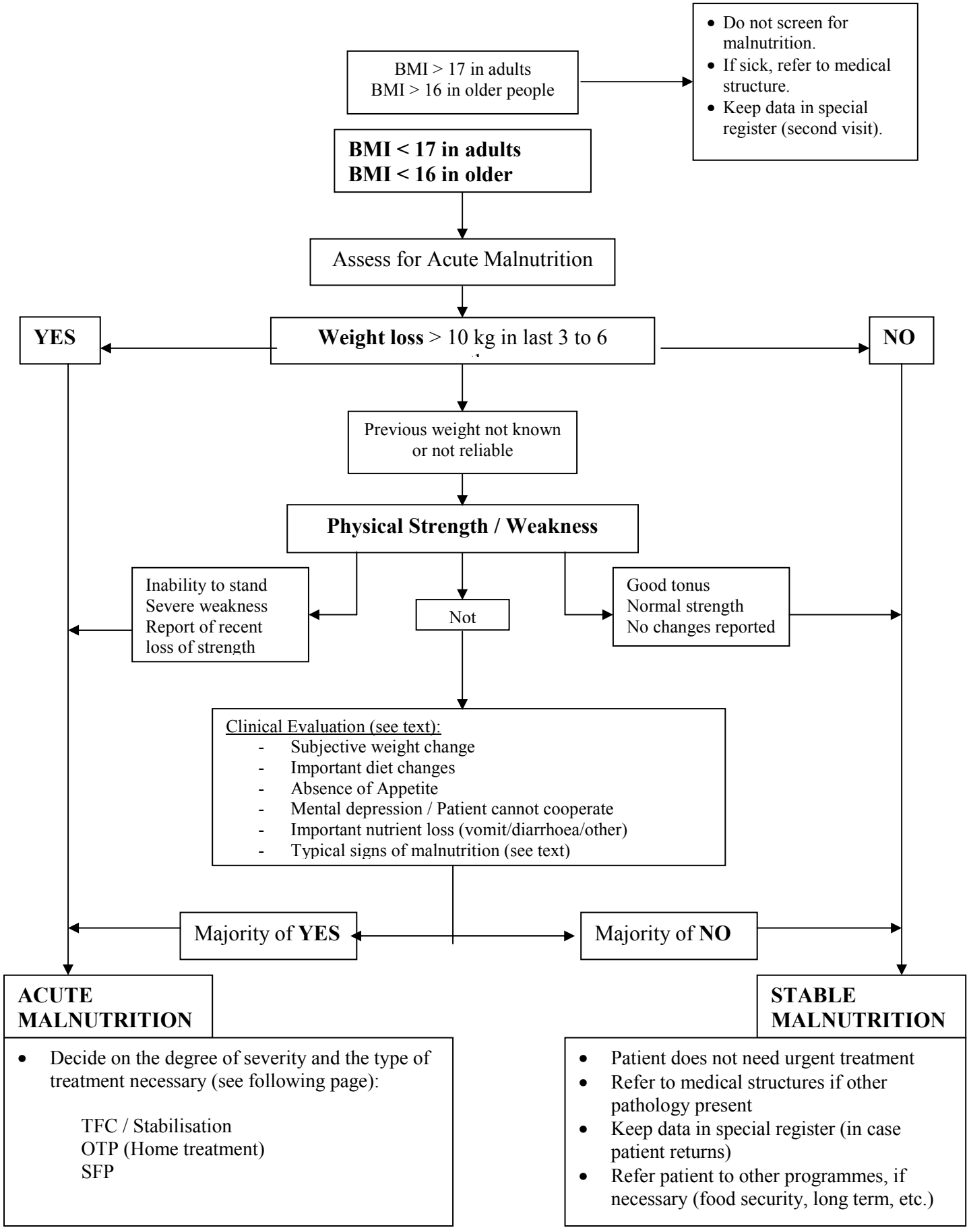
Assessment of Acute Malnutrition (Marasmus) in Adolescents:

Despite intensive debate, there are no agreed and tested anthropometric indices for the diagnosis of Acute Malnutrition in adolescents. Extended Weight-for-Height tables are the most accepted criteria, and those preferred in ACF (the appropriate tables can be found in ACF-guidelines). These tables represent an extrapolation from the data used to define Acute Malnutrition in children. They can be used for older children and adolescents before the growth spurt.

However, Weight-for-Height tables will tend to overestimate malnutrition in boys and girls that have completed their growth. In these cases, the approach proposed in these guidelines for adults may be more appropriate. For individuals that have started the puberty but not completed growth, both approaches can be used to get an individual assessment of the patient, taking into account that, for them, Weight-for-Height tables give a valuable indication of their nutritional status, but not a definitive diagnosis. The state of evolution of puberty and adolescence can be assessed by direct observation of changes in primary and secondary sexual features. These include the size of penis and testis, pubic hair, breast and, easiest to observe in any culture, axillary hair.

Conversely, some individuals may reach the age of 18 or more and not have completed physical growth. These boys and girls may have the habitus of an adolescent, despite their age. This is particularly frequent in areas where Chronic Malnutrition is prevalent (late puberty). BMI will grossly overestimate thinness in these patients. As an example, a young man measuring 160 cm and weighting 40 kg would be considered thin, as his BMI is 15.6 kg/m^2. However, if this individual had an adolescent body habitus and diagnosed as such, we would note that his Weight-for-Height percentage of the median is 83.4, which would exclude Acute Malnutrition.

Flow chart for acute malnutrition in adults without oedema:



Malnutrition Oedema:

The characteristics of patients with oedematous malnutrition are similar for adolescents, adults and older people presenting this syndrome. They are described as one group.

The oedema of malnutrition is an accumulation of fluids in the interstitial space, producing swelling of the affected area. The oedema is only clinically evident after a collection of at least two litters of fluid (sometimes more!). Therefore, its development may be associated with an increase in weight and sometimes oliguria. The swelling of oedema masks veins, tendons and bones normally visible in the feet. A severe oedema may represent the accumulation of 10 or more litres of extracellular fluid.

1. Characteristics of the oedema of malnutrition:

- **Pitting**: depression remains after 3 - 5 seconds thumb pressure.
- **Bilateral** and **symmetrical**.
- **Dependent**: it accumulates first in lower extremities, and can then affect the trunk, hands and other parts of the body. It can begin as periorbital oedema.
- **Moving**: the location of the oedema can change throughout the day, depending on the position of the patient. In patients that are lying down all day, it can often be found in the sacral region of the lower back or in genital areas.

The severity of oedema is measured in relation to its extension:

Levels of oedema [20]:

0	Absent
+	Obvious oedema on the foot or ankle
++	Oedema up to the knee (tibial)
+++	Oedema above the knee (can affect up to inguinal area, arms, face and even express itself as total body oedema (anasarca)

--- Consider Acute Malnutrition from Level ++ oedema ---

Opposite to what is usually described for children, liquid retention in adults can affect visceral regions, including the frequent development of **ascites** and, in the most severe cases, pericardial and pleural **effusions**.

[20] A different classification can be found in other texts, with the following levels: 0 Absent; 1 Minimal oedema on the foot or ankle; 2 Obvious oedema on the foot or ankle; 3 Oedema up to the knee (tibial); 4 Oedema up to the groin (inguinal area); 5 Total body oedema (anasarca). We prefer the one presented in the text in order to follow the same classification for children and adults, and avoid confusion.

Oedematous malnutrition is not usually painful, as the liquid accumulated is not in tension. The skin above the oedema may be thinner or present with desquamous lesions or ulcers. The skin is usually the same temperature as the rest of the body.

Only in patients with severe oedema over a longer period of time, can there be signs of local pain, and even limb impotence. In these cases, there is usually indurated skin over the oedema area. The differential diagnosis with other pathologies is more difficult, and recovery is slower.

Oedematous malnutrition in adults usually develops in days/weeks, but the history of the oedema can date back several months. In some cases, there is a history of changes in intensity of oedema, and even spontaneous recession.

2. Other characteristics of the adult patient with oedematous malnutrition:

All the symptoms of kwashiorkor usually observed in children can be seen in adults, but they are less common. These include the presence of:

- Anorexia,
- Weakness (like in marasmus),
- Enlarged liver (fatty liver),
- Full moon face (signs of excess of circulating cortisol),
- Skin lesions: atrophy, patches of erythema or hyper pigmentation, desquamation. Skin breakdown and ulceration (giving a typical aspect similar to burns),
- Pale sparse hair and hair loss (weak roots),
- Decolouration of skin and hair,
- Gynecomastia,
- Thinness is common, but subcutaneous fat can be present,
- Associated infections (see below) and other sings of immune depression,
- There may be hypovolaemia despite the oedema,
- Amenorrhea in women; sexual impotence in men. Loss of libido.

Irritability is less common, perhaps because the adult patient can express pain and distress better, but the patient presents with more “diseased” effect, as with adult patients with marasmus.

If these symptoms are present, the diagnosis of malnutrition oedema is almost certain. However, most of these symptoms will only be present in the most severe cases. It is possible to see patients with severe malnutrition oedema and few other associated symptoms. In these cases, a good differential diagnosis is necessary to exclude other causes of oedema.

3. Differential diagnosis of oedema:

The differential diagnosis of oedematous malnutrition is usually made with other conditions presenting with generalised or bilateral oedema. This will take into account not only the characteristics of the oedema, but also the presence of other clinical signs

and a history of the patient that may suggest the cause of the oedema and the diagnosis.

From a population point of view, one of the main characteristics of oedema of malnutrition is that it tends to happen in clusters of "epidemic" forms. In other words, a sudden increase of cases of bilateral pitting oedema in a localised population is probably due to malnutrition, particularly if there are conditions of food insecurity, displacement and public health disruption. In these situations, exploration in the community will reveal cases of individuals dying after having developed skin decolouration and oedema. In these circumstances, it can be assumed that most bilateral oedema that has had recent onset can be related to malnutrition (sophisticated differential diagnosis of the cause of oedema may not be necessary).

In other instances, malnutrition oedema can present in individuals (usually those most vulnerable) as isolated cases. Even during a famine, other causes of bilateral oedema may be present. These other causes need to be discarded with a proper differential diagnosis. A detailed description of conditions that can cause bilateral pitting oedema similar to malnutrition oedema is in Appendix 5.

The prevalence of most diseases producing oedema increases with age. This factor needs to be taken into account to evaluate the chances that the patient has non-nutritional oedema (for example, the differential diagnosis of bilateral pitting oedema in a young adult is straightforward, while it can be difficult in an older adult).

Other forms of malnutrition oedema:

Re-feeding oedema:

After a long period on a diet low in calories, proteins and salts, the sudden reintroduction of these nutrients can produce oedema (possibly due to the mineral corticoid effects of insulin and their effect on electrolyte balance). This is usually observed in individuals that, following a food crisis, recover a good diet (e.g. returnees). They are usually weakened and thin, due to the food crisis, but not necessarily malnourished. They may present to screening centres complaining of recent appearance of oedema. The oedema of these patients has usually appeared in a few days/hours, is bilateral and symmetrical, and pitting, but only affects feet or lower limbs. It is not usually intense, and can change over the course of the day. Typically, these patients do not present other associated symptoms of malnutrition oedema, nor of other diseases. They may even look energetic and in good mental and physical shape. The lacks of associated symptoms, plus the recent history of an improvement in the diet, make the differential diagnosis. If admitted to a TFC, the oedema of these patients regresses in only two or three days (even less), thanks to the low sodium and low protein content of F75. If admitted by mistake, the patient can be discharged home in the following days, with the recommendation of avoiding salty and protein rich foods for some days, or they can be kept in the centre to reintroduce a normal diet progressively. Re-feeding oedema is often also observed in TFCs at the time of introducing F100, even among patients admitted with no oedema. The management of these cases is presented in the following chapter.

WHO TO TREAT:

Treatment options for patients with Acute Malnutrition:

TFC / Stabilisation:

Patients diagnosed with Acute Malnutrition (after exclusion of Stable Malnutrition) and with:

- Bilateral Oedema (++ or more), or
- BMI < 13 kg/m2, whatever the presentation[21], or
- Objective Weight loss > 10 % in 3 / 6 months, or
- Unable to stand, or
- Recent severe weakness otherwise unexplained, or
- Severe apathy (patient cannot feed himself), or
- Patient that hasn't eaten at all in the last week, or
- Anorexia, or
- Complications with deterioration of general status of the patient (severe infection, shock or other –see list of acute complications below).

In all cases, re-evaluate after some days and consider transfer to OTP. The patients in Stabilisation can progress to Phase II or to OTP when they show the following signs:

- No severe illness or severe medical complications (minor illnesses and infections can be treated at home), and
- No oedema, and
- Good appetite, and
- Able to walk, and
- Strong enough to prepare the food for themselves and carry out basic daily tasks, and
- Have a supportive social environment (in case of older adults living on their own, discuss the pros and cons of the treatment at home with the patient!).

OTP (Home Treatment):

Patients with Acute Malnutrition can be admitted directly into OTP if they present with:

- No oedema (or + only), or
- BMI between 13 and 15 who do not meet the criteria for admission in Stabilisation centre (13 and 14 for older adults), or
- Patients with failure of treatment in SFP with unknown cause (if medical cause, keep in SFP and address to medical services).

[21] Cases of Stable Malnutrition below a BMI of 13 have been observed in the field, but they are exceptional. All patients below this threshold should be admitted to TFC.

In all cases, these patients need to present with enough strength and ability to cater for themselves and to walk to the weekly distributions. They must also have sufficient appetite.

SFP:

Admit directly to SFP:

- Patients with BMI between 15 and 17 who do not need meet the criteria for TFC (BMI between14 and 16 for older adults) (see above).

Some general considerations:

In older adults, always evaluate the capacity of the patient to follow treatment at home and ability to walk to and from the distribution centres. If this is not a possibility, consider treatment in a centre or other solutions.

If the patient is suspected of having an underlying chronic disease (e.g. tuberculosis), refer for diagnosis and treatment and ensure that he/she will be able to receive both treatments if necessary.

Patients with severe illness and those presenting with associated chronic disease will probably need the intervention of a physician, or support from a hospital. In all cases, the Stabilisation Phase of treatment is of radical importance for the recovery of the patient, independently of the other therapies proposed by the physician. This treatment can be given at the TFC or the Hospital, depending on the organisation set-up in each project. **Wherever the treatment takes place, the nutritional support (usually F75) and follow-up of the patient must be continued**: even while transferred to hospital, the patient remains a TFC beneficiary. The practice of refusing admission to the TFC and delaying nutritional treatment "until major illnesses have been taken in charge" cannot be justified: it denies the patient the opportunity to recover and the right to receive appropriate medical and nutritional treatment[22].

[22] In the past, some TFCs excluded the admission of patients presenting conditions such as: Heart disease, Renal disease, Diabetes, Cancer, Ulcers, Gastritis, Infectious diarrhoea, Meningitis, or Surgical problems, as well as those with a Psychiatric condition or with no caregiver. Most of these situations are amongst the most frequent causes or consequences of malnutrition. By applying this policy, it is easy to devoid a TFC of its purpose! Instead, appropriate protocols of understanding should be developed with the local hospital or other health services, or ACF services upgraded, to ensure that these patients will receive appropriate treatment and follow up for associated medical problems, while they receive medical and nutritional treatment from the TFC.

Other services:

Patients who fit the description of Stable Malnutrition (mainly, thinness, stable weight and no severe complications) should not be admitted to TFC or SFC. However, even if their life is not at risk in the short term, their status is not "normal". It therefore remains ACF's responsibility to ensure that they will benefit from the programmes and services in place. For this:

- invite the patient to come for screening after three/four weeks if possible (sooner if the patient's situation deteriorates), to evaluate weight change (for this, it is important to keep track of the patient –in a specially dedicated registration book-, so that weight can be compared at this visit, with that in the next visit),

- ensure that the patient has access to General Food Distribution or other food or social support services,

- consider the inclusion of the patient in other support programmes, like Food Security projects, Community Kitchens, etc.

- if the patient presents with mild disease, ensure they are seen in a Health Centre or equivalent services,

- if there is suspicion of underlying chronic disease (e.g. tuberculosis), refer for diagnosis in the appropriate services (e.g. National Tuberculosis Programme).

Complications and Associated Disease:

1. Usual Complications of Severe Acute Malnutrition:

- **Anaemia**: Presenting as pallor and fall in haematocrit. After some days of treatment of Severe Acute Malnutrition, haemodilution may simulate anaemia (pseudo-anaemia). Only consider anaemia that has been present since admission for treatment.

- **Diarrhoea**: Restore fluids with water. Use ReSoMal for cases of rehydration only. Watery diarrhoea is common in patients on milk treatment. Patients taking RUTFs may observe changes in stools during the first days ("pasty stools" due to increase in fat content). Reassure the patient and ensure they are taking water with the RUTF. Provide specific treatment only if associated fever, bloody diarrhoea or if diarrhoea reveals malabsorption and interferes with weight gain.

- **Acute infections**: Can appear in any body system. As in children, they can be silent (present few symptoms, including no fever). They can be increased in case of immune depression (due to malnutrition or to other condition): candidiasis, respiratory, urinary, ORL infections, etc.

- **Hypoglycaemia**: This can be a consequence of infection or consecutive meals missed. If severe, treat as infection. Emergency treatment as in children.

- **Hypothermia**: In the malnourished patient hypothermia is usually a consequence of infection. Warm the patient and treat as infection.

- **Congestive heart failure**: often expressed as tachypnoea, tachycardia, dyspnoea, and sings of cardiac overload. It is more frequent after overfeeding or fluid overload (associated with rehydration or transfusion).

- **Arrhythmias**: extrasystols and bradycardia are often related to a reduction in blood potassium and weakness of the heart. Tachycardia and other arrythmias can present as a consequence of anaemia, infection and other complications.

2. Underlying chronic diseases:

- **HIV/AIDS:** There are no clinical symptoms or signs that permit the suspicion of HIV/AIDS in a patient presenting with Severe Acute Malnutrition. Even the most severe presentations resembling AIDS, with complicating infections and severe attain of the patient, can be the consequence of the immune depression produced by malnutrition itself. The only definitive diagnosis is provided by a positive blood test for HIV. Referring the patient for diagnosis when possible, and treating all patients as if they were HIV negative until proven otherwise, is the best attitudes to have in the context of an emergency.

- **Pulmonary Tuberculosis:** suspected when patients present with cough of three weeks or more. Refer for diagnosis and treatment at the local National Tuberculosis Programme or equivalent services.

- **Extra-pulmonary Tuberculosis:** This is relatively frequent in patients with immune depression, including malnourished patients. Presentation can include: pleural TB, lymphadenitis, Bone TB, Abdominal TB, Meningitis, etc. If suspected refer to the National Tuberculosis Programme for diagnosis and treatment.

- **Cancer:** Suspected by clinical symptoms present (tumours, abdominal mass, etc.) and can usually only be confirmed in an equipped hospital. Depending on the clinical state of the patient and the evolution of the tumor, nutritional rehabilitation may improve prognosis.

- **Visceral Schistosomiasis** (bilharziosis), only in endemic areas (see differential diagnosis of oedema for a basic description).

- **Kala-Azar**: only in endemic areas. Suspect when malnutrition is associated to persistent fever, abdominal distension or pain due to splenomegaly. Other symptoms may include diarrhoea, moderate hepatomegaly, lymphadenopathy, pedal oedema, darkened skin and cough.

- **Alcoholism** and alcoholic hepatitis can cause malnutrition. Refer the patient to appropriate services if possible.

- **Mental disease / neglect**: Patients with mental disease, handicapped individuals and elderly patients usually depend on others for their basic care, including hygiene and feeding. Malnutrition may be the consequence of abandonment and neglect by caregivers. This is particularly frequent during emergencies, when resources for the family are limited, and affects individuals with mental disease (true or imagined by the family, including epilepsy) and elderly deemed “non-productive” or a “burden” for the family. Start treatment in the TFC and refer to appropriate mental and social services when possible (but avoid blaming relatives, as this is counter-productive and often unfair).

HIV screening in TFC?

Ideally, all patients presenting with severe weight loss or Acute Malnutrition should take a diagnostic test for HIV, and those presenting with a cough for more than 3 weeks a TB screening. This may not be possible in all places were ACF works. If possible, refer to the organisation or the National Programme in charge of diagnosing and treating these diseases to organise the diagnostic procedures, ensure that all the correct ethical procedures are in place and best available treatment is given, while continuing re-feeding protocols.

Confirmed HIV/AIDS and Acute Malnutrition:

Weight loss is an important feature of HIV/AIDS ("HIV wasting syndrome" or "slim disease"). The main causes of weight loss and malnutrition in HIV and AIDS include:

- chronic diarrhoea (produced by HIV itself, or by secondary infection),
- chronic fever plus weakness directly associated with HIV/AIDS (i.e. wasting syndrome),
- opportunistic diseases and other secondary infections (e.g. tuberculosis),
- altered body metabolism and anorexia,
- reduced food intake as a consequence of anorexia (in relation with infections), difficulty to swallow (oral and esophageal candidiasis) but also due to poverty and exclusion.

The nutritional needs of patients with HIV/AIDS are increased, even if the patient is asymptomatic, and during recovery of infections(including when the patient receives anti-retroviral treatment). In all cases, malnutrition has been related to poorer clinical outcomes of AIDS. **HIV/AIDS patients with clinical Acute Malnutrition should receive the same nutritional treatment as other malnourished patients, in addition to other specific treatment.**

Other HIV/AIDS patients that have not developed malnutrition may need some sort of nutritional support, except if their nutritional intake at home is demonstrated to be appropriate.

BMI, together with CD4 count, have been shown to be the best indicators of prognosis in HIV/AIDS patients, even after anti-retroviral treatment has been started. Two considerations need to be made when treating malnourished patients with confirmed HIV:

- Consider the effect of anti-retrovirals on the nutritional status of the patient (hyperlipidemia, fat redistribution and insulin resistance are possible side effects of Protease Inhibitors). If necessary, adapt HAART protocol.
- The choice of antibiotics for prophylaxis and syndromic treatment of infections needs to be adapted for patients with HIV (e.g. Co-trimoxazole for respiratory infections and for routine treatment on admission, or Ciprofloxacin for diarrhoea and urinary infections)

3. Other associated conditions:

Often, adult patients admitted to ACF programmes will present with other conditions that need treatment, relatively independent of malnutrition or aggravated by it. These can include wounds (during malnutrition wounds may take longer to heal), traumas, gynaecological infection or STDs, malaria, and many others. Depending on the set-up of the intervention, these can be treated in the TFC or OTP, Health Centre, by a visiting physician from hospital, etc. Most will tend to improve as the nutritional status of the patient recovers. However, they may need specific treatment as well.

On the other hand, it is known that some diseases may regress during Acute Malnutrition, in particular autoimmune diseases (including rheumatic conditions, and some types of diabetes mellitus), allergic diseases and hormonal syndromes (hyperthyroidism). These conditions will reappear when the patient recovers nutritional status. Other acute conditions that depend on the inflammatory system for clinical expression may be asymptomatic during Severe Acute Malnutrition and only become apparent when the patient recovers a good nutritional status (this may include infections, even Tuberculosis).

4. Primary or Secondary Malnutrition:

The treatment of Severe Acute Malnutrition is the same, whether it is Primary (as a consequence of lack of nutrition intake) or Secondary (as a consequence of disease). The only important difference is that in the second case other treatments may be necessary for the complete recovery of the patient. However, it is not always necessary to know whether a particular disease is the cause of malnutrition, in order to decide to treat it. In all cases, ensure that the patient receives the nutritional treatment, in the centre, at home or in hospital, and do all possible for the patient to be treated for concurrent disease (in the centre or otherwise).

There is a double interest of knowing if patients' malnutrition is due to food and nutritional factors or to disease:

1) to be able to diagnose the condition and treat the patient accordingly (or refer the patient to the appropriate preventive or curative services), and
2) to have a better knowledge of the causes of malnutrition in the area, and adapt intervention strategies accordingly (e.g. in some locations it may be better to set up an HIV/AIDS screening programme with nutritional support for HIV patients, while in others it may be better to set up a TFC and refer HIV patients to specific services).

This information is of interest for patients presenting with Stable Malnutrition as well as for those presenting with Acute Malnutrition. In patients with Severe Acute Malnutrition, and particularly in those severely ill, this information can be collected in the days following the admission, when the patient starts to feel better and is in a better condition to talk.

In order to help define whether malnutrition is primary or secondary, a chronological history of the disease can be collected, considering the following points:

Ask the patient to describe if the initial weight loss (or debut of weakness) was associated with any symptoms or other diseases. Also ask for symptoms of infectious disease, including diarrhoea or fever and about any treatment received at the time and the results. Does the patient think that there is a relation between those symptoms and the loss of weight/strength? Which was first?

If the patient reports other associated symptoms at the time of loosing weight try to ascertain if those symptoms preceded the feeling of weakness, or followed it. It is better to use weakness than weight loss for this purpose, as the symptom is noticed earlier (many patients can loose several kilograms before noticing it: this is usual in areas where people do not use belts or where loose clothes are the norm).

Get information on the duration of the symptoms *before* the patient started feeling the weight loss or the loss of strength (for example: cough 3 months before weight loss).

Consider that the first phases of Acute Malnutrition are often accompanied by diarrhoea or acute infection, and that the feeling of weakness is often expressed as "vertigo" or other symptoms that may mislead the patient into thinking that there was another disease, rather than the malnutrition itself.

Despite patient's explications, it is not wise to assume a cause and effect relationship between the symptoms explained and the presence of malnutrition. Except in some cases in which the timing is clear, most conditions could be the cause of malnutrition or its consequence. They could also be coincidental with no cause-effect relation at all.

While asking for the conditions around the initial loss of weight and its date, the patient may refer to political and social events, like the time when they became a refugee in the woods for example. A good knowledge of these events will help to date the time line of the problem, and better understand the process and its characteristics.

5. PATIENTS WITH SEVERE ILLNESS!

Some patients may arrive to the centre in a severe state. The treatment of these patients is a priority. The evaluation of the nutritional status can be made once the patient is stabilised. Severe conditions often observed on admission include:

- loss of consciousness, confusion
- shock
- severe dehydration
- hypothermia
- severe pain
- severe vomiting
- severe weakness (including inability to swallow)

The recommended action to take in these cases is presented in the next chapter.

Future perspectives for diagnosing Acute Malnutrition:

As presented in the previous chapters, recent developments point towards the objective evaluation of muscular function (strength and weakness) as a possible simple diagnosis method to identify patients with Severe Acute Malnutrition. In field conditions, this is likely to take the form of evaluation of handgrip strength, or other related simple measurement.

The protocols for diagnosis and the criteria of admission proposed here are based on recent research and field experience, BUT they are not definitive. They need to be tested and validated, and enriched by extra field experience. In order to do so, these guidelines can be developed and standardised in the form of a decision flow chart or a scoring system, and the results of its implementation evaluated in relation to the results of other tests and treatment.

The criteria by which to decide the best treatment for each patient (presented above) are based on clinical observation and some objective measurements. These skills can be perfected with experience. For this reason, it is important that you evaluate the consequences of the diagnostic and therapeutic decisions taken (by following up the evolution of patients admitted to the centres AND of those not admitted). In this way, the quality of the decisions can be improved and, if the results of the experience are communicated, the protocols themselves can evolve in the right directions.

VII. Treatment of malnutrition in emergencies:

The treatment of malnutrition involves the provision of specific foods and the treatment and prevention of specific complications. It is misleading to divide the treatment into "nutritional" and "medical" treatment, as both are part of the same protocol (e.g. therapeutic milks address some "medical" aspects of the malnutrition, and some of the drugs provided are nutritional supplements or are necessary for the nutrients to be absorbed correctly). Some of the complications and associated diseases will need, however, specific medical expertise. The characteristics of these complications (e.g. tuberculosis) will determine the facilities where the treatment will take place. This will be different in each project, depending on the strategic choices made by the intervening agencies: some TFCs may be able to treat a large spectrum of associated disease, others will rely on a nearby hospital or vertical programmes and it is possible that some services will not be available at all in emergency situations. In conclusion, a comprehensive treatment of Acute Malnutrition will need the intervention of several actors (NGOs, Health Services, Non-Health programmes): their coordination and communication will be key for the success of the treatment.

Today, there are many possible strategies to address Acute Malnutrition (centre based, home based (OTP), hospital based, etc.). Several of them may be present in the same programme, working in coordination, and the set-up will be different depending on the epidemiological characteristics of malnutrition in the area (see next chapter) and even evolve along the implementation of a programme in the field. They are presented individually here for clarity:

Moderate Acute Malnutrition – SFC treatment.

Despite the different energy requirements of adults, the amounts of food distributed in SFC and its composition are usually equivalent to those given for children. This is meant to ease the functioning of the centres. The amounts should be adapted to the local circumstances as well as the types of foods distributed. As with children, the foods distributed to adults should be fortified with vitamins and minerals (see ACF Guidelines).

The quantities to distribute are:

Wet feeding: 500 – 1000 Kcal / person / day

Dry feeding: 1000 – 1500 Kcal / person / day

The food ration should provide:

- 10 to 15 % of energy from proteins (1 gram of protein = 4 Kcal),
- 30 to 35 % of energy from fat (1 gram of fat = 9 Kcal),
- 50 to 55% of energy from carbohydrates (1 gram of carbohydrates = 4 Kcal).

Dry rations are larger than wet ones to take into account sharing of the product with other family members. This sharing may be particularly important if the beneficiary is

the person in charge of feeding the rest of the family (head of household), in which case, even this quantity may be insufficient (explore if this is the case in front of an adult patient in dry SFC feeding not responding to treatment).

Adults (and in particular older people) are less likely to eat foods that they are not used to. Make every effort to identify which foods could be used that are acceptable to them, and adapt recipes to ensure appropriate protein, fat and micronutrient requirements. In addition, some foods may be difficult to prepare or digest for older patients. Cooking demonstrations and health education in the centres are a good opportunity to evaluate whether the foods distributed are adapted to older patients ability to prepare and eat them. If foods are not considered appropriate, discuss possible changes or adaptations.

Systematic treatment in SFC:

Vitamin A: 200 000 IU in single dose (except pregnant patients, women of reproductive age or those that already received it at TFC).

Anti-helminthics: 500 mg single dose.

Folic acid: 1 cp of Iron-Folic Acid (200 mg of Iron; 40 mg of Folic Acid), weekly, for one month.

The consultation made by the nurse in the SFC should be an opportunity to evaluate the presence of other diseases, as well as the development of signs of weakness or other malnutrition related signs. These signs may be the evidence of a deterioration of the patient, on top of weight loss, and involve the referral of the patient to TFC (see diagnosis of Acute Malnutrition).

Follow-up of the patient in SFC and discharge criteria:

Weight gain is the main indicator of evolution of the patient in SFC. If the patient does not gain weight or develops weakness or other signs and complications of malnutrition, evaluate for a possible referral to TFC or OTP. If the patient does not gain weight and there are no signs of malnutrition, evaluate the use of the food at home, and the appropriateness of the treatment given.

Consider discharge when the patient presents progressive weight gain and attains a BMI of 17.5 kg/m^2.

A note on SFCs: *Since the implementation of Home Treatment, the need and efficacy of SFC, particularly for adults, is being questioned. In some occasions, it may be preferable to ensure a General Food Ration for these patients (General Food Distribution or other). This will depend on the set-up of the project and previous results with SFC in the area. The appropriate use of SFC programmes for children and adults is currently being studied and debated. In the near future greater clarification will be available.*

Severe Acute Malnutrition – Therapeutic treatment:

The main procedures and protocols used for children are also valid for adults. The amounts of food and dosages are adapted to the different mass and energy requirements of adults.

See "Who to treat", in the precedent chapter, for advice on deciding need of Stabilisation phase in TFC and when to send the patient for Home Treatment.

TFC (in-patient treatment)

Phase I (Stabilisation centre):

F75 milk

Adolescents (12 to 17 years)	65 ml/kg/day (50 kcal/kg/day)
Adults (18 to 50 years)	55 ml/kg/day (40 kcal/kg/day)
Older persons (> 50 years)	45 ml/kg/day (35 kcal/kg/day)

Transition phase:

F100 milk

Use the same amounts as for F75 in Phase I. Ideally increase progressively to attain the quantities given in Phase II (use patient's appetite and tolerance as a guide).

Adolescents (12 to 17 years)	65 ml/kg/day (65 kcal/kg/day)
Adults (18 to 50 years)	55 ml/kg/day (55 kcal/kg/day)
Older persons (> 50 years)	45 ml/kg/day (45 kcal/kg/day)

Phase II:

F100 milk

Adolescents (12 to 17 years)	100 ml/kg/day (100 Kcal/kg/day).
Adults (18 to 50 years)	80 ml/kg/day (80 Kcal/kg/day).
Older persons (> 50 years)	70 ml/kg/day (70 Kcal/kg/day).

See ACF-guidelines for tables to calculate the amounts of milk by weight of the patient. The preparation of the milk, number of feeds per day, as well as the criteria to evolve from one phase to the next are as with the children.

As with children, the use of nasogastric tube should be exceptional. The dislike of milk is not a reason for force-feeding the patient. Alternatively, F100 may be replaced in some patients (with RUTFs), under the responsibility of the nurse in charge or the physician. In Phase II, propose "family plate" and "porridges" to diversify the diet, but ensure that this does not replace the therapeutic feeds.

Home treatment (therapeutic)

Home Treatment replaces Phase II of treatment in the TFC. The protocol is the same for patients that have followed previous stabilisation in the TFC as for those who have not. The patients are expected to visit an OTP once a week for evaluation of their status and distribution of treatment for the following week.

These tables summarize the amounts of RUTF to be distributed during the weekly visits (adapted from ACF Guidelines for the management of the severely malnourished).

Class of weight (kg)	PLUMPY'NUT®		BP100®	
	sachet per **day**	sachet per **week**	bars per **day**	bars per **week**
3.0 - 3.4	1 ¼	8	2	14
3.5 - 4.9	1 ½	10	2 ½	17 ½
5.0 – 6.9	2	15	4	28
7.0 – 9.9	3	20	5	35
10.0 - 14.9	4	30	7	49
15.0 – 19.9	5	35	9	63
20.0 – 29.9	6	40	10	70
30.0 - 39.9	7	50	12	84
40 - 60	8	55	14	98

Systematic Treatment in TFC:

Vit. A: 200 000 IU single dose (except pregnant patients or those of reproductive age)

Folic Acid: 5 mg single dose (as a loading dose. After that, the quantity in therapeutic milk will be enough to complete stores)

Amoxicillin: 60 mg/kg/day distributed in 3 doses per day, for 7 to 10 days.

Antimalarials: In malaria endemic zones, follow National Protocol for a curative treatment.

Mebendazole: 500 mg single dose. Only in Phase 2.

Specific treatment:

It is beyond the objective of these guidelines to present the diagnosis and treatment of all the medical conditions that can be seen in a TFC. A good summary can be found in the usual literature, including ACF-guidelines, WHO "Management of Severe Malnutrition" (WHO 1999) and Oxford Handbook of Tropical Medicine (Eddleston 2005).

It is important to stress that when treating any condition in the malnourished patient the dosage of drugs has to be calculated for the weight of the patient, to avoid overdose (remember that most recommendations of doses of drugs for adults are made assuming a weight of 60 kg !).

Some of the most common complications and treatment accidents in adults during therapeutic treatment are the same as with children. In order to control and avoid them, review ACF-guidelines, and in particular:

- Differential diagnosis of Dehydration and Severe Acute Malnutrition.
- Differential diagnosis of Septic Shock and Dehydration.
- Criteria for use of oral and IV fluids, and follow up of rehydration.
- Diagnosis of anaemia and differential diagnosis with haemodilution during treatment of malnutrition.
- Differential diagnosis of pulmonary oedema and respiratory infection.
- Diagnosis of infectious state from the start of treatment (paucity of symptoms), and use of systematic antibiotics.

Early mobilisation of the patient is important for mental and physical recovery. Patients should be encouraged to stay out of the wards during daytime, and if possible get involved in physical activities (from walking to active participation in the life of the centre).

Severe illness and complications during therapeutic treatment:

These are usually suspected at the first visit of the patient. If they are present, all other evaluation and diagnosis of the patient should be made after initial care has started.

- **Loss of consciousness:** Look for usual signs of coma and evaluate it. Consider hypoglycaemia and treat it, if necessary with an IV bolus (1 ml/kg of 50 % Glucose followed by 50 ml of 10 % glucose by nasogastric tube). Refer to physician. Initiate refeeding with F75 as soon as possible (via a nasogastric tube if necessary).

- **Extreme weakness:** Start refeeding with F75 as soon as possible, consider constant debit refeeding with F75 via nasogastric tube (under strict supervision!!) until the patient recovers consciousness and strength.

- **Septic Shock:** Cold hands or feet, weak pulse, decreased consciousness. Make differential diagnosis with dehydration. Treat hypoglycaemia and infection. Refeeding with F75 as soon as possible.

- **Severe dehydration:** Differential diagnosis with malnutrition and shock. Start rehydration with ReSoMal *per os* if possible, otherwise use IV fluids and follow up closely (attention, IV fluids are too often the cause of cardiac overload an pulmonary oedema in malnourished patients). If no positive evolution after 6 hours of rehydration, consider diagnosis of septic shock.

- **Hypothermia:** Consider severe infection and Septic Shock. Warm the patient through physical means (covers, etc.) and start antibiotics.

- **Patient in severe pain:** In the presence of severe abdominal pain see physician to discard a surgical complication before starting refeeding. Give regular oral or IV glucose to avoid hypoglycaemia.

- **Patient not able to swallow:** Analyze the cause (oral infection, candidiasis, vomiting, oesophagic problem, dyspnoea or other) and treat. Try nasogastric feeding with care.

- **Patient with severe vomiting:** Feed the patient regularly in small quantities. Diagnose the cause of vomiting and treat. If incoercible vomiting, try anti-emetics and observe reaction.

Special situations:

Refeeding oedema

A fast transition to a diet rich in sodium and proteins, like F100, plus the increase in intravascular fluid produced during the first stages of recovery of Acute Malnutrition can overwhelm the circulatory system. In some cases, this will result in pulmonary oedema and signs of cardiac overload. In others, this excess liquid can be placed in the interstitial space, producing oedema. This can be seen in TFC patients during Transition Phase and Phase II of treatment, and in patients treated at home. The patient can be at risk if liquids continue accumulating in the vascular system, producing cardiac overload and pulmonary oedema.

The treatment of re-feeding oedema is simple; it is usually enough to stop feeds for four to six hours and then resume F75 diet (meanwhile it is wise to provide regularly small amounts of water diluted sugar to avoid hypoglycaemia). If the patient presents with severe signs of cardiac congestion, do not start re-feeding until improvement of signs, even if this takes longer than 6 hours. In cardiac congestion and re-feeding oedema, if the patient does not improve with liquid restriction, one dose of diuretics can help (Furosemide), but is seldom needed, and also not always effective in the severely malnourished patient.

Once the volume of oedema starts to decrease the nutrition protocol has to be continued, with special care in the transition from F75 to F100: the increase in the amounts of liquid given to the patient has to be done progressively, rather than all at once, and the response of the patient monitored day by day. These patients are "high risk" and therefore need to be closely monitored by an experienced nurse or doctor.

Re-feeding glucose intolerance

This very rare condition is observed in patients following a fast re-feeding with glucose products. It usually appears in patients that do not present with any other problems and particularly in some with a good appetite, during Phase II. The clinical picture is that of a severe diabetes mellitus, with polyuria, polydipsia and polyphagia. The patient does not gain weight despite eating enough food. Analysis of urine is intensively positive to glucose. In some cases, the clinical picture can start directly with diabetic coma, usually ketoacidosic.

No specific treatment is described in the literature. Normally it should be controlled with Insulin or a relative reduction of glucose in the diet (this can be achieved with reconstituted F100, from DSM). Once the situation of the patient is stabilised, the doses of Insulin can be reduced, until the patient can be discharged with no specific treatment. For severe cases in ketoacidotic coma, management may require intensive medical treatment (Insulin, potassium, rehydration) and a laboratory to monitor evolution and adjust treatment (these are rarely available in the context of TFCs). Most centres will never see a case of re-feeding glucose intolerance. If one is suspected it is advised to contact headquarters to search for appropriate advice with experts.

All re-feeding problems are more likely to occur in patients that have not eaten in the days before the admission.

"New infections" during recovery:

As with children with severe Acute Malnutrition, the recovery of the immune and inflammatory functions may result in the expression of diseases that were pre-existing, but unnoticed. This can go from the expression of the symptoms of malaria acquired before admission to the centre to the development of symptoms of tuberculosis. In these cases, it is not necessarily the consequence of contamination in the centre. In all cases, treat as necessary, including referral of the patient to hospital or other specific services, while continuing the nutrition protocol.

Monitoring the patient:

As for children, the follow-up of the patient is based in the evolution of weight and the regression of oedema. Oedema usually starts disappearing after three-four days under treatment. Average weight gains above 5 g/kg/day are acceptable, though they are usually much higher. If the patient does not evolve in this way, consider the quality of the feeds, their frequency and acceptability and make a complete medical examination to discard the presence of other associated pathology.

General attitude, presence of apathy and muscular strength are very valuable indicators of the progression of the patient. They should not be ignored.

Fast regression of oedema is usually a sign of good evolution of the patient. It could also be an indication that the patient had re-feeding oedema, rather than oedema of malnutrition. In such cases, discharge the patient after the successful introduction of F100, with recommendation to avoid foods rich in salt or proteins for some days.

The follow-up of MUAC can be useful, as a cross-check measure with weight. Weight can increase because of fluid accumulation, in the form of oedema or in the lungs, liver or other cavities. In these cases, the disparity between fast weight increase with no MUAC change, together with other associated symptoms, should alert the clinician to proceed to a complete evaluation of the patient[23].

Other clinical observations may help understand the evolution of the patient. During the first days of treatment, the excess of fluids in circulation coupled with the incapacity of the kidneys to concentrate urine and regulate diuresis usually results in important polyuria and nocturia. The return to a normal circulating volume and complete renal activity will be marked by the regression of these symptoms, particularly the nocturia, after some days in Phase II. This may have been of use in the

[23] At present there are no agreed recommendations of what is a « desirable » MUAC change during treatment of Acute Malnutrition. The clinician should be alerted when weight increases despite no change in MUAC (or MUAC loss) during at least one week. In these cases, a complete clinical evaluation of the patient should investigate the possibility of failure of treatment or fluid accumulation. However, a fast weight increase (several kilograms in few days) should always be suspected as fluid accumulation whatever the MUAC of the patient, and associated symptoms of fluid overload searched for.

diagnosis of other conditions: for example, if signs of anaemia appear before the phase of polyuria it is possible that we are observing haemodilution, rather than a true anaemia (though haemodilution can also be observed in the absence of other signs of hypervolemia).

When a patient is transferred to hospital, the TFC team must not only ensure the regular feeding of the patient following appropriate protocols, but also the nutritional and medical follow-up, in coordination with the hospital staff. The complications described in this chapter may be unknown to the hospital staff. Good communication between the two services is key to improve the survival of most complicated cases.

The recovery of sexual function in patients that had developed impotence, in men, and the return of menstruation after amenorrhea in women, together with the return of libido are conclusive signs of nutritional and health recovery. Ideally (for all), they should happen when the patient is at home.

Evolution of treatment:

In general, adult patients respond well to treatment in TFC. The main problem with treatment is the usual refusal of the patient to take a diet exclusively based on milk. The recovery of appetite is often spectacular, with very large amounts of food being taken at every meal after just a few days in the centre. Ideally, all patients should be sent to Home Treatment (OTP) as soon as they recover some strength and appetite. For patients that cannot be sent home, due to lack of coverage of the programme or for other reasons, treatment can be continued in the TFC with RUTF, or a combination of RUTF and therapeutic milk. Other foods (family plate) can be added since the recovery of appetite, but it is important to give priority to the nutrient rich meals of Therapeutic milk or RUTF to avoid that appetite is satisfied exclusively with the less nutrient dense foods.

This recommendation should also be made for patients who follow treatment at home. In particular, some patients may be tempted by salt and protein rich meals at home, and alcohol (e.g. local beer or other fermented drinks). All of these should be avoided during the first weeks of treatment at home, to avoid the risks of re-feeding oedema (see box) and other possible accidents.

Often, adult patients that do not respond to treatment in TFC are suspected of presenting with underlying HIV. This attitude is unprofessional: it should be eradicated from TFCs. Severe Acute Malnutrition can present with all the same symptoms of HIV/AIDS (due to immune deficiency of nutritional origin), and they cannot be differentiated by symptomatic means. The lack of response to treatment is not a good guide to suspect HIV, as:

- HIV positive patients can have a good evolution of treatment
- Many patients with a poor response can be diagnosed with other conditions, different from HIV, which can be easily treated in a TFC or Hospital.

The only way to confirm HIV diagnosis is via laboratory test confirming the serology of the patient. This should guide the need for treatment with anti-retroviral, where available. Otherwise, the nutritional treatment is the same regardless of HIV status.

Discharge criteria:

All patients should be kept on treatment (at TFC or in Home Treatment) until they reach:

- BMI of 17.5 kg/m^2 (16.5 in older patients), and
- no oedema.

However, the patient can be considered recovered once he/she shows a steady weight gain (at least three weeks at home), recovery of physical strength and all the associated acute diseases are under control.

If the circumstances that produced the malnutrition are still present (lack of regular access to food, economic problems or a specific underlying disease), the patient should be invited to follow-up after several weeks to reassess his/her status (through SFC, for example). Consider referring the patient towards the appropriate programmes, including medical, Food Security, social or other.

REPORTING OF SFC AND TFC PROGRAMMES:

Always keep separate statistics for adults. In the reporting and statistics, do not mix these patients with pregnant and lactating women: they represent different problems and should be reported separately.

Weight gain and recovery rates equivalent to those obtained with children are usually observed in TFC, they are the reference standards by which to evaluate the quality of the programme.

Ideally, it would be interesting to keep track of the main causes of malnutrition (at least the percentages of suspected primary and secondary malnutrition, and the relative frequency of Stable Malnutrition in relation to Acute Malnutrition) as this may have great importance in the design and adaptation of the strategy of intervention (see following section).

Follow up of the patients must be done, including those that were treated in hospital. Remember that a death that takes place in hospital should be registered as "death", not as "transfer" as long as the patient is still in TFC treatment, and followed up by the TFC, independently of the cause of the death.

The results of adult treatment in the centres may be a guide to help assess the quality of diagnosis and decide on the strategies best adapted to the situation. If the rates of recovery are remarkably better than those obtained for children in the same centre, one could question if there are not too many patients with Stable Malnutrition or Moderate Malnutrition being admitted to the centres. If, on the contrary, most patients have negative outcomes, or are transferred to hospital, one could consider if the main causes of Acute Malnutrition are chronic or acute diseases, and the programme could be adapted to address them (see following section).

VIII. Programme strategies:

The type of intervention needed to address Acute Malnutrition in adults may be adapted to the main causes of malnutrition in a particular crisis. If most cases of Acute Malnutrition are secondary to lack of food (primary malnutrition) then an approach based on the set up of Supplementary and Therapeutic Feeding Centres, with Home Treatment, is the most appropriate. However, if most cases of Acute Malnutrition were secondary to HIV/AIDS, tuberculosis or other chronic disease (Kala-Azar, Schistosomiasis, etc.) then the appropriate strategy for the control and treatment of those diseases would be more appropriate, with a nutritional component that can include therapeutic feeding and other food supports.

Indeed, the relative frequency of primary and secondary malnutrition may vary over time during the same crisis. More often, at the beginning of a severe food crisis, most cases of Acute Malnutrition will be primary. As the situation improves, the percentage of cases secondary to diseases will seem to increase. If the relative frequency of primary and secondary cases is monitored continuously, this may allow the appropriate adaptations to the programme strategy, and help decide on the phasing out of the centres and hand over to other partners.

In practical terms, when a high prevalence of secondary malnutrition is expected, the set up of the programme will need to be adapted to it. This includes the recruitment of a physician (local or expatriate) and nurses with experience in the management of medical problems and the provision of medicines necessary to treat the associated conditions and prevalent diseases (in addition to the standard TFC pharmacy). Special advice and training may be needed as well, but this will be different depending on the endemic profile of the area and means available[24].

Usually there is no need to set up specific nutrition programmes for adults: They can be integrated in the same centres with children. However, in crisis affecting big numbers of individuals, where several centres for the treatment of malnutrition are open, specific centres for adults may be considered.

[24] Therefore, the initial evaluation of the situation should consider the need to request expert advice for the set up of the programme, specific training and support, etc. This can take the form of a permanent presence of an expert in the centre, or specific training and supervision visits.

IX. Population assessment of malnutrition in adults:

Assessments of nutritional status in adults are not routinely made during humanitarian crises. The assessment of the status of children (mainly through cluster surveys) is supposed to represent the overall status of the population. This implies that if there is a high rate of malnutrition among children one should ALWAYS wonder if the situation is the same for adults or if they are not affected for the moment.

A group of experts met in Nairobi in 2001 to discuss the use of assessment of adult malnutrition in emergencies. They agreed that it is appropriate to consider assessing adult malnutrition in addition to children's malnutrition in specific circumstances. They proposed the following examples:

- If the crude mortality rates begin to approximate or surpass the under-five mortality rates, suggesting that the over-five population is as vulnerable as the under-five population.
- If the prevalence of malnutrition is very high in the under-fives and is not due to a health problem mainly affecting that age group.
- If there is reasonable doubt that the nutritional status of children does not reflect the adult nutritional situation, for example in Bosnia or Kosovo it was suspected that older people were particularly vulnerable to malnutrition.
- If many adults attempt to enrol in selective feeding programmes or present to health posts.
- If anecdotal reports of adult malnutrition are received.
- If there is low coverage of food aid in dependant populations.
- If data is required as an advocacy tool to lever resources.

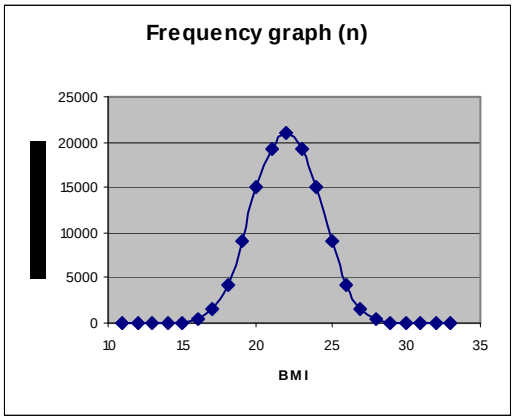
The sampling methods recommended for surveys of adult malnutrition are the same as those used for surveys of child malnutrition. The same sampling scheme can therefore be used (see specific guidelines). A separate survey should be done for children and for adults. If the data collection for the two surveys is done at the same time, it is important to measure all adults living in the household, even if there are no children living there. Adults are more often absent from their homes than children, particularly during working hours and during the week. In order to avoid selection bias it is very important to inform the population beforehand and ensure that all adult members of the household will be present the day of the survey. Most often, adults absent from home at the time of the surveys are healthy men, and therefore the survey will tend to overestimate the prevalence of malnutrition in adults (but check if this is the case in your survey!).

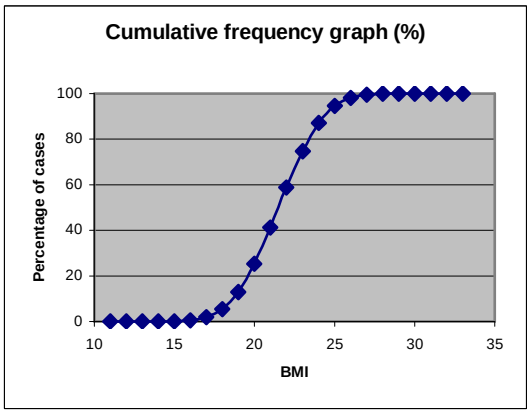
The target population of adult surveys includes individuals above the age of 18 of both sexes. Pregnant women should be excluded. Depending on the objectives of the survey and the information collected beforehand, one can decide to include older

persons, exclude them, or even to carry out a survey exclusively addressed to this group.

Surveys of the nutritional status of adults should measure at least BMI (weight and height), MUAC and bilateral oedema, following the recommendations given in the previous chapters.

As the value of specific cut-off points of these indicators is subject to discussion, the data should be reported as continuous data (at least mean, standard deviation and/or quartiles) and a graph of the cumulative frequencies of each indicator presented in the report. For example, the same data collected in a survey of adult BMI is presented in the two graphs. The first version (left) presents the usual curve of frequency of cases. The second (right) presents the same information in cumulative frequencies (percentages). In order to build this graph, the information collected has been added to calculate the cumulative frequency, and this was transformed into percentages. Both graphs were done in Excel using the "Scatter point with lines" type of graph.





It is easier to estimate the number of patients under any cut-off point of BMI in the second graph. Simply identify the cut-off point of interest in the x-axes and report it to the line, the y-axes will give the percentage of the population surveyed having this BMI or lower.

As explained in the previous chapters, neither BMI nor MUAC can give a complete picture of the rates of Acute Malnutrition in a population. In order to understand the true nature of the problem we need to be able to know the relative frequency of patients with actual Acute Malnutrition (actively loosing weight) and those with Stable Malnutrition, among patients with a low BMI. There is no simple technique to collect this information during a survey. For this reason, in order to be able to interpreter the results of the survey we need to obtain additional information on the baseline rates of diseases that could produce malnutrition (HIV/AIDS, tuberculosis), a clear picture of the food security situation, the likelihood of food shortages at the household level, previous rates of low BMI in the same population, as well as other

social and family factors such as composition families and movements of population. In addition, one can select a sub sample of individuals with low BMI in the survey to ascertain if they present with Acute Malnutrition or Stable Malnutrition (following diagnostic guidelines above), and extrapolate to the whole sample.

Due to phenotypic differences, the BMI of different populations cannot be directly compared. In order to do that it needs to be adjusted for Cormic Index. This requires the measurement of standing height and sitting height for each individual in the sample. See box for explanations.

Adjusting BMI for Cormic Index (copied from Collins *et al* 2000a):

In order to standardise BMI to take into account changes in SH/S ratio (Sitting height to standing height ratio) we recommend using the equations below to calculate BMI standardised to the actual SH/S for the population under study

Male subjects: BMI = 0.78 (SH/S)-18.43
Female subjects: BMI = 1.19 (SH/S)-40.34

SH/S ratios should be expressed as a percentage.

The observed BMIs can then be standardised to a SH/S ratio of 0.52 by adding the differences between the observed BMI and BMI standardised for the population SH/S to a BMI standardised to 0.52 using the equation below:

BMIstd = BMI0.52 + (BMIob – BMIes)

Where: BMIstd = standardised BMI,
BMI0.52 = estimated BMI as SH/S of 0.52
BMIob = actual BMI
BMIes = estimated BMI at actual SH/S

Examples:

1. A Male population "A" has a mean BMI of 18.5 kg/m^2 and a mean SH/S ratio of 50 %. The BMI0.52 is 0.78*52-18.43 = 22.13. The BMIes = 0.78*50-18.43 = 20.57. Therefore the BMIstd = 22.13 + (18.5 – 20.57) =20.06 kg/m^2
2. A Female population "A" has a mean BMI of 17.0 kg/m^2 and a mean SH/S ratio of 54 %. The BMI0.52 = 1.19*52-40.34 = 23.92. The BMIes = 1.19*54-40.34 =21.54. Therefore the BMIstd = 21.54 + (17.0 – 23.92) = 14.62 kg/m^2

Always contact headquarters for interpretation and comparison of BMI surveys!

X. Conclusion.

The treatment of malnutrition in adults is no more difficult than that of children. The problems found with it in the past are related to the inadequacy of guidelines, in particular for the diagnosis of Acute Malnutrition. These guidelines have presented a new approach for this task, based in part on the clinical presentation of the patient and the history of weight loss and weakness.

The guidelines presented here are not definitive. The main recommendations presented are still under discussion and investigation, including the best way to identify patients in need of treatment, new adaptations to the treatment, the diagnosis and treatment of patients with an underlying chronic disease such as HIV/AIDS or Tuberculosis, and the assessment of malnutrition in populations. Some promising techniques have been suggested that could facilitate diagnoses and treatment of malnutrition in the field such as the evaluation of handgrip strength. In addition, the increased access to tests to diagnose HIV in the field and guide treatment should provide further support for the correct management of patients affected, malnourished or not.

Most of the advances presented in these guidelines are the fruit of questioning from the field and investigations made in the midst of emergency interventions in ACF. Permanent criticism and systematic lesson learning from the field, together with imaginative thinking and commitment to make things move forward, should contribute to the improvement of these guidelines in the future, to the benefit of the individuals suffering from malnutrition and the societies where they live.

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Appendix 1. Subjective Global Assessment (SGA) of nutritional status.

A. History.

1. Weight change and height:
   Current height: _____ cm. Weight: _____ kg.
   Overall loss in the last 6 months: _____ kg; _____ %
   Change in the last 2 weeks (use + or -): _____kg; _____ %
2. Dietary intake change (relative to usual intake):
   No Change
   Change duration : _____ days
   Type: Suboptimal solid diet
   Hypocaloric liquids
   Starvation
   Supplement : (circle) nil, vitamins, minerals
3. Gastrointestinal symptoms persisting >2 weeks.
   None
   Nausea
   Vomiting
   Diarrhoea
   Pain At rest On eating
4. Functional capacity:
   No dysfunction
   Dysfunction duration _____ days
   Type: Working suboptimally
   Ambulatory but not working
   Bedridden
5. Disease and its relation to nutritional requirements:
   Primary diagnosis: _____
   Metabolic demand (stress):
   No stress
   Moderate stress
   High stress (burns, sepsis, severe trauma).

B. Physical status (for each trait specify: 0 = normal, 1 = mild deficit, 2 = established deficit).
   Loss of subcutaneous fat
   Muscle wasting
   Oedema
   Ascites
   Mucosal lesions
   Cutaneous and hair changes

SGA GRADE: _____

Comments on the implementation of the SGA of nutrition status:

Body weight may change if there is accumulation of fluids, like in oedema.

Dietary intake is subjectively assessed. It only needs to be evaluated if current diet is adequate.

Presence of gastrointestinal symptoms is considered as a potential cause of nutrient loss, not as symptoms of malnutrition itself.

Patient's functional capacity is defined by the person making the assessment and the subjective impression of the patient.

"Metabolic stress" makes reference to the underlying disease and is evaluated by the person performing the assessment.

The features of physical examination are evaluated as normal, mild, moderate or severe. The loss of subcutaneous fat is measured in the tricipital region and the midaxilary line at the level of the lower ribs (subjective impression). Muscle wasting is evaluated in the temporal areas, deltoids and quadriceps (loss of mass and tone detected by palpation –but can be modified by neurological deficits).

The presence of oedema is assessed in the ankle and sacral regions. This can be modified by coexisting diseases (renal or congestive heart failure, for example).

Mucosal and cutaneous lesions are noted, as well as the colour and appearance of hair.

With the findings of the history and physical examination the patient is categorised as being well-nourished (category A), having moderate or suspected malnutrition (category B) or having severe malnutrition (category C). The rank is assigned by subjective weighting.

Appendix 2. Measurement of Height: Estimating the height of a person who cannot stand up straight (Ismail, Manandhar, 2000)

"Half span" measurement is used to estimate height according to the following calculation:

Height (m) = 0.73 x (2 x the half-span [m]) + 0.43

Measuring the half-span

1. Ask the subject to stand, with his back pressed against a support (wall, door).
2. Place a finger on the middle of the person's chin and slide it down the front of his throat until it meets a U-shaped bone at the base of the neck (the top of the sternum, between the two clavicles). This is the departure point for the measurement.
3. Ask the subject to extend the left arm, with the palm of the hand facing forward. The elbow, wrist and hand must all be held out straight. Support the arm if necessary.
4. Put the zero end of the measuring tape at the farthest extremity of the arm (the end of the middle finger of the outstretched arm) and hold it there.
5. Stretch the tape along the arm until it reaches the point established in (2).
6. Note this measurement to the nearest 5 mm. Write down the measurement and ask the subject to lower the arm.
7. Measure the same arm once more.
8. If the difference between the two measurements is less than 0.5 cm, work out the average of the two measurements. If it is greater than 0.5 cm, repeat the measurements again.

Appendix 3. Measurement of Weight:

Recommendations for using vertical scales:

- Place the scale on a horizontal flat surface (if the measurements are "suspicious" try placing the scale in a different place, ensuring it is completely flat).
- Minimize the displacement of the scales, even within the centre (this affects the scales and will result in inaccuracies).
- Check the accuracy of the scale often, against a known weight.
- Crosscheck any "strange" readings with a second scale, or with the same scale in a different place.
- Get new scales if the ones you are using are unreliable.

Measuring weight in patients that cannot stand:

Patients that are unable to stand can be weighed using a hanging scale of 50 Kg (similar to that used for children, but with a larger range). These can be obtained from the same distributors.

To measure the patient, hang a resistant hammock from the hook of the scale and sit the patient in it carefully.

Re-calculation of weight in amputated patients:

In order to calculate the correct BMI, the weight of the patient who has major amputations can be re-calculated following this guide:

Estimated weight of missing limbs:

Upper limb: 4.9 % of total weight (upper arm 2.7%; forearm, 1.6%; hand, 0.6%).
Lower limb: 15.6% of total weight (thigh 9.7%; lower leg 4.5%; foot 1.4%).

Recalculate weight adding the estimated weight of the missing limb. Calculate BMI with this weight in order to decide if the patient is thin. Follow the same procedure backwards to calculate the target weight. From this point, weight the patient normally to follow up evolution until target weight.

Appendix 4. Table of BMI from Height and Weight.

Height	Body Mass index					
(cm)	18.5	18	17.5	17	16.5	16
140	36.3	35.3	34.3	33.3	32.3	31.4
141	36.8	35.8	34.8	33.8	32.8	31.8
142	37.3	36.3	35.3	34.3	33.3	32.3
143	37.8	36.8	35.8	34.8	33.7	32.7
144	38.4	37.3	36.3	35.3	34.2	33.2
145	38.9	37.8	36.8	35.7	34.7	33.6
146	39.4	38.4	37.3	36.2	35.2	34.1
147	40.0	38.9	37.8	36.7	35.7	34.6
148	40.5	39.4	38.3	37.2	36.1	35.0
149	41.1	40.0	38.9	37.7	36.6	35.5
150	41.6	40.5	39.4	38.3	37.1	36.0
151	42.2	41.0	39.9	38.8	37.6	36.5
152	42.7	41.6	40.4	39.3	38.1	37.0
153	43.3	42.1	41.0	39.8	38.6	37.5
154	43.9	42.7	41.5	40.3	39.1	37.9
155	44.4	43.2	42.0	40.8	39.6	38.4
156	45.0	43.8	42.6	41.4	40.2	38.9
157	45.6	44.4	43.1	41.9	40.7	39.4
158	46.2	44.9	43.7	42.4	41.2	39.9
159	46.8	45.5	44.2	43.0	41.7	40.4
160	47.4	46.1	44.8	43.5	42.2	41.0
161	48.0	46.7	45.4	44.1	42.8	41.5
162	48.6	47.2	45.9	44.6	43.3	42.0
163	49.2	47.8	46.5	45.2	43.8	42.5
164	49.8	48.4	47.1	45.7	44.4	43.0

Height	Body Mass Index					
(cm)	18.5	18	17.5	17	16.5	16
165	50.4	49.0	47.6	46.3	44.9	43.6
166	51.0	49.6	48.2	46.8	45.5	44.1
167	51.6	50.2	48.8	47.4	46.0	44.6
168	52.2	50.8	49.4	48.0	46.6	45.2
169	52.8	51.4	50.0	48.6	47.1	45.7
170	53.5	52.0	50.6	49.1	47.7	46.2
171	54.1	52.6	51.2	49.7	48.2	46.8
172	54.7	53.3	51.8	50.3	48.8	47.3
173	55.4	53.9	52.4	50.9	49.4	47.9
174	56.0	54.5	53.0	51.5	50.0	48.4
175	56.7	55.1	53.6	52.1	50.5	49.0
176	57.3	55.8	54.2	52.7	51.1	49.6
177	58.0	56.4	54.8	53.3	51.7	50.1
178	58.6	57.0	55.4	53.9	52.3	50.7
179	59.3	57.7	56.1	54.5	52.9	51.3
180	59.9	58.3	56.7	55.1	53.5	51.8
181	60.6	59.0	57.3	55.7	54.1	52.4
182	61.3	59.6	58.0	56.3	54.7	53.0
183	62.0	60.3	58.6	56.9	55.3	53.6
184	62.6	60.9	59.2	57.6	55.9	54.2
185	63.3	61.6	59.9	58.2	56.5	54.8
186	64.0	62.3	60.5	58.8	57.1	55.4
187	64.7	62.9	61.2	59.4	57.7	56.0
188	65.4	63.6	61.9	60.1	58.3	56.6
189	66.1	64.3	62.5	60.7	58.9	57.2
190	66.8	65.0	63.2	61.4	59.6	57.8

Appendix 5. Differential diagnosis of Bilateral Oedema.

Causes of oedema that share characteristics with malnutrition oedema:

Cardiac Oedema: Congestive Heart Failure. -

Produced by:
Cardiac insufficiency of miocardial or valvular origin, particularly when it affects the right ventricles. It can also be produced by constrictive pericarditis.

Characteristics of oedema:
Oedema of cardiac origin presents as bilateral pitting oedema. It is dependent (initially in lower limbs and then ascending) and tends to increase as the day progresses and decrease during the night (fluctuating). It can appear in different locations, depending on the posture that the patient adopts (recumbent oedema). The importance of oedema is usually related to the level of cardiac insufficiency. It can attain ascites and anasarca. The skin over the oedema area is soft, shiny and not hot. In chronic cases, it may appear cyanotic (though this is difficult to see in black skin). Oedema that appears only in the morning after standing up for a long period and that is reabsorbed rapidly after mobilisation of the patient is probably of cardiac origin, not nutritional.

Symptoms and history of the condition:
In chronic (right) cardiac failure, the patient presents with dyspnoea that increases with effort. The history of dyspnoea usually precedes that of oedema of several weeks/months. The amount of oedema may have increased and decreased over time. The patient usually has a previous history of cardiac disease.

Cardiac failure is evidenced by the presence of dyspnoea, tachypnoea, basal crackles, painful hepatomegaly, positive hepatojugular reflux, and signs of chronic vascular stasis (varicose veins in lower limbs). Heart sounds are reduced, but a gallop rhythm can be audible (tachycardia), as well as systolic and diastolic murmurs. Central venous pressure is increased (jugular column) and arterial pressure reduced. It evolves to Acute Pulmonary Oedema.

In acute heart failure, there is an important loss of cardiac output and dyspnoea progresses rapidly, with tachycardia, possible thoracic pain, cyanosis, hypotension and cold sweats. The patient is usually agitated. Heart can be enlarged (tip of the heart may be palpable). Acute heart failure is an emergency, as it usually evolves towards pulmonary oedema.

Suspect cardiac origin…
… in the presence of bilateral, dependent, fluctuant pitting oedema when the patient has a history of cardiac disease, the oedema is not associated to hair or skin lesions, and if it is associated to dyspnoea and heart symptoms (other than extrasystols, that are typical of the electrolyte disturbances of malnutrition –low potassium-), particularly if they preceded the development of oedema.

However, Severe Acute Malnutrition may produce cardiac failure, particularly during treatment with fluids. In this case, the history of previous cardiac symptoms and the order of apparition of oedema and dyspnoea will guide the differential diagnosis.

If both chronic cardiac insufficiency and malnutrition are present, start treatment in TFC and contact the physician. If a patient with chronic cardiac insufficiency is admitted to TFC, oedema may regress thanks to the low sodium content of the diet (F75), but there will be a risk of precipitating Congestive Cardiac Failure due to the amount of liquids administered. In case of doubt, supervise the patient closely and limit fluids.

Oedema of Renal origin: Nephrotic Syndrome and Nephritic Syndrome. -

Produced by:
Glomerulonephritis, Acute nephritis, and other causes of renal failure.

Characteristics of oedema:
Nephrotic syndrome presents with significant bilateral pitting oedema. It usually has a fast progression, and is very extended and profound. The pit left by thumb pressure is profound and plastic (it can be "modelled"). The oedema starts in lax tissue, often in periorbital and perimaleolar areas (around the ankles). The skin of the oedematous area is shiny and not hot. In severe cases, it can affect cavities (ascites, hydrothorax, etc.). For its characteristics, it can be easily confounded with malnutrition oedema.
The oedema of Nephritic Syndrome is usually slower to set up. It covers a smaller region. It usually also starts in soft areas, and needs to be suspected if oedema started in periorbital area.

Symptoms and history of the condition:
The oedema of Nephrotic syndrome is associated to important proteinuria. That of Nephritic syndrome may present, in addition, haematuria and arterial hypertension. If it is the consequence of acute nephritis, there may be a history of streptococci infection in the three weeks preceding it. Both can present with anuria or polyuria as well as other renal symptoms.

Suspect renal origin…
… in any oedema that started first in periorbital area or other lax tissue. The presence of massive proteinuria (detectable with dip-sticks) supports the diagnosis, but moderate proteinuria can be present in oedematous malnutrition. Anuria and polyuria support the diagnosis as well, but the can be present in malnutrition as well (the second most often once treatment has been started). Oedema of renal origin (particular nephrotic syndrome) and that of oedematous malnutrition are difficult to distinguish. The presence of related symptoms of renal disease and the absence of symptoms of malnutrition, together with the epidemiological context, are the keys for the differential diagnosis. If a laboratory is available, it can provide definitive diagnosis.

Hepatic origin: Cirrhosis and Portal Hypertension. -

Produced by:
Chronic hepatic failure with portal hypertension. Acute hepatic failure (hepatitis, acute intoxication from drugs or other cause). Cirrhosis is usually present (alcoholic, toxic, cancer or other). Hepatic schistosomiasis (Bilharzia) in endemic areas.

Characteristics of oedema:
Bilateral pitting oedema. Usually associated with significant ascites. Dependent and recumbent, it never affects levels above the liver (no periorbital or upper limbs areas affected).

Symptoms and history of the condition:
Chronic hepatic failure has a long and insidious evolution. At the time of the development of oedema, significant ascites is present, with signs of portal hypertension (collateral circulation in abdomen, spider angiomas, etc.). Hepatomegalia of cirrhotic characteristics (when it can be explored) is usually present. If jaundice is present, it contributes decisively to the differential diagnosis, but it can be absent (e.g. schistosomiasis). Chronic hepatic failure can evolve to concentration problems, drowsiness, confusion, and coma. Fetor hepaticus (bad breath) and flapping tremor are common signs of severe liver damage.

Suspect hepatic origin…
… in the presence of typical hepatic signs and the absence of signs of malnutrition. A history of hepatic disease and presence of risk factors (alcoholism, living in endemic area of schistosomiasis – particularly if the patient is usually exposed to infested water-, consumption of toxic drugs or herbs, etc.) increases the suspicion of hepatic disease.

The presence of ascites is not enough to decide on the hepatic origin of oedema, as it is often present in adult malnutrition as well. The analysis of ascites liquid is not always helpful: in malnutrition, it will present the characteristics of a transudate (like in cirrhosis, nephrotic syndrome or congestive heart failure). Malnutrition cannot be excluded if it presented as an exudate, as this could be the consequence of an infected ascites liquid (infected after malnutrition was already present) or be a sign of malignancy (that could have produced malnutrition).

Oedema of Venous origin:

Produced by:
Chronic venous insufficiency, venous thrombosis, etc.

Characteristics of oedema:
Oedema can be unilateral or bilateral pitting oedema, depending on the areas affected. It can be localised to a specific area. Skin above the oedema may be pale or blue (cyanotic). It usually develops over a long time, with progressive swelling after standing up or walking for some time. In the initial stages it can disappear after mobilisation of the limb ("muscular pump").

Symptoms and history of the condition:
Possible signs of phlebitis, local cyanosis, collateral veins and varices (venous distension). The patient may also report pain when walking that alleviates when the legs are elevated.

Suspect venous origin…
…in the absence of signs of systemic disease and through the history of the disease and the characteristics of the oedema.

Other situations where bilateral oedema can be observed:

The list of conditions that can present with generalised oedema or bilateral oedema of lower limbs is long. This section presents some of those that can present to a nutrition centre and may require a differential diagnosis with Oedematous Malnutrition. The differential diagnosis is usually easy once considered. None of them requires nutritional support *per se*. If identified, they should be referred to the appropriate health services.

Pregnancy: Liquid retention can be limited to legs, or generalised. Usually present in the last phases of pregnancy. It disappears after delivery. Pre-eclampsia is a serious complication of pregnancy that appears in the third trimester with generalised oedema and hypertension.

Obesity, heat, "walking oedema": these situations can produce a moderate leg swelling that can be confounded with malnutrition oedema. In each case, it is associated to difficulties to venous return, with no associated symptoms. It usually regresses after a short rest.

Cushing syndrome: Chronic glucocorticoid excess easily recognisable for the aspect of the patient. Not usually confounded with malnutrition.

Hypothyroidism (Mixoedema): Chronic lack of thyroid hormone. Long and insidious evolution. Oedema is pretibial and is associated to periorbital puffiness. Other symptoms and signs of hypothyroidism should be present to suspect this diagnosis (asthenia; sensitivity to cold; dry, pale and cold skin; constipation; hypothermia at evolved state; obesity; etc.).

"Wet" Beri-beri (thiamine deficiency): Right heart failure with high cardiac output. Courses with oedema and oliguria. Signs of hypercirculation (including hot limbs and typical pulse and cardiac auscultation). Can be associated to "Dry" Beri-beri (typical neuropathy). Not frequent, except during outbreaks and in chronic alcoholism.

Hypoproteinemia: Some rare diseases producing loss of proteins. The oedema is similar to that of malnutrition or nephrotic syndromes, but without the associated symptoms.

Inflammatory oedema: Not usually bilateral. It presents anywhere in the skin with signs of inflammation (localised erythema, pain, a possible infectious focus, ganglions and lymphangitis).

Generalised Allergic reaction: Sudden start, after exposure to the allergen. Important swelling. Affected areas are red and hot. Often in face. Special case: Quincke's Oedema. It is a medical emergency: if identified, it should be treated on the spot by a physician and referred to hospital as soon as possible

Types of oedema that do not have the same characteristics of malnutrition oedema:

Unilateral oedema: usually due to obstruction of venous or lymphatic drainage of one limb. When it is more localised it can be due to soft tissue infection, and in these cases the other signs of inflammation will be present. Venous oedema may be associated with cyanosis of the leg.

Asymmetrical oedema: Same causes than unilateral oedema, if the obstruction is bilateral. May be a result of unilateral oedema superimposed in another cause, including malnutrition.

Non-pitting oedema: Usually due to lymphatic retention, it produces a "hard" oedema. Observed particularly in areas where people walk barefoot, and where filariasis is prevalent (elephantiasis). Lymphatic cancers can produce it as well, but they are less frequent. Other types of oedema can have a "hard" appearance if they have a long duration, but this is exceptional in malnutrition oedema.

Appendix 6. Typical pharmacy requirements for a TFC treating adults.

This kit is destined for the treatment of 100 adults in TFC for 3 months or more

NAME	Needs in unit	Unit	Packaging	Price/packaging in Euro	Needs/package	T
ORAL						
Acetylsalicylic acid 500 mg	1000 tabs		box of 1000 tabs	2.45	1	
Aluminium hydroxide 500 mg	1000 tabs		box of 1000 tabs	2.6	1	
Amoxycillin 500 mg	3000 tabs		box of 1000 tabs	21	3	
Butylscopolamine bromide 10mg coated	1000 tabs		box of 1000 tabs	10.2	1	
Ciprofloxacin 100 mg	1000 tabs		box of 100 tabs	2.05	10	
Chloramphenicol 250 mg	1000 tabs		box of 1000 tabs	11.9	1	
Cloxacillin 500 mg	1000 tabs		box of 1000 tabs	21.45	1	
Cotrimoxazole 480 mg	1000 tabs		box of 1000 tabs	7.65	1	
Ibuprofen 200 mg	1000 tabs		box of 1000 tabs	3.3	1	
Mebendazole 100 mg	1000 tabs		box of 1000 tabs	10	1	
Metronidazole 250 mg	1000 tabs		box of 1000 tabs	3.5	1	
Nystatin 500,000 IU	300 tabs		box of 100 tabs	5.75	3	
Paracetamol 500 mg	1000 tabs		box of 1000 tabs	3.1	1	
Phenoxypenicillin 250 mg	1000 tabs		box of 1000 tabs	9.6	1	
Ferrous sulphate 200 mg	5000 tabs		box of 1000 tabs	1.1	5	
Quinine 300 mg	1000 tabs		box of 1000 tabs	23.55	1	
INJECTABLES						
Ampicillin 1 g	100 vials		box of 50 vials	4.65	2	
Chloramphenicol 1 g	100 vials		box of 50 vials	9.4	2	
Ceftriaxone	30 vials		box of 10 vials	9.15	3	
Water for injectable preparations 5 ml	400 vials		box of 50 vials	1.2	8	
Furosemide 40 mg	10 vials		box of 10 vials	4.85	1	
Quinine 600 mg./ 2 ml	100 vials		box of 100 vials	12.1	1	
DRIPS						

Dextrose 5% in water, 500 ml without set	20 bags		box of 20 bags	14.7	1	
Ringer-Lactate solution, 500 ml without set	20 bags		box of 20 bags	9.2	1	
RENEWABLE EQUIPMENT						
Needle 19 g intramuscular	100		box of 100 pcs	1.2	1	
Needle 21 g intramuscular	300		box of 100 pcs	1.2	3	
Needle 18 g preparation	300		box of 100 pcs	1.5	3	
Catheter 20g	100		box of 100 pcs	7	1	
Gauze compresses 10*10 cm, sterile	90		pack of 45 pcs	1.75	2	
Gauze compresses 10*10 cm, non sterile	100		pack of 100 pcs	1.55	1	
Infusion giving set w. airinlet & needle	40		1 piece	0.18	40	
Syringe, disposable, 20 ml	100		box of 100 pcs	8.3	1	
Syringe, disposable, 10 ml	200		box of 100 pcs	2.95	2	
Syringe, disposable, 5 ml	100		box of 100 pcs	2.25	1	
Nasogastric tube no 14	1		box of 25 pcs	4.3	1	
TOTAL KIT 4				**370.85**		