

REVIEW ARTICLE

Starvation—By “Ill” or by “Will”

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ABSTRACT

Starvation is a severe deficiency in caloric energy intake. It is the most extreme form of malnutrition. In humans, prolonged starvation can cause permanent organ damage and eventually, death. According to the World Health Organization (WHO), hunger is the single gravest threat to the world's public health. The basic cause of starvation is an imbalance between energy intake and energy expenditure. In other words, the body expends more energy than it takes in. This imbalance can arise from one or more medical conditions and/or circumstantial situations. This review attempts to summarize the literature available on the various causes of starvation which may include any disease process or may occur by a voluntary act of shunning of food. The article discusses the body's response to starvation, which may be short-term starvation or prolonged starvation. The article also focuses on whether diabetes is a cause or an effect of starvation.

Keywords: Diabetes mellitus, Malnutrition, Starvation, Voluntary starvation.

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INTRODUCTION

Starvation describes the most severe form of malnutrition, in which there occurs a severe deficiency in energy intake resulting in a metabolic response focused on the sustenance of the vital organs to enable the survival of the affected individual.¹ In humans, prolonged starvation can cause permanent organ damage and eventually, death. The term ‘inanition’ refers to the symptoms and effects of starvation.

According to the WHO, hunger is the single gravest threat to the world's public health; malnutrition being

the biggest contributor to child mortality, present in half of all cases.² Undernutrition is a contributory factor in the death of 3.1 million children under five every year.

Food intake in humans is a punctuated process but energy is used up continuously by the body. Human body adapts well to short or long-term starvation, using their reserve stores of carbohydrates, fat and protein, and by mechanisms, which reduce energy expenditure as well as conserve the body protein. Food intake replenishes these stores by increase in the glycogen stores and re-esterification of triglycerides.³

STARVATION STATISTICS

After a prolonged period of decline, global hunger (measured by the number of undernourished people) is on the rise again, posing a challenge to international goals of eradicating hunger by 2030 by the United Nations.

According to the State of Food Security and Nutrition in the World 2017 report, the number of undernourished people in the world increased to an estimated 815 million in 2016, up from 777 million in 2015. Data from the report showed that India is home to 190.7 million of them—a 14.5% prevalence of hunger vis-a-vis its total population.

The data further showed that 38.4% of children under five in India are stunted, while 51.4% of women in reproductive ages are anemic. The report defines stunting as the result of long-term nutritional deprivation which may affect mental development, school performance and intellectual capacity.⁴

CAUSES OF STARVATION

Starvation may be caused either by an imbalance between inadequate caloric intake or due to an inability to digest food properly. Environmental circumstances such as draughts, famines or other natural catastrophes affecting the agriculture, poverty, or forceful withholding in certain geopolitical circumstances such as war or political prison camps may contribute to the unavailability of food. This occurs most commonly in under developed countries. In developed countries, the primary causes of starvation are medical which include diseases such as anorexia nervosa, bulimia nervosa, eating disorder not otherwise specified (EDNOS), celiac disease, coma, major depressive disorder, diabetes mellitus, digestive disease or constant vomiting etc.⁵ There are some clinical conditions such as recovering from surgery or burns etc., in which the person may be

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too fatigued or incapacitated to eat enough during their period of convalescence.

The Bengal famine of 1943 was a major famine in the Bengal province in British India during World War II (Fig. 1). An estimated 2 million,¹⁻³ out of a population of 60.3 million, died of starvation, malaria and other diseases aggravated by malnutrition, population displacement, unsanitary conditions and lack of health care. Millions were impoverished as the crisis overwhelmed large segments of the economy and social fabric. Historians have frequently characterised the famine as “man-made”, asserting that wartime colonial policies created and then exacerbated the crisis. A minority view holds that the famine arose from natural causes.

The photograph which was published in the newspaper, *The Statesman* in 1943 shows the plight of the sufferings of the people during the Bengal Famine. It made worldwide headlines and triggered government to act and save many lives.⁶

No demographic or geographic group was completely immune to increased deaths by disease, although deaths from starvation were confined to the rural poor.⁷

Deaths by starvation had peaked by November 1943⁸ by December, disease had become the most common cause of death.⁹ Disease-related mortality then continued to take its toll through early-to-mid 1944.¹⁰

Among diseases, malaria was the biggest killer.¹¹ Other famine-related deaths resulted from dysentery and diarrhea, typically through consumption of poor-quality food or deterioration of the digestive system caused by malnutrition.¹²

The two waves—starvation and disease—also interacted and amplified one another, increasing the excess mortality.¹³ Widespread starvation and malnutrition first compromised immune systems, and reduced resistance to disease led to death by opportunistic infections.¹⁴ Second, the social disruption and dismal conditions caused by a cascading breakdown of social systems brought mass migration, overcrowding, poor sanitation, poor water

quality and waste disposal, increased vermin, and unburied dead. All of these factors are closely associated with the increased spread of infectious disease.

BIOCHEMICAL CHANGES

In the postabsorptive state or the well-fed state (2–4 hours after meals), the available nutrients are gathered as glycogen, triacylglycerol (TAG) and proteins. So during this period transient increase in plasma glucose, amino acids and TAG (as chylomicrons) occurs. The pancreas respond to elevated levels of insulin and decreased levels of glucagon which amounts for the anabolic phase which makes glucose available to all tissues. The liver replenishes its glycogen stores, replaces hepatic proteins and increases TAG synthesis. Adipose tissues increase TAG synthesis and storage whereas the muscles increase the protein synthesis. The brain uses glucose exclusively in the fed state.¹⁵

During starvation, growth hormone levels rise, although response to growth hormone appears to decrease, and insulin like growth factor-1 (IGF-I) levels decrease inspite of the elevation in levels of growth hormone. However, lipolysis is enhanced. There is a relatively little change in glucocorticoid levels in starvation; however, normal levels are required for survival of prolonged fasting. In contrast, catecholamine levels rise in response to the initial hypoglycemia during fasting. Decreased thyroid hormone production, and in particular, decreased conversion of T4 to active T3 in peripheral tissues results in decreased basal metabolic rate. This results in an increased efficiency of fuel utilization and in reduced protein breakdown during caloric restriction and starvation. However, reduction in thyroid hormone levels requires several days to have significant effects.¹⁶ According to a study, a decrease in metabolic rate of 20–30% is induced by prolonged starvation. There also occurs upregulation of the expression of anabolic genes of the skeletal muscle and cartilage metabolism.^{17,18}

Bodily Responses to Starvation

Reaction to fasting is dependent on energy reserves, duration of starvation and any additional stressful influences.

Energy reserves

Fat is stored in adipocytes as triglycerides. One gram of pure triglycerides yields 9 Kcal, but a gram of adipose tissue yields 7 Kcal because it also contains less energy dense material such as proteins, electrolytes and water. One gram of protein yields approximately 4 Kcal, although, a gram of muscle, being 75% water, has an



Fig. 1: The Bengal Famine, *The Statesman*, 22 August 1943⁶

energy value of only 1 Kcal. Carbohydrate also has an energy value of approximately 4 Kcal/g (range 3.6–4.1) but its reserves in the body (liver and muscle glycogen) are limited to 500–800 g and are quickly exhausted.⁵

Duration of starvation

Short-term (<72 hours) Starvation

With short periods of starvation, there is diminished insulin and increased glucagon and catecholamine secretion leading to glycogenolysis and lipolysis. Triglycerides are broken down in adipose tissue releasing free fatty acids (FFAs) and glycerol into the circulation from where they are transported (FFAs bound to protein) for energy to organs such as skeletal and cardiac muscles, kidneys and liver. The glucose needs of the brain and erythrocytes are met initially from glycogenolysis (24 hours) but later from gluconeogenesis. Metabolic rate increases initially, but begins to decrease after 2 days⁵ (Figs 2 and 3).⁶

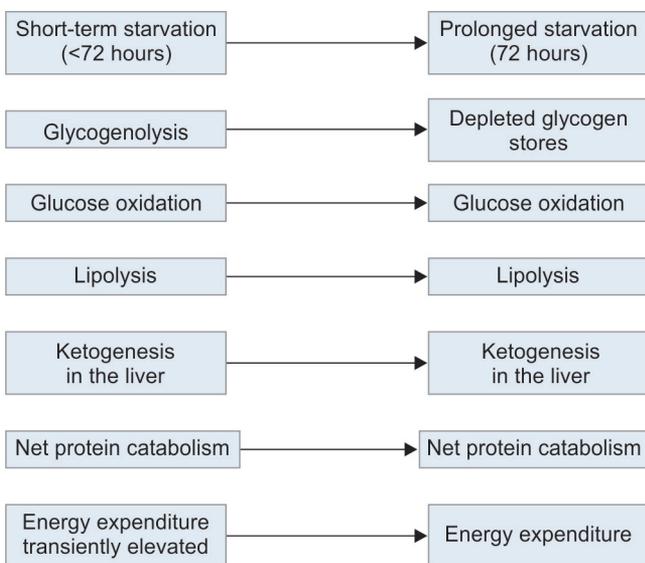


Fig. 2: Difference between metabolic reactions to short-term and long-term starvation

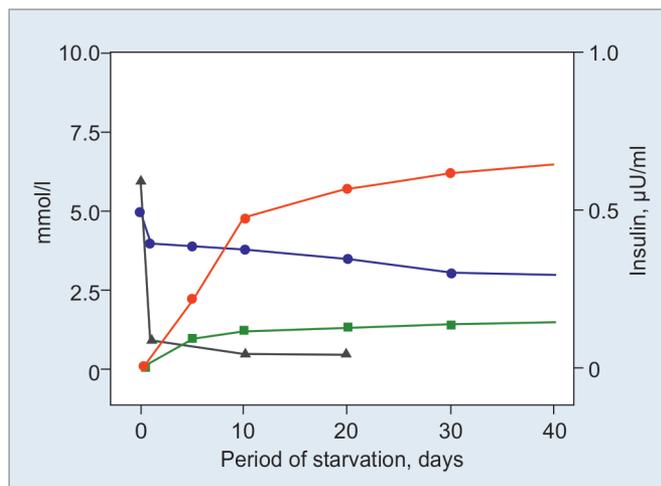


Fig. 3: Fuel choice during starvation

Prolonged (>72 hours) Starvation

Beyond 72 hours of starvation, insulin levels decrease further. Glycogen levels fall and glucose is derived from gluconeogenesis. As fatty acids cannot be converted to glucose, this process in liver and kidneys depends on a continuous supply of raw material in the form of amino acids from muscles, glycerol from adipose tissue and lactate from anaerobic glycolysis in muscles (Cori cycle). During gluconeogenesis from amino acids, the carbon skeleton enters the gluconeogenic pathway and the amino groups are converted to urea and excreted, leading to negative nitrogen balance and loss of up to 75 g of protein (300 g of muscle) daily.

This process is slowed and protein is conserved in two ways, firstly by a decline in metabolic rate by 10–15% and secondly by reduced glucose demand as the brain (which consumes 20% of total energy expenditure) adapts to using ketones as fuel. Starvation ensues when protein remains the only source of energy for the body.

With low food intake, diet induced thermogenesis is also reduced. In addition, depleted individuals show a decline in voluntary physical activity. All these factors contribute to a decrease in total energy expenditure.⁵

The plasma levels of fatty acids and ketone bodies increase in starvation, whereas that of glucose decreases.

SIGNS AND SYMPTOMS

According to a study, complete starvation leads to death within 8 to 12 weeks in adults. There are isolated cases of individuals living up to 25 weeks without food. Starvation begins when an individual has lost about 30% of his normal body weight. Once the loss reaches 40%, death is almost inevitable.

Individuals experiencing starvation lose substantial adipose tissue and muscle mass by the process of Catabolism, wherein the body breaks down its own muscles and other tissues in order to keep vital systems such as the nervous system and heart muscle (myocardium) functioning.¹⁹⁻²² Vitamin deficiencies are a common result of starvation, often leading to anemia, beriberi, pellagra, and scurvy. These diseases collectively can also cause diarrhea, skin rashes, edema, and heart failure. Individuals are often irritable and lethargic.

Early symptoms include impulsivity, irritability and hyperactivity. Atrophy of the stomach weakens the perception of hunger, since the perception is controlled by the percentage of the stomach that is empty. Victims of starvation are often too weak to sense thirst, and therefore become dehydrated.

All movements become painful due to muscle atrophy and skin becomes dry and cracked due to severe dehydration. Weakened body becomes susceptible to

many more diseases, for example, fungi which often grow under the esophagus, making swallowing painful. Starvation patients often suffer from infections—malaria, cholera, typhus, pneumonia, gastroenteritis. Cancrum oris, an infective gangrene of the mouth eroding the lip and cheeks, is a dreadful catastrophe which occasionally occurs in famines both among children and adults. The atrophied intestinal glands and the paper-thin walls of the digestive tract are unable to digest and absorb properly even a bland diet. In almost all famines, there is outbreak of diarrhoea without bacteriological organisms.

The energy deficiency in starvation causes fatigue and renders the victim more apathetic over time. As the starving person becomes too weak to move or even eat, their interaction with the surrounding world diminishes.

There is also an inability to fight diseases, and in females, irregular menstruation can occur.

Starvation and Stress

This occurs when the starved individual is subjected to stress in the form of trauma, sepsis and critical illness. In this situation, the normal adaptive responses of simple starvation, which conserve body protein, are over-ridden by the neuroendocrine and cytokine effects of injury. Metabolic rate rises rather than falls, ketosis is minimal, protein catabolism accelerates to meet the demands for tissue repair and of gluconeogenesis and there is hyperglycaemia and glucose intolerance. Salt and water retention is exacerbated and this may result in a kwashiorkor-like state with oedema and hypoalbuminaemia. The latter may also be exacerbated by protein deficiency. In severe wasting due to energy and protein deficiency, but without oedema, the practice is similar to that seen in marasmus caused by famine. In past, patients were described as suffering from ‘kwashiorkor’ or ‘marasmic’ type starvation depending on the differing proportions of substrate deficiency. According to new concept, kwashiorkor-like malnutrition is result of undernutrition and presence of disease or inflammatory process and fluid retention⁵ (Table 1).

Malnutrition and the Response to Injury

The bodily response to injury provides the substrates necessary for survival and in extreme cases, it results in dysfunction and tissue loss that threatens the mere survival of the individual. Patients with prior malnutrition, who develop acute illness, have less reserve to face that illness and do less well, usually with higher mortality, more complications and prolonged recovery.

Such patients have lower nitrogen excretion simply because they have less protein to mobilize. If surgery is planned on such a subject, a 2-week period of nutritional support needs to be given to improve physiological functions and lessen surgical risk. Short periods of feeding are not associated with much real tissue gain. Indeed, in convalescence, the synthesis rate of muscle proteins is only 1%/day, so that although adipose tissue may be repleted rapidly, it may take months to restore lean mass⁵ (Table 2). In chronic diseases, e.g. inflammatory bowel disease, there may not only be protein energy malnutrition but also single or multiple mineral and micronutrient deficiencies, which also affect important functions.

STARVATION IN UTERO

Malnutrition and starvation endured during famine may affect not only children and adults, but also fetuses in utero. The possibility of intrauterine programming of musculoskeletal disease developed in the adult human

Table 1: Simple starvation versus stress

Biochemical parameters	Simple (>72 hours)	Stress
Metabolic rate	↓	↑
Protein catabolism (relatively)	↓	↑
Protein synthesis (relatively)	↓	↑
Protein turnover	↓	↑
Nitrogen balance	↓	↓↓
Gluconeogenesis	↓	↑
Ketosis	↑↑	--
Glucose turnover	↓	↑
Blood glucose	↓	↑
Salt water retention	↑	↑↑↑
Plasma albumin	--	↓↓

Table 2: Difference in response to stress in the forms of infection, disease, surgery or trauma

Complications	Well nourished	Severe malnutrition
Protein catabolism	Appropriate to needs	Insufficient for needs
Muscle strength	Adequate	Inadequate to an illness
Septic complications	Low	Frequent
Bed sores	Rare	Frequent
Wound healing	Normal	Delayed
Hospital stay	Normal	Prolonged
Rehabilitation	Normal	Impaired

being was initially proposed by Lucas.²³ It is generally assumed that earlier the onset of maternal malnutrition or starvation during the development of fetus, the greater the effect on the bone mass and quality at birth.²⁴

EFFECTS OF STARVATION ON BONE GROWTH

All studies examining the connection between starvation and the bone metabolism in laboratory animal models and humans found evidence of either developmental delays, stunted bone growth, decreased bone mineral density or decreased cortical strength. Hermanussen et al.²⁵ demonstrated that stunted growth of long bones in both intact and GH-deficient rats induced by starvation was not repairable through a reinitiating of feeding as the growth spurts responsible for growth simply ceased during starvation and did not increase once feeding was commenced. Banu et al.^{26,27} were able to demonstrate a significant loss of endocortical bone as well as cancellous bone area and cancellous bone mineral content in rats with food restrictions in addition to concomitant decreases in tibial muscle mass.

STARVATION BY WILL

One of the most well known cases of voluntary starvation, is the hunger strike of Mahatma Gandhi. During his protest, Gandhi ate absolutely no food and only took sips of water for 21 days, and survived. What extraordinary about this case is the fact that Gandhi was very lean when he started his hunger-strike, thus not having much energy reserve from the outset. Also, it must be noted that during his life, Gandhi is reported to have performed a total of 14 hunger strikes.²⁸

A hunger strike is a method of non-violent resistance or pressure in which participants fast as an act of political protest, or to provoke feelings of guilt in others, usually with the objective to achieve a specific goal, such as a policy change. Most hunger strikers will take liquids but not solid food.

In cases where an entity (usually the state) has or is able to obtain custody of the hunger striker (such as a prisoner), the hunger strike is often terminated by the custodial entity through the use of force-feeding

Mahatma Gandhi was imprisoned in 1922, 1930, 1933 and 1942. Because of Gandhi's stature around the world, British authorities were loath to allow him to die in their custody. It is likely that Britain's reputation would have suffered as a result of such an event. Gandhi engaged in several famous hunger strikes to protest British rule of India. Fasting was a non-violent way of communicating the message and sometimes dramatically achieve the reason for the protest. This was keeping with the rules of *Satyagraha*.

In addition to *Gandhi*, various others used the hunger strike option during the Indian independence movement.

Such figures include Jatin Das (who fasted to death) and Bhagat Singh. It was only on the 116th day of their fast, on October 5, 1929 that Bhagat Singh and Dutt gave up their strike (surpassing the 97-day world record for hunger strikes which was set by an Irish revolutionary).²⁹ During this hunger strike that lasted 116 days and ended with the British succumbing to his wishes, he gained much popularity among the common Indians. Before the strike his popularity was limited mainly to the Punjab region.

Religious Point of View

We have often heard of *Rishi's* and *Yogi's* observing fast on their path to salvation. The scientific and mythological evidence for the same can be sought for in the holy book, '*Srimad Bhagvatam*'. In the book, it has been explained as to how people who shunned food were able to lead their way to the *Eternal Lord*. In reference to Dhruva Maharaja, who started to meditate in *Madhuvana* near river *Yamuna*, observed fasting in night. For the first month he ate only fruits and berries every third day, only to keep his body and soul together while worshipping the Supreme. In the second month, he ate every six days and that too dry grass and leaves. In the third month, he drank only water every nine days. Thus he remained completely in trance and kept worshipping. In the fourth month he became a master of his breathing, and inhaled air very twelfth day. Then, by the fifth month he was able to stand on one leg and kept praying focussing on the Immortal.³⁰

ANOREXIA NERVOSA

Anorexia nervosa is one of the most commonly encountered psychiatric disorder more common in females than in males. It is characterized by a self-induced restriction of caloric intake along with a variety of presentations including distorted self-perception and compulsive eating patterns.^{31,32} The physiological effects of anorexia nervosa resemble those of starvation including weight loss, muscle wasting, and amenorrhea. Osteoporosis seen in anorexia nervosa is thought to be a possible consequence of the lack of estrogen, calcium and Vitamin D, hypercortisolemia, or duration of the illness.³³⁻³⁷ The self-induced starvation of anorexia nervosa has been implicated in the development of osteoporosis in the young females.³⁸ Several studies have found a significantly decreased bone mineral density as well as an increased rate of bone resorption and a decreased rate of bone formation.³⁹⁻⁴¹

Complications

In extreme cases of anorexia nervosa, the subject may suffer from stress fractures, particularly if the individual performs strenuous exercise.⁴² Maugars et al. reported on

five females who were diagnosed with anorexia nervosa through the discovery of osteoporotic vertebral compression fractures or peripheral insufficiency fractures after a disease duration spanning 7–24 years.⁴³ Hypercaloric nutritional therapies in combination with calcium- and vitamin-D supplementation have been demonstrated to be helpful in restoring bone health. Estrogen replacement therapy has shown promising results if given in combination with low doses of recombinant human IGF-1 (rhIGF-1).

Other Medical Complications

These include loss of subcutaneous fat tissue, orthostatic hypotension, bradycardia, impaired menstrual function, hair loss, and hypothermia. Many are believed to be the result of the severe caloric restriction, which suppresses the hypothalamic-pituitary axis.⁴⁴ Biochemical mediators of this process include cortisol, leptin, growth hormone, and IGF-1, all essential mediators of growth and development in an adolescent.⁴⁵ This suppression results in a block in the production of luteinizing hormone and follicle-stimulating hormone. There is no production of estrogen, hence no ovulation, and subsequent amenorrhea. The amenorrhea may sometimes be the reason the patient seeks medical attention.

TREATMENT OF SUBJECTS SUFFERING FROM STARVATION

Starving patients can be treated, but this must be done cautiously to avoid complications in the form of refeeding syndrome.⁴⁶ Rest and warmth must be provided and maintained. Small sips of water mixed with glucose should be given at regular intervals. Fruit juices can also be given. Later, food can be given gradually in small quantities. The quantity of food can be increased over time. Proteins may be administered intravenously to raise the level of serum proteins.⁴⁷

Most famine victims, owing to alimentary dysfunction, cannot consume large quantities of food. The patient's desire for food is immense but his digestive capacities limit the intake. Food intake should necessarily be limited. The choice of food is vital. Many starving people in the prison camp of World War II died from diarrhoea and collapsed after they had been given bully-beef, baked beans which they could not readily digest. Only bland foods can be tolerated by the thin-walled intestines lacking essential digestive enzymes. Frequent small feeds of skimmed milk, under constant personal attention and nursing care can be given. A variety of mild flavoring essences may be useful to stimulate the appetite. Slightly sour foods are usually acceptable. Starving patients may tolerate moderate amounts of fat or edible oils, which

provide a larger energy intake. There may be a temporary increase in *oedema* with re-feeding, so the intake of salt should be restricted. A time may come in severe starvation when the patient refuses all food. The outlook is then very grave wherein, nasogastric or parenteral feeding provides the only hope.

Treatment of Anorexia Nervosa

A thorough and detailed history and physical examination is advisable. Particular emphasis should be placed on the diet and social history, as well as the past history, in order to delineate the needs of the patient and explore the impact that nutrition and physical activity could have had on the patient's symptoms and current status, as well as her future growth and development. Education plays an important part in the treatment of patients with anorexia nervosa, as many adolescents with anorexia nervosa have misconceptions about what constitutes “healthy eating”. Cognitive-behavioral therapy (CBT) can be instituted in order to try and break, as well as restructure the strict food rules and rituals, and erroneous beliefs that the patient may have.^{48,49}

Refeeding Syndrome

Refeeding syndrome can be defined as the potentially fatal shifts in fluids and electrolytes that may occur in malnourished patients receiving artificial refeeding (whether enterally or parenterally). These shifts result from hormonal and metabolic changes and may cause serious clinical complications. The hallmark biochemical feature of refeeding syndrome is hypophosphataemia. However, the syndrome is complex and may also feature abnormal sodium and fluid balance; changes in glucose, protein, and fat metabolism; thiamine deficiency; hypokalaemia; and hypomagnesaemia.⁴⁶

During refeeding, increase in the blood glucose levels leads to increased insulin and decreased secretion of glucagon. Insulin stimulates glycogen, fat, and protein synthesis. This process requires minerals such as phosphate and magnesium and cofactors such as thiamine. Insulin stimulates the absorption of potassium into the cells through the sodium-potassium ATPase symporter, which also transports glucose into the cells. Magnesium and phosphate are also taken up into the cells. Water follows by osmosis. These processes result in a decrease in the serum levels of phosphate, potassium, and magnesium, all of which are already depleted. The clinical features of the refeeding syndrome occur as a result of the functional deficits of these electrolytes and the rapid change in basal metabolic rate.

If the syndrome is detected, the rate of feeding should be slowed down and essential electrolytes should be

replenished. The hospital specialist dietetics team should be involved.

DIABETES AND STARVATION—ARE THEY ALIKE?

Diabetes mimics fasting especially in the responses of liver, muscle cells, and adipose tissues. Even with high blood glucose levels, intracellular levels may be very low due to insulin receptor malfunction. With low serum ratios of insulin to glucagon and high levels of fatty acids, liver produces glucose while other tissues use fatty acids and ketones instead of glucose. Muscle glycogen almost disappears, and muscle protein is broken down to support gluconeogenesis. Cardiac and skeletal muscles meet their energy needs from ketones and fatty acids. Fat cells actively release fatty acids under the lipolytic stimuli of glucagon, catecholamines, and insulin deficiency (Fig. 4).

CAN STARVATION INDUCE DIABETES?

Fasting can induce glucose intolerance and altered glucose metabolism. Many studies have been put forward in this context. According to a study, seven initially obese individuals who, during the course of a strenuous weight-reduction program, developed diabetes mellitus: non-insulin-dependent DM in five cases and insulin-dependent DM in two cases. None had any sign of prior diabetic symptoms. Although weight reduction is encouraged in obesity, crash diets without proper medical surveillance may have deleterious effects. The metabolic situation in extremely low-calorie diets may be comparable to that in starvation. The induction of a diabetic state during such diets can be explained on the basis of increased insulin resistance in states of starvation and anorexia nervosa, with a concomitant role in stress hormones.⁵¹

Thrifty Phenotype Hypothesis

It proposes that the epidemiological associations between poor fetal and infant growth and the subsequent development of Type 2 diabetes and the metabolic syndrome result from the effects of poor nutrition in early life, which produces permanent changes in glucose-insulin metabolism.⁵²

Reduced fetal growth is strongly associated with a number of chronic conditions later in life. This increased susceptibility results from adaptations made by the fetus in an environment limited in its supply of nutrients. Proponents of this idea say that in poor nutritional conditions, a pregnant woman can modify the development of her unborn child such that it will be prepared for survival in an environment in which resources are likely to be short, resulting in a thrifty phenotype (Hales & Barker, 1992.⁵³ It is sometimes called Barker’s hypothesis, after Professor David J. P. Barker, researching at the University of Southampton who published the theory in 1990.⁵⁴

Early-life metabolic adaptations help in survival of the organism by selecting an appropriate trajectory of growth in response to environmental cues.

Individuals with a thrifty phenotype will have “a smaller body size, a lowered metabolic rate and a reduced level of behavioural activity adaptations to an environment that is chronically short of food”.⁵⁵ Those with a thrifty phenotype who actually develop in an affluent environment may be more prone to metabolic disorders, such as obesity and Type II diabetes, whereas those who have received a positive maternal forecast will be adapted to good conditions and therefore better able to cope with rich diets. This idea is now widely (if not universally) accepted and is a source of concern for

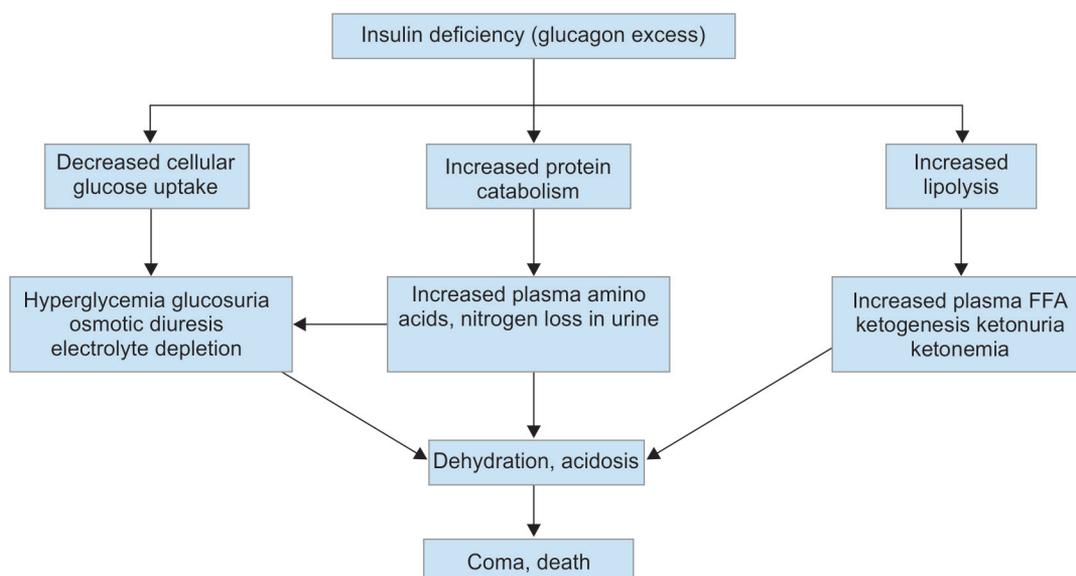


Fig. 4: Nutrition for diabetes mellitus⁵⁰

societies undergoing a transition from sparse to better nutrition.^{56,57}

STARVATION DIETS AS A PART OF TREATMENT OF DIABETES MELLITUS

Severely calorie-restricted diets were the best therapy for diabetes for a brief period, from 1915 until the introduction of insulin in 1922. These were termed as “starvation diets” –diets based on repeated fasting and prolonged undernourishment –as the most advanced treatment for diabetes mellitus, not as a cure, but for relief of symptoms and maximum extension of life. Fasting did reduce glucose levels in diabetics, but prolonged calorie-restricted diets introduced new hazards, most obviously death by starvation. It is accepted today that calorie restriction is beneficial for diabetics who are overweight. But for those of normal or lower weight, severe calorie restriction could lessen resistance to infection, and for children it might stunt growth.⁵⁸

CONCLUSION

Human body ideally adapts well to a short-term or a long-term starvation, by making use of the reserve stores of carbohydrates, fat and protein. There occurs an adaptive reduction of energy expenditure along with conservation of body protein. Energy stores are replenished during feeding period. Long-term partial or total cessation of energy intake leads to marasmic wasting. With the addition of the stress response, catabolism and wasting are accelerated and the normal adaptive responses to simple starvation are over-ridden. Weight loss in either situation results in impaired mental and physical function, as well as poorer clinical outcome.

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