Death by Starvation and Dehydration

16

Case Study

A 22-year-old mother presented at her family physician in a small rural town one evening in March and handed over an evidently deceased infant wearing an old, soiled diaper and dressed in a snowsuit. The mother reported that she had given her 14-month-old daughter a bottle of milk only 1 h or so earlier, but later stated that this had been around lunchtime. Due to the condition of the body, the family physician informed the police.

When questioned, the young mother claimed that she had not been able to cope with caring for her daughter and had not been looking after her in recent weeks. She had repeatedly told her husband that the child was asleep and that he should not wake her. The husband had last seen the child 3 weeks previously. The mother had not appealed for assistance to any of the people who could have helped her, since she feared that her child would be taken away from her. More recently, due to the squalid condition of her home, she had not allowed anyone entrance.

On-site investigations confirmed the squalid condition of the house. "It smelled like a pet shop," reported one of the police officers. The level of humidity inside the house was high, almost comparable to a subtropical climate. Every conceivable surface area was filled with aquariums. Two well-nourished dogs were also found in the home; the father of the child had been walking the dogs while the mother had brought the dead infant to the home physician. Both parents were drug users.

At autopsy, the 14-month-old infant weighed 5,996 g at a crown–heel length of 73 cm. Extreme dehydration was observed, alongside subcutaneous and pararenal fatty tissue depletion, muscle atrophy, facial wrinkling, straw-like hair, nutritional edema on the upper and lower extremities, and extensive diaper dermatitis. In addition, massive cerebral edema (cerebral weight, 1,021 g), a thin parchment-like pericardium, and thickened blood were all striking.

Although death by starvation and/or dehydration is relatively rare in industrialized countries, chronic malnutrition and death due to starvation are not uncommon when seen from a global perspective. Fatalities of this kind occur in the setting of extreme cachexia due to, for example, tumor cachexia, anorexia nervosa, hunger strike, age-related cachexia, accident-related burial, malabsorption and malassimilation diseases, congenital cardiac defects and anomalies, and other consumptive diseases such as infections, tuberculosis, and thyroid dysfunction. If malnutrition is the

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cause of death in fatal cachexia, one refers to actual death by starvation. Fatalities due to starvation are seen in forensic medicine in the case of:

- Neglect of infants and young children
- Psychiatric patients
- Anorexia nervosa patients (women > men)
- Accident victims cut off from food supplies
- Prisoners left to starve to death
- Hunger strikes (usually for political reasons)
- Refusal to eat for other reasons, e.g., elderly, sick persons
- Withholding food from elderly care-dependent individuals: Homicide by omission

In addition to identifying death by starvation, and irrespective of whether the immediate cause of death is purulent bronchopneumonia for instance, a number of other questions require answers:

- Starvation persisted over what period of time?
- Is it possible to classify the starvation in terms of severity?
- Was the gradually worsening and possibly life-threatening condition of the victim recognizable to family members, parents, the authorities, etc., prior to death?

There is no clear differentiation between the terms "cachexia" and "marasmus" mentioned here. Common terms and definitions used in relation to starvation and death by starvation include:

Cachexia: Generalized atrophy of the organism involving more than 20 % weight loss (may vary according to initial weight).

Marasmus: Generalized muscle atrophy associated with severe emaciation due to starvation dystrophy (undernutrition).

Kwashiorkor: A tropical form of protein–energy malnutrition (PEM) due to protein deficiency despite an otherwise sufficient intake of calories in the form of carbohydrates.

Inanition: Depletion of the body's energy reserves. Although the process is reversible for a long period of time, it is not possible to reliably determine the point at which it becomes irreversible.

Nutritional edema can be seen in advanced stages of starvation.

Nutritional edema: Extracellular buildup of water in tissue and body cavities (ascites) due to malnutrition or in the context of nutritional dystrophy resulting from protein deficiency.

A state of chronic malnutrition can also develop in the case of regular or intermittent yet quantitatively and qualitatively insufficient nutrition. This can make assertions about the duration of starvation up to the time of death equally as challenging as absent or unreliable information about body weight prior to the period of starvation. Nevertheless, using the body weight determined at either forensic physical examination or autopsy and applying age-appropriate standard values as well as classifications proposed in the literature (see below), an estimate of the duration of starvation can be made by way of extrapolation. However, it is important to clarify here whether complete nutritional abstention or rather occasional but insufficient food intake has taken place. Of note is also the fact that marked weight loss, or failure to gain weight contrary to expectations, should prompt the responsible persons or guardians to take action, irrespective of whether these individuals are medical lay people!

16.1 Death by Starvation

As a basic principle, starvation and ultimately death by starvation result when an individual's daily calorie intake falls below daily calorie requirements.

Important: **The World Health Organization (WHO) defines malnutrition as the cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions**.

However, death by starvation can only be diagnosed when—taking histological and chemical–toxicological analyses into consideration—all other causes of death can be reliably ruled out, and moreover, the circumstances and findings of the specific case do not present any precluding factors.

16.1.1 Starvation and Death by Starvation in Adults

In the early phase of starvation, i.e., the first 24 h, rapidly available energy reserves from stored carbohydrates, most notably glycogen, are primarily used up. If no food intake takes place after this

period, glucose needs are then met by means of proteolysis and gluconeogenesis, which can persist for some weeks and produces a negative nitrogen balance. The body responds to insufficient food intake by adapting to a catabolic metabolism (starvation adaption). The organism's basal metabolic rate drops after approximately 8–10 days. The body continues to call upon its own energy reserves, fats (lipolysis) with accompanying ketogenesis, as well as protein, leading to increased levels of acetone in urine. Since ketone bodies are released during starvation, metabolic acidosis develops (starvation acidosis). Reduced metabolic function results in a drop in body temperature of around 1 °C. Starvation-related depletion produces a number of findings:

- Marked weight loss depending on the duration of starvation or malnutrition.
- Rarefied subcutaneous fat tissue.
- Rarefaction of fatty tissues within the body, most notably fatty tissue of the greater omentum, the mesentery, and the adipose capsule of the kidney.
- In some cases, gelatinous transformation of fatty tissue.
- Atrophic skeletal muscles.
- Narrow and largely empty (at most, bilious mucous and scant stool) gastrointestinal lumen due to contraction, thin gastric wall.
- Fecaliths in the gastrointestinal lumen.
- The gallbladder is frequently filled with greenish bile.
- Atrophy and weight loss in internal organs (except the brain).
- Atrophy also seen in endocrine organs and lymphatic tissue.
- In extreme cases, even subepicardial fatty tissue is strongly rarefied and hepatocyte vacuolization may be observed in the liver.

Death occurs after weight loss of up to 40 % of the body's initial weight, possibly more in the case of preexisting obesity.

In principle, the extent of starvation dystrophy can be estimated by determining the body mass index (BMI), even though this value is typically used to classify overweight (Table 16.1). However, the BMI does not permit any assertions regarding the severity or duration of starvation to be made—it merely describes a state.

Table 16.1 Determining nutritional status using body mass index (BMI)

Classification	BMI (kg/m²)
Underweight	<18.5
Normal	18.5–24.9
Grade I: overweight	25.0 bis 29.9
Grade II: obese	30.0 bis 39.9
Grade III: extreme obesity	>40.0

The main focus of attention in cases of death by starvation (and dehydration) is on macroscopic findings. However, there are histological findings that correlate with macroscopic findings and patient history: subcutaneous fatty tissue with gelatinous atrophy (Fig. 16.1).

Although a final aspiration may occasionally be seen, infection (bronchial pneumonia, urogenital infection, etc.) should also be considered as a possible direct cause of death. Bone tissue demonstrates increased resorption lacunae and osteoclasts in the case of advanced rickets, while frontal lobe atrophy and acute renal tubular necrosis have both been described.

Anorexia Nervosa: A psychiatric disease associated with a severe eating disorder. Although girls and young women are most commonly affected, anorexia nervosa is also seen in men (ration of women to men, ca. 10:1). Patients control their eating habits to the point of rigid dieting or long periods of starvation (restrictive form of anorexia nervosa). Sometimes, the sensation of hunger is suppressed by drinking large volumes of fluids, or weight loss is accelerated by self-induced vomiting, taking diuretics/appetite suppressants, etc. (active form of anorexia nervosa, binge-purging type). These patients, who often undertake excessive physical or sporting activity, are frequently achievement-oriented and show a tendency towards perfectionism. Anorexia nervosa patients are distinctly underweight, often to the extent of complete cachexia. Not infrequently, body weight is more than 25 % below age-specific norms (in adults, a BMI of <17.5). Once a certain weight threshold has been fallen below, extensive disruption to the endocrine system is seen, including amenorrhea and loss of libido. Psychological and psychiatric comorbidities manifest more frequently (depression as well as anxiety, personality, and addictive disorders).

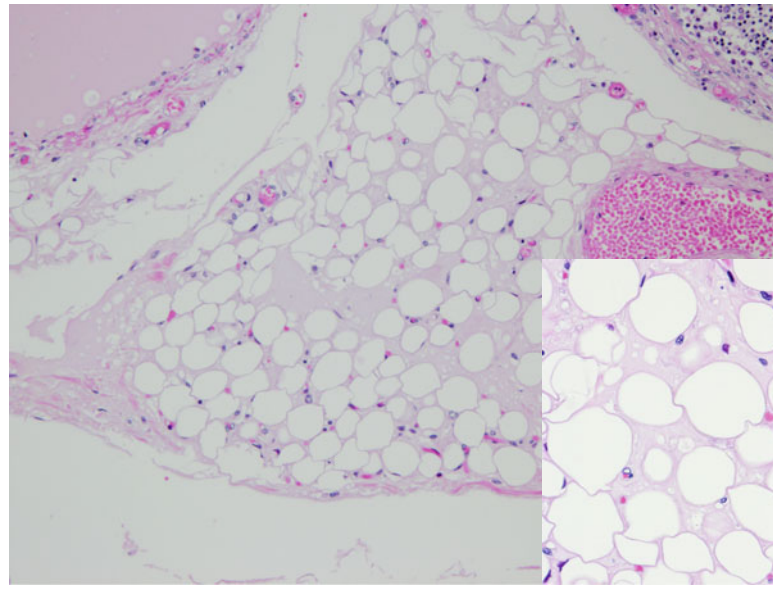

Fig. 16.1 Gelatinous atrophy in fatty tissue due to hunger striking (77 days) (H&E×100; H&E×400)

Furthermore, an electrolyte and vitamin imbalance occurs, albumin and overall protein levels in blood are reduced, and skin becomes dry and flaky; anemia and liver dysfunction are sometimes concomitant. Next, liver enzymes are increased (transaminase, lipase), and the onset of osteoporosis and kidney dysfunction is seen in later stages, as well as possible peripheral neuronal damage and seizures. In this setting, the sudden death of a relatively young patient, likely in a state of extreme chronic malnutrition, occasionally prompts a forensic autopsy to determine the cause of death. In contrast to other cases of chronic malnutrition due to neglect (in children, care-dependent persons, etc.) or hunger strike, the patient history can be followed regularly over a long time period in the case of anorexia nervosa, while the condition of the body at the time of death also suggests the diagnosis. Anorexia nervosa has a mortality rate of 10 %.

16.1.2 Starvation and Death by Starvation in Children

Alongside the abovementioned findings, additional signs seen in children in terms of age and initial weight range from growth retardation to short stature. Significantly reduced body weight is also seen in children at autopsy (Fig. 16.2; see Fig. 20.13).

Adverse effects are classified by applying anthropometric parameters, such as those specified by the WHO. According to the WHO classification, the extent and duration of PEM are subdivided into three groups:

Stunting: Insufficient height relative to age. This anthropometric parameter reflects growth in height and is an indicator of growth failure as a result of long-term malnutrition.

Wasting: Insufficient weight relative to height, an indicator of acute weight loss.

Underweight: Insufficient weight relative to age.

According to the WHO definition, one of the above forms of malnutrition is present if the corresponding anthropometric finding in a child falls below two standard deviations from the median of the same-age reference group.

In order to better assess the extent of starvation or chronic malnutrition in children, PEM classifications were developed in the field of pediatrics on the basis of anthropometric data. Alongside the Wellcome classification of PEM (Table 16.2) and the Gomez classification (Table 16.3), the Waterlow classification is the most commonly used to estimate nutritional status (Table 16.4).

The Gomez classification has the disadvantage that smaller children are still classified as

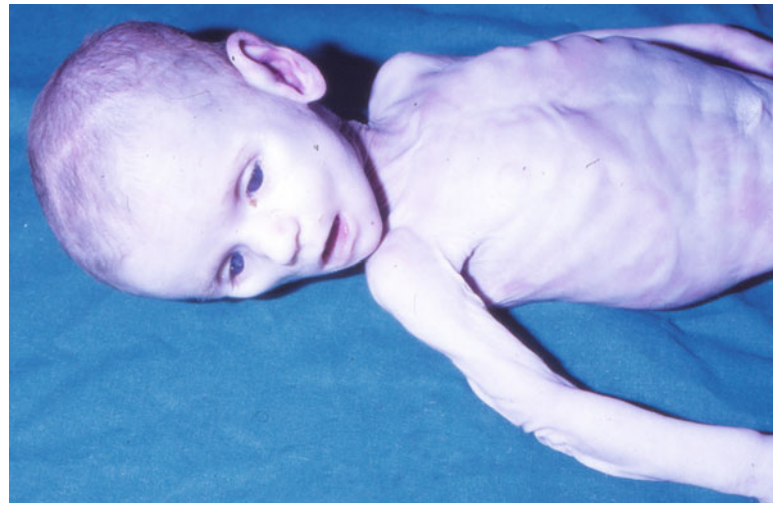

Fig. 16.2 Death by starvation, female, 9 weeks, significantly reduced body weight

Table 16.2 The Wellcome classification of protein–energy malnutrition (PEM)

	Percentage of benchmark values for weight/age (%)	Edema
Subclinical PEM	80–60	–
Marasmus	<60	–
Kwashiorkor	>60	+
Marasmic kwashiorkor	<60	+

Table 16.3 Classification of protein–energy malnutrition (PEM) according to Gomez

	Percentage of standard weight (age) (%)
Gomez I	90–76
Gomez II	75–61
Gomez III	<61

malnourished even if their weight is adequate relative to their height. Thus, if there is a high percentage of children of shorter height in a particular region compared to children in industrialized countries, applying the Gomez classification will result in a considerable overestimation of malnutrition.

The Waterlow classification uses the age-independent weight-to-height relationship as well as the child's height. Growth retardation is interpreted in the classification as a sign of chronic PEM, i.e., a previous state of starvation. However, the best indicator of acute PEM is insufficient weight relative to height (wasting). Severe acute malnutrition is present in the case of a weight-to-height relationship of less than 70 % of the standard value. The Waterlow classification estimates chronic starvation dystrophy and retardation by using weight-to-height relationships and height-to-age relationships as follows:

Table 16.4 Waterlow classification for the estimation of malnutrition

Weight/height Height/age	Value > 80 %	Value < 80 %
Value >80 %	Normal	Acutely malnourished
Value <80 %	Chronically malnourished	Chronically and acutely malnourished

Weight/height–height/age

Two fields are defined for each parameter—above two standard deviations and below two standard deviations, altogether yielding four defined categories (Table 16.4):

- Normal nutrition
- Acute malnutrition
- Chronic malnutrition
- Acute and chronic malnutrition

Other interpretations of this classification additionally differentiate on the basis of:

1. *Degree of retardation*: >95 %, normal; 95–87.5 %, mild; 87.5–80 %, moderate; <80 %, severe
2. *Degree of acute malnutrition*: >90 %, normal; 90–80 %, mild; 80–70 %, moderate; <70 %, severe

Table 16.5 Classification of malnutrition in children

	Mild malnutrition	Moderate malnutrition	Severe malnutrition
Percent ideal body weight (%)	80–90	70–79	<70
Percent of usual body weight (%)	90–95	80–89	<80
Albumin (g/dl)	2.8–3.4	2.1–2.7	<2.1
Transferrin (mg/dl)	150–200	100–149	<100
Total lymphocyte count (per µl)	1,200–2,000	800–1,199	<800

Weight and height are not the only parameters used to assess the degree of chronic malnutrition in children–variations in certain laboratory parameters are also correlated with the severity of malnutrition (Table 16.5).

Duration of Starvation. According to data in the literature, if there is a complete lack of food intake, a weight reduction of 0.7–1 %/day of a child's initial weight can be assumed when estimating the duration of starvation or death by starvation. Using the weight measured at the time of examination or autopsy should yield a time period that approximately corresponds to the duration of starvation.

16.2 Death by Dehydration

Whereas an individual can survive a lack of nutrition for a relatively long time period (see below), fluid deprivation or insufficient water intake can cause death in a considerably shorter period of time.

Although daily fluid requirements depend on ambient temperature and level of physical activity, a person requires a minimum of between 1 and 2 l/day. The feeling of thirst becomes apparent from 0.5 to 3 % loss of body fluids. The body needs water in the blood vessels to transport nutrients and cells, as well as in individual cells to maintain cell metabolism and regulate temperature. Failure to drink water causes dehydration.

Hypertonic Dehydration (or exsiccosis): Reduction in body water. Death by dehydration occurs as a result of hypertonic dehydration, i.e., primarily Na levels in plasma remain almost unchanged despite loss of water.

Severe states of dehydration are associated with central nervous disorders, most notably impaired consciousness and reduced vigilance. Hypertonic dehydration causes hypovolemia, a drop in cardiac output and blood pressure, as well as hypovolemic shock.

Loss of skin turgor and raised skin folds are striking at autopsy (Fig. 20.13). The speed of death by dehydration is highly temperature-dependent, occurring within 1 day in a hot desert or after many days under cool and shady conditions, whereby perspiration is also a relevant factor.

16.3 Causes of Death

In the case of death due to starvation or dehydration, death is caused by a variety of pathologic changes or diseases.

Lack of fluids (death by dehydration) can lead to an electrolyte imbalance accompanied by ventricular arrhythmia, as well as to hypovolemic shock and subsequent cardiovascular failure.

Death due to lack of nutrition can occur either early on in the course of starvation or at a later stage depending on specific circumstances such as age, preexisting diseases, preexisting nutritional status, fluid intake, and ambient temperature. Morgulis (1923) reported a time period of 17–76 days (median 40 days), while Prokop (1966) reported 60 days for nutrition deprivation only and 8–21 days for combined nutrition and fluid deprivation.

In the case of death due to chronic malnutrition (death by starvation), acute lethal hypoglycemia is the most likely immediate cause of death alongside secondary infections, e.g., purulent bronchopneumonia, ascending urethritis, and pyelonephritis. In addition to these findings, hepatic steatosis of varying degree has been reported histologically.

In certain cases, postmortem biochemical investigations can help in the diagnosis of hypoglycemia:

Hypoglycemia. As with the diagnosis of hyperglycemia or diabetic coma as the cause of death, measuring glucose and lactate levels in cerebrospinal fluid or vitreous humor can also help in the setting of hypoglycemia. Although postmortem values are subject to significant variation depending on the postmortem interval and ambient temperature, it is still helpful to calculate the sum value of glucose and lactate measured in cerebrospinal fluid or vitreous humor (sum value in mg/dl according to Traub). Thus, lower-range sum values of 50–80 mg/dl in cerebrospinal fluid and 100–160 mg/dl in vitreous humor are given for fatal hypoglycemia. However, there is no systematic investigation that involves determining glucose and lactate values in cerebrospinal fluid and vitreous humor in the case of death by starvation or dehydration. This should be borne in mind when interpreting the values measured, since their reliability is limited in cases of fatal starvation/dehydration.

Hyperglycemia and Diabetic Coma. Values exceeding 362 mg/dl in cerebrospinal fluid indicate a high likelihood of lethal diabetic coma once other causes of death have been ruled out; an upper value of 410 mg/dl has been reported in the literature for vitreous humor. Both values are believed to remain relatively stable for up to 10 days post-mortem under cold-storage conditions. HbA-1c determination can be used to estimate antemortem glucose levels, whereby an increased value is an indication of preexisting hypoglycemia of long standing.

Selected References and Further Reading

Adelsberger L (1946) Medical observations in Auschwitz concentration camp. Lancet 2:317–319

Adelson L (1963) Homicide by starvation: the nutritional variant of the battered child. JAMA 186:458–460

Cahill GF (1970) Starvation in man. N Engl J Med 282:668–675

Colomb V (2003) Dénutrition de l'enfant. Rev Prat 53:263–267

Davis JH, Rao VJ, Valdes-Dapena M (1984) A forensic approach to a starved child. J Forensic Sci 29:663–669

Dettmeyer R (2011) Forensic histopathology. Fundamentals and perspectives. Springer, Berlin/Heidelberg/New York

Ellerstein NS, Ostrov BE (1985) Growth pattern in children hospitalized because of caloric-deprivation failure to thrive. Am J Dis Child 139:164–166

Garnett ES, Barnard DL, Ford J, Goodbody RA, Woodehouse MA (1969) Gross fragmentation of cardiac myofibrils after therapeutic starvation for obesity. Lancet 1:914–916

Gomez F, Galvan RR, Cravioto J, Frenk S (1955) Malnutrition in infancy and childhood with special reference to Kwashiokor. Adv Pediatr 131–169

Grover Z (2009) Protein energy malnutrition. Pediatr Clin North Am 56:1055–1068

Herrmann B, Dettmeyer R, Banaschak S, Thyen U (2010) Kindesmisshandlung, 2nd edn. Springer, Berlin/Heidelberg/New York

Hughes EA, Stevens LH, Wilkinson AW (1964) Some aspects of starvation in the newborn baby. Arch Dis Child 39:598–604

Isner JM, Roberts WC, Heymsfield SB, Yager J (1985) Anorexia nervosa and sudden death. Ann Intern Med 102:49–52

Janssen W (1977) Histologische Befunde bei Hunger-Schäden. In: Forensische Histologie, Janssen Wth edn. Schmidt-Römhild, Lübeck, pp 327–333

Kernbach G, Brinkmann B (1983) Postmortale Pathochemie für die Feststellung der Todesursache "Coma diabeticum". Pathologe 4:235–240

Leiter LA, Marliss EB (1982) Survival during fasting may depend on fat as well as protein stores. JAMA 248:2306–2307

Listernick R, Christoffel K, Pace J, Chiaramonte J (1985) Severe primary malnutrition in US children. Am J Dis Child 139:1157–1160

Madea B (2005) Death as a result of starvation – diagnostic criteria. In: Tsokos M (ed) Forensic pathology reviews, vol 2. Humana Press, Totowa, pp 1–23

Meade JL, Brissie RM (1985) Infanticide by starvation: calculation of caloric deficit to determine degree of deprivation. J Forensic Sci 30:1263–1268

Melchior JC (2003) Diagnostic et dépistage de la dénutrition. Rev Prat 53:254–258

Mollison PL (1948) Observation of cases of starvation at Belsen. Br Med J 1:4–8

Morgulis S (1923) Experimentelle Unterernährung. In: Morgulis S (ed) Hunger und Unterernährung. Springer, Berlin, pp 71–85

Nagao M, Maeno Y, Koyama H, Seko-Nakamura Y, Monma-Ohtaki J, Iwasa M et al (2004) Estimation of caloric deficit in a fatal case of starvation resulting from child neglect. J Forensic Sci 49:1073–1076

Piercecchi-Marti MD, Pelissier-Alicot AL, Leonetti G, Tervé JP, Cianfarani F, Pellisier JF (2004) Pellagra: a rare disease observed in a victim of mental and physical abuse. Am J Forensic Med Pathol 25:342–344

Piercecchi-Marti MD, Louis-Borrione CL, Bartoli C, Sanvoison A, Panuel M, Pelissier-Alicot AL, Leonetti G (2006) Malnutrition, a rare form of child abuse: diagnostic criteria. J Forensic Sci 51:670–673

Prokop O (1966) Das Verhungern. In: Prokop O (ed) Forensische Medizin. VEB Verlag, Berlin, pp 141–143

Ratcliffe PJ, Bevan JS (1985) Severe hypoglycaemia and sudden death in anorexia nervosa. Psychol Med 15:679–681

Rich LM, Caine MR, Findling JW, Shaker JL (1990) Hypoglycemic coma in anorexia nervosa. Arch Intern Med 150:894–895

Rißе M, Rummel J, Tsokos M, Dettmeyer R, Büttner A, Lehmann H, Püschel K (2010) Death due to starvation and thirst. Extreme forms of fatal negligence in childhood. Rechtsmedizin 20:211–218

Ritz S, Kaatsch HJ (1990) Postmortale Diagnostik von tödlichen diabetischen Stoffwechselentgleisungen: Welchen Stellenwert haben Liquor- und Glaskörperflüssigkeitssummenwerte sowie der HBA-1-Wert? Pathologe 11:158–165

Roberts IF, West RJ, Ogilvie D, Dillon MJ (1979) Malnutrition in infants receiving cult diets: a form of child abuse. Br Med J 1:296–298

Sarvesvaran E (1992) Homicide by starvation. Am J Forensic Med Pathol 13:264–267

Saukko P, Knight B (2004) Neglect, starvation and hypothermia. In: Knight's forensic pathology, 3rd edn. Arnold, London, pp 412–420

Sippel H, Möttönen M (1982) Combined glucose and lactate values in vitreous humour for post-mortem diagnosis of diabetes mellitus. Forensic Sci Int 19:217–222

Steinhauer JR, Volk A, Hardy R, Konrad R, Daly T, Robinson CA (2002) Detection of ketosis in vitreous at autopsy after embalming. J Forensic Sci 47:221–223

Sturner WO, Sullivan A, Suzuki K (1983) Lactic acid concentrations in vitreous humour: their use in asphyxial deaths in children. J Forensic Sci 28:222–230

Traub F (1969) Methode zur Erkennung von tödlichen Zuckerstoffwechselstörungen an der Leiche (Diabetes mellitus und Hypoglykämie). Zbl All Path 112:390–399

Waterlow JC (1972) Classification and definition of protein-caloric malnutrition. Br Med J 2:566–569

Waterlow JC (1973) Note on the assessment and classification of protein-energy malnutrition in children. Lancet 87–89

Waterlow JC (1979) The anthropometric assessment of malnutrition in children. Lancet 2:250–251

Waterlow JC, Buzina R, Keller W, Lane M, Nichaman MZ, Tanner JM (1977) The presentation and use of height and weight data for comparing the nutritional status of groups of children under the age of 10 years. Bull World Health Organ 55:489–498

United Nations Administrative Committee on Coordination – Sub-Committee on Nutrition (ACC/SCN) in collaboration with International Food Policy Research Institute (IFPRI) (2000) The 4th Report on World Nutrition Situation. Geneva, Switzerland, pp 5–17