3 Fasting

The Fasting State. *Fasting begins approximately 2 to 4 hours after a meal, when blood glucose levels return to basal levels, and continues until blood glucose levels begin to rise after the start of the next meal. Within about 1 hour after a meal, blood glucose levels begin to fall. Consequently**, insulin** levels decline, and **glucagon** levels rise. These changes in hormone levels trigger the **release of fuels** from the body stores. **Liver glycogen** is **degraded** by the process of **glycogenolysis**, which supplies glucose to the blood. **Adipose triacylglycerols** are **mobilized** by the process of **lipolysis**, which releases fatty acids and glycerol into the blood. Use of **fatty acids** as a fuel increases with the length of the fast; they are the **major fuel** oxidized during overnight fasting.*

*Fuel Oxidation. During fasting, **glucose** continues to be **oxidized** by **glucose-dependent tissues** such as the brain and red blood cells, and **fatty acids** are oxidized by tissues such as muscle and liver. Muscle and most other tissues oxidize fatty acids completely to CO_2 and H_2O. However, the **liver** partially oxidizes fatty acids to smaller molecules called **ketone bodies**, which are released into the blood. Muscle, kidney, and certain other tissues derive energy from completely oxidizing ketone bodies in the tricarboxylic acid (TCA) cycle.*

*Maintenance of Blood Glucose. As fasting progresses, the **liver produces glucose** not only by **glycogenolysis** (the release of glucose from glycogen), but also by a second process called **gluconeogenesis** (the synthesis of glucose from noncarbohydrate compounds. The major **sources of carbon** for gluconeogenesis are **lactate**, **glycerol**, and **amino acids**. When the carbons of the amino acids are converted to glucose by the liver, their **nitrogen** is converted to **urea**.*

*Starvation. When we fast for 3 or more days, we are in the starved state. **Muscle** continues to burn fatty acids but **decreases** its use of **ketone bodies**. As a result, the concentration of ketone bodies rises in the blood to a level at which the **brain** begins to **oxidize** them for energy. The brain then needs less glucose, so the liver decreases its rate of gluconeogenesis. Consequently, less **protein** in muscle and other tissues is degraded to supply amino acids for gluconeogenesis, Protein sparing preserves vital functions for as long as possible. Because of these changes in the fuel utilization patterns of various tissues, humans can survive for extended periods without ingesting food.*

THE WAITING ROOM

Percy Veere had been admitted to the hospital with a diagnosis of mental depression associated with malnutrition (see Chap. 1). At the time of admission, his body weight of 125 lb gave him a body mass index (BMI) of 17.5 (healthy range, 18.5–24.9). His serum albumin was 10% below the low end of the normal range, and he exhibited signs of iron and vitamin deficiencies.

Pathways named with the suffix "lysis" are those in which complex molecules are broken down or "lysed" into smaller units. For instance, in glycogenolysis, glycogen is lysed into glucose subunits; in glycolysis, glucose is lysed into two pyruvate molecules; in lipolysis, triacylglycerols are lysed into fatty acids and glycerol; in proteolysis, proteins are lysed into their constituent amino acids.

Gluconeogenesis means formation (genesis) of new (neo) glucose, and by definition, converts new (noncarbohydrate) precursors to glucose.

Degrees of protein–energy malnutrition (marasmus) are classified according to BMI.

Protein–energy Malnutrition	BMI (kg/cm^2)
I	17.0–18.4
II	16.0–16.9
III	<16.0

Percy Veere has grade I protein–energy malnutrition. At his height of 71 inches, his body weight would have to be above 132 lb to achieve a BMI greater than 18.5. **Ann O'Rexia** has grade III malnutrition. At 66 inches, she needs a body weight greater than 114 lb to achieve a BMI of 18.5.

Additional tests were made to help evaluate Mr. Veere's degree of malnutrition and his progress toward recovery. His arm circumference and triceps skinfold were measured, and his mid upper arm muscle circumference was calculated (see Chap. 2, Anthropometric Measurements). His serum transferrin, as well as his serum albumin, were measured. Fasting blood glucose and serum ketone body concentration were determined on blood samples drawn the next day before breakfast. A 24-hour urine specimen was collected to determine ketone body excretion and creatinine excretion for calculation of the creatinine–height index, a measure of protein depletion from skeletal muscle.

Ann O'Rexia was receiving psychological counseling for anorexia nervosa, but with little success (see Chap. 1). She saw her gynecologist because she had not had a menstrual period for 5 months. She also complained of becoming easily fatigued. The physician recognized that Ann's body weight of 85 lb was now less than 65% of her ideal weight. (Her BMI was now 13.7.) The physician recommended immediate hospitalization. The admission diagnosis was severe malnutrition secondary to anorexia nervosa. Clinical findings included decreased body core temperature, blood pressure, and pulse (adaptive responses to malnutrition). Her physician ordered measurements of blood glucose and ketone body levels and made a spot check for ketone bodies in the urine as well as ordering tests to assess the functioning of her heart and kidneys.

I.   THE FASTING STATE

Blood glucose levels peak approximately 1 hour after eating and then decrease as tissues oxidize glucose or convert it to storage forms of fuel. By 2 hours after a meal, the level returns to the fasting range (between 80 and 100 mg/dL). This decrease in blood glucose causes the pancreas to decrease its secretion of insulin, and the serum insulin level decreases. The liver responds to this hormonal signal by starting to degrade its glycogen stores and release glucose into the blood.

If we eat another meal within a few hours, we return to the fed state. However, if we continue to fast for a 12-hour period, we enter the basal state (also known as the postabsorptive state). A person is generally considered to be in the basal state after an overnight fast, when no food has been eaten since dinner the previous evening. By this time, the serum insulin level is low and glucagon is rising. Figure 3.1 illustrates the main features of the basal state.

A.   Blood Glucose and the Role of the Liver during Fasting

The liver maintains blood glucose levels during fasting, and its role is thus critical. Glucose is the major fuel for tissues such as the brain and neural tissue, and the sole fuel for red blood cells. Most neurons lack enzymes required for oxidation of fatty acids, but can use ketone bodies to a limited extent. Red blood cells lack mitochondria, which contain the enzymes of fatty acid and ketone body oxidation, and can use only glucose as a fuel. Therefore, it is imperative that blood glucose not decrease too rapidly nor fall too low.

Initially, liver glycogen stores are degraded to supply glucose to the blood, but these stores are limited. Although liver glycogen levels may increase to 200 to 300 g after a meal, only approximately 80 g remain after an overnight fast. Fortunately, the liver has another mechanism for producing blood glucose, known as gluconeogenesis. In gluconeogenesis, lactate, glycerol, and amino acids are used as carbon sources to synthesize glucose. As fasting continues, gluconeogenesis progressively adds to the glucose produced by glycogenolysis in the liver.

Percy Veere had not eaten much on his first day of hospitalization. His fasting blood glucose determined on the morning of his second day of hospitalization was 72 mg/dL (normal, overnight fasting = 80–100 mg/dL). Thus, in spite of his malnutrition and his overnight fast, his blood glucose was being maintained at nearly normal levels through gluconeogenesis using amino acid precursors. If his blood glucose had decreased below 50 to 60 mg/dL during fasting, his brain would have been unable to absorb glucose fast enough to obtain the glucose needed for energy and neurotransmitter synthesis, resulting in coma and eventual death. Although many other tissues, such as the red blood cell, are also totally or partially dependent on glucose for energy, they are able to function at lower concentrations of blood glucose than the brain.
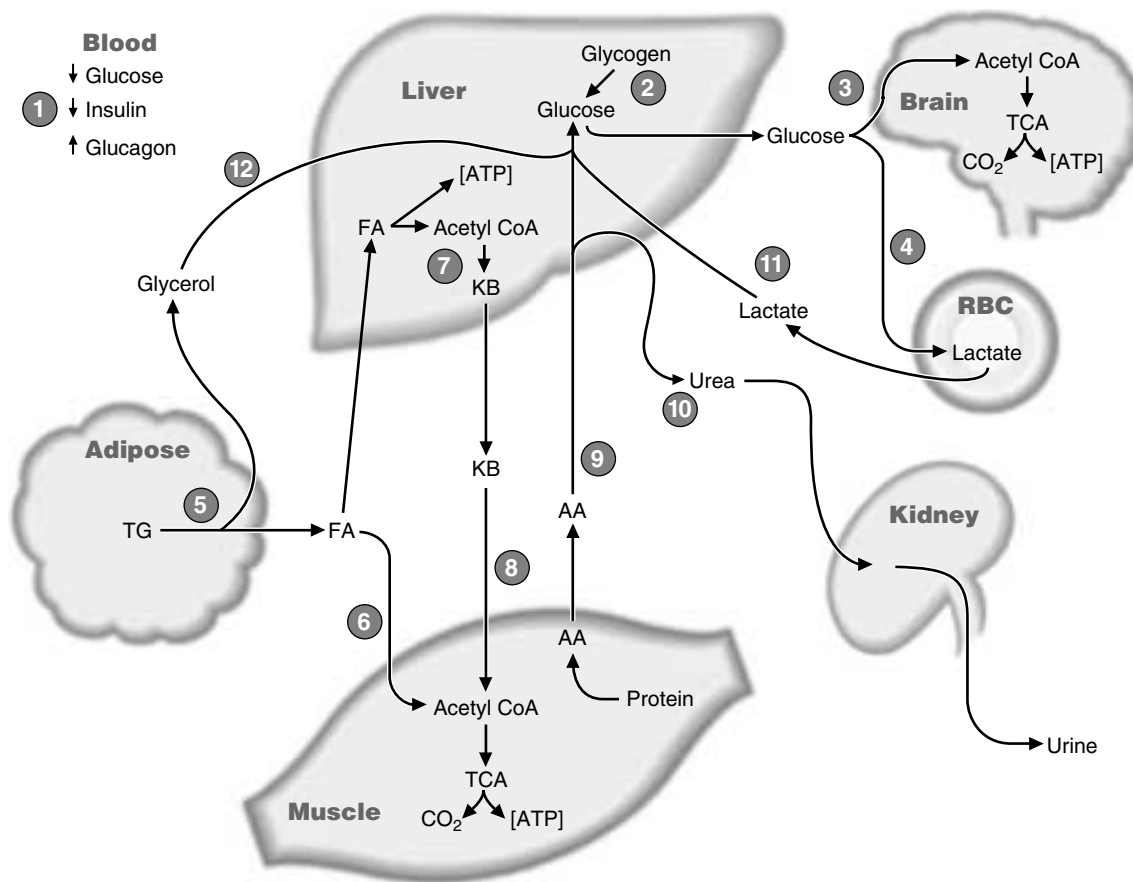
Fig. 3.1. Basal state. This state occurs after an overnight (12-hour) fast. The circled numbers serve as a guide indicating the approximate order in which the processes begin to occur. KB = ketone bodies. Other abbreviations are defined in Figure 2.1.

Lactate is a product of glycolysis in red blood cells and exercising muscle, glycerol is obtained from lipolysis of adipose triacylglycerols, and amino acids are generated by the breakdown of protein. Because our muscle mass is so large, most of the amino acid is supplied from degradation of muscle protein. These compounds travel in the blood to the liver, where they are converted to glucose by gluconeogenesis. Because the nitrogen of the amino acids can form ammonia, which is toxic to the body, the liver converts this nitrogen to urea. Urea has two amino groups for just one carbon (NH_2-CO-NH_2). It is a very soluble, nontoxic compound that can be readily excreted by the kidneys and thus is an efficient means for disposing of excess ammonia.

As fasting progresses, gluconeogenesis becomes increasingly more important as a source of blood glucose. After a day or so of fasting, liver glycogen stores are depleted and gluconeogenesis is the only source of blood glucose.

B. Role of Adipose Tissue During Fasting

Adipose triacylglycerols are the major source of energy during fasting. They supply fatty acids, which are quantitatively the major fuel for the human body. Fatty acids are not only oxidized directly by various tissues of the body; they are also partially oxidized in the liver to 4-carbon products called ketone bodies. Ketone bodies are subsequently oxidized as a fuel by other tissues.

As blood insulin levels decrease and blood glucagon levels rise, adipose triacylglycerols are mobilized by a process known as lipolysis. They are converted to fatty acids and glycerol, which enter the blood.

On the second day of hospitalization, **Percy Veere's** serum ketone body level was 110 μM. (Normal valve after a 12-hour fast, is approximately 70 μM.) No ketone bodies were detectable in his urine. At this stage of protein–energy malnutrition, Mr. Veere still has remaining fat stores. After 12 hours of fasting, most of his tissues are using fatty acids as a major fuel, and the liver is beginning to produce ketone bodies from fatty acids. As these ketone bodies increase in the blood, their use as a fuel will increase.

It is important to realize that most fatty acids cannot provide carbon for gluconeogenesis. Thus, of the vast store of food energy in adipose tissue triacylglycerols, only the small glycerol portion travels to the liver to enter the gluconeogenic pathway.

Fatty acids serve as a fuel for muscle, kidney, and most other tissues. They are oxidized to acetyl CoA, and subsequently to CO_2 and H_2O in the TCA cycle, producing energy in the form of adenosine triphosphate (ATP). In addition to the ATP required to maintain cellular integrity, muscle uses ATP for contraction, and the kidney uses it for urinary transport processes.

Most of the fatty acids that enter the liver are converted to ketone bodies rather than being completely oxidized to CO_2. The process of conversion of fatty acids to acetyl CoA produces a considerable amount of energy (ATP), which drives the reactions of the liver under these conditions. The acetyl CoA is converted to the ketone bodies acetoacetate and β-hydroxybutyrate, which are released into the blood (Fig. 3.2).

The liver lacks an enzyme required for ketone body oxidation. However, ketone bodies can be further oxidized by most other cells with mitochondria, such as muscle and kidney. In these tissues, acetoacetate and β-hydroxybutyrate are converted to acetyl CoA and then oxidized in the TCA cycle, with subsequent generation of ATP.

C. Summary of the Metabolic Changes during a Brief Fast

In the initial stages of fasting, stored fuels are used for energy (see Fig. 3.1). The liver plays a key role by maintaining blood glucose levels in the range of 80 to 100 mg/dL, first by glycogenolysis and subsequently by gluconeogenesis. Lactate, glycerol, and amino acids serve as carbon sources for gluconeogenesis. Amino acids are supplied by muscle. Their nitrogen is converted in the liver to urea, which is excreted by the kidneys.

Fatty acids, which are released from adipose tissue by the process of lipolysis, serve as the body's major fuel during fasting. The liver oxidizes most of its fatty acids only partially, converting them to ketone bodies, which are released into the blood. Thus, during the initial stages of fasting, blood levels of fatty acids and ketone bodies begin to increase. Muscle uses fatty acids, ketone bodies, and (when exercising and while supplies last) glucose from muscle glycogen. Many other tissues use either fatty acids or ketone bodies. However, red blood cells, the brain, and other neural tissues use mainly glucose. The metabolic capacities of different tissues with respect to pathways of fuel metabolism are summarized in Table 3.1.

II. METABOLIC CHANGES DURING PROLONGED FASTING

If the pattern of fuel utilization that occurs during a brief fast were to persist for an extended period, the body's protein would be quite rapidly consumed to the point at which critical functions would be compromised. Fortunately, metabolic changes occur during prolonged fasting that conserve (spare) muscle protein by causing muscle protein turnover to decrease. Figure 3.3 shows the main features of metabolism during prolonged fasting (starvation).

B. Role of Liver During Prolonged Fasting

After 3 to 5 days of fasting, when the body enters the starved state, muscle decreases its use of ketone bodies and depends mainly on fatty acids for its fuel. The
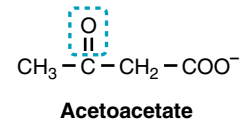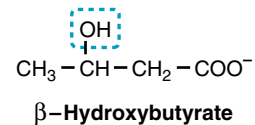

Fig. 3.2. β-Hydroxybutyrate and acetoacetate. These compounds are ketone bodies formed in the iner. A third ketone body, acetone (CH_3-CO-CH_3), is produced by nonenzymatic decarboxylation of acetoacetate. However, acetone is expired in the breath and not metabolized to a significant extent in the body.

The liver synthesizes a number of serum proteins and releases them into the blood. These proteins decrease in the blood during protein malnutrition. Two of these serum proteins, albumin and transferrin (an iron-binding transport protein), are often measured to assess the state of protein malnutrition. Serum albumin is the traditional standard of protein malnutrition. Neither measurement is specific for protein malnutrition. Serum albumin and transferrin levels decrease with hepatic disease, certain renal diseases, surgery, and a number of other conditions, in addition to protein malnutrition. Serum transferrin levels also decrease in iron deficiency. **Percy Veere's** values were below the normal range for both of these proteins, indicating that his muscle mass is unable to supply sufficient amino acids to sustain both synthesis of serum proteins by the liver and gluconeogenesis.

Table 3.1. Metabolic Capacities of Various Tissues

| Process	Liver	Adipose Tissue	Kidney Cortex	Muscle	Brain	RBC
TCA cycle (acetyl CoA → CO_2 + H_2O)	+++	++	+++	+++	+++	— —
β-Oxidation of fatty acids	+++	— —	++	+++	— —	— —
Ketone body formation	+++	— —	+	— —	— —	— —
Ketone body utilization	— —	+	+	+++	+++ (prolonged starvation)	— —
Glycolysis (glucose → CO_2 + H_2O)	+++	++	++	+++	+++	— —
Lactate production (glucose → lactate)	+	+	— — —	+++ exercise	+	+++
Glycogen metabolism (synthesis and degradation)	+++	+	+	+++	+	— —
Gluconeogenesis (lactate, amino acids, glycerol → glucose)	+++	— —	+	— —	— —	— —
Urea cycle (ammonia → urea)	+++	— —	— —	— —	— —	— —
Lipogenesis (glucose → fatty acids)	+++	+	— —	— —	— —	— —
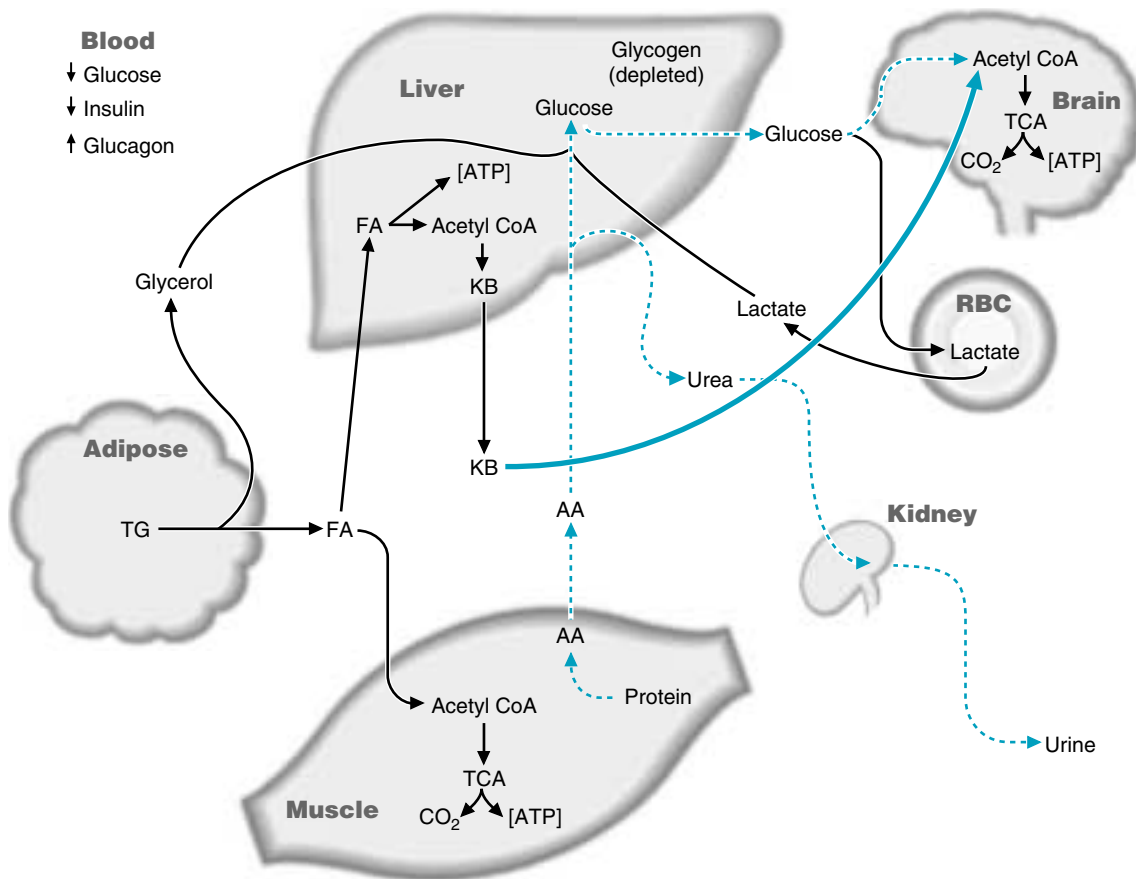
Fig. 3.3. Starved state. Abbreviations are defined in Figures 2.1 and 3.1. Dashed lines indicate processes that have decreased, and the heavy solid line indicates a process that has increased relative to the fasting state.

liver, however, continues to convert fatty acids to ketone bodies. The result is that the concentration of ketone bodies rises in the blood (Fig. 3.4). The brain begins to take up these ketone bodies from the blood and to oxidize them for energy. Therefore, the brain needs less glucose than it did after an overnight fast (Table 3.2).

Glucose is still required, however, as an energy source for red blood cells, and the brain continues to use a limited amount of glucose, which it oxidizes for energy and uses as a source of carbon for the synthesis of neurotransmitters. Overall, however, glucose is "spared" (conserved). Less glucose is used by the body, and, therefore, the liver needs to produce less glucose per hour during prolonged fasting than during shorter periods of fasting.

Because the stores of glycogen in the liver are depleted by approximately 30 hours of fasting, gluconeogenesis is the only process by which the liver can supply glucose to the blood if fasting continues. The amino acid pool, produced by the breakdown of protein, continues to serve as a major source of carbon for gluconeogenesis. A fraction of this amino acid pool is also being used for biosynthetic functions (e.g., synthesis of heme and neurotransmitters) and new protein synthesis, processes that must continue during fasting. However, as a result of the decreased rate of gluconeogenesis during prolonged fasting, protein is "spared"; less protein is degraded to supply amino acids for gluconeogenesis.

While converting amino acid carbon to glucose in gluconeogenesis, the liver also converts the nitrogen of these amino acids to urea. Consequently, because glucose production decreases during prolonged fasting compared with early fasting, urea production also decreases (Fig. 3.5).

B. Role of Adipose Tissue During Prolonged Fasting

During prolonged fasting, adipose tissue continues to break down its triacylglycerol stores, providing fatty acids and glycerol to the blood. These fatty acids serve as the major source of fuel for the body. The glycerol is converted to glucose, whereas the fatty acids are oxidized to CO_2 and H_2O by tissues such as muscle. In the liver, fatty acids are converted to ketone bodies that are oxidized by many tissues, including the brain.

A number of factors determine how long we can fast and still survive. The amount of adipose tissue is one factor, because adipose tissue supplies the body with its major source of fuel. However, body protein levels can also determine the length of time we can fast. Glucose is still used during prolonged fasting (starvation), but in greatly reduced amounts. Although we degrade protein to supply amino acids for gluconeogenesis at a slower rate during starvation than during the first days of a fast, we are still losing protein that serves vital functions for our tissues. Protein can become so depleted that the heart, kidney, and other vital tissues stop functioning, or we can develop an infection and not have adequate reserves to mount an immune response. In addition to fuel problems, we are also deprived of the vitamin and mineral precursors of coenzymes and other compounds necessary for tissue function. Because of either a lack of ATP or a decreased intake of electrolytes, the electrolyte composition of the blood or cells could become incompatible with life. Ultimately, we die of starvation.

Ann O'Rexia's admission laboratory studies showed a blood glucose level of 65 mg/dL (normal fasting blood glucose = 80 − 100 mg/dL). Her serum ketone body concentration was 4,200 μM (normal = ~70 μM). The Ketostix (Bayer Diagnostics, Mishawaha, IN) urine test was moderately positive, indicating that ketone bodies were present in the urine. In her starved state, ketone body use by her brain is helping to conserve protein in her muscles and vital organs.
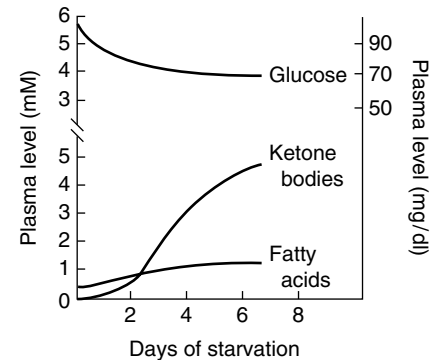
Fig. 3.4. Changes in the concentration of fuels in the blood during prolonged fasting.

Death by starvation occurs with loss of roughly 40% of body weight, when approximately 30 to 50% of body protein has been lost, or 70 to 95% of body fat stores. Generally, this occurs at BMIs of approximately 13 for men, and 11 for women.
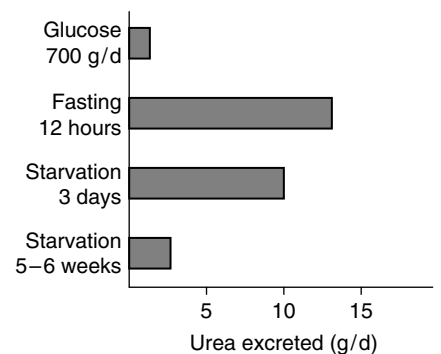
Fig. 3.5. Changes in urea excretion during fasting. Urea production is very low in a person consuming only glucose. It increases during fasting as muscle protein is broken down to supply amino acids for gluconeogenesis. However, as fasting progresses, urea synthesis decreases. Because the brain meets some of its energy needs by oxidizing ketone bodies after 3 to 5 days of fasting, gluconeogenesis decreases, sparing protein in muscle and other tissues.

Table 3.2. Metabolic Changes during Prolonged Fasting Compared with Fasting 24 Hours

Muscle	↓	Use of ketone bodies
Brain	↑	Use of ketone bodies
Liver	↓	Gluconeogenesis
Muscle	↓	Protein degradation
Liver	↓	Production of urea

Creatinine–Height Index. The most widely used biochemical marker for estimating body muscle mass is the 24-hour urinary creatinine excretion. Creatinine is a degradation product formed in active muscle at a constant rate, in proportion to the amount of muscle tissue present in a patient. In a protein-malnourished individual, urinary creatinine will decrease in proportion to the decrease in muscle mass. To assess depletion of muscle mass, creatinine excreted is expressed relative to the height, the creatinine–height index (CHI). The amount of creatinine (in milligrams) excreted by the subject in 24 hours is divided by the amount of creatinine excreted by a normal, healthy subject of the same height and sex. The resulting ratio is multiplied by 100 to express it as a percentage. **Percy Veere**'s CHI was 85% (80–90% of normal indicates a mild deficit; 60–80% indicates a moderate deficit; less than 60% of normal indicates a severe deficit of muscle mass).

Fig. 3.6. Photograph of a patient with anorexia nervosa. From a MedCom slide, 1970.

CLINICAL COMMENTS

Percy Veere. As a result of his severely suppressed appetite for food, Percy Veere has developed a mild degree of protein–calorie malnutrition. When prolonged, this type of protein malnutrition can cause changes in the villi of the small intestine that reduce its absorptive capacity for what little food is ingested.

Despite his insufficient intake of dietary carbohydrates, Mr. Veere's blood glucose level is 72 mg/dL, close to the lower limit (80 mg/dL) of the normal range for a well-nourished, healthy person after a 12-hour fast. This is the finding you would expect; it reflects the liver's capacity to maintain adequate levels of blood glucose by means of gluconeogenesis, even during prolonged and moderately severe caloric restriction. Amino acids from degradation of protein, principally in skeletal muscle, supply most of the precursors for gluconeogenesis.

Percy Veere has several indicators of his protein malnutrition: his serum albumin and transferrin levels are below normal, his mid-upper-arm muscle circumference (MUAMC) is at the 12th percentile, and his creatinine–height index (CHI) was at 85%. The low levels of serum proteins reflect a low dietary protein intake, and possibly diminished capacity to absorb dietary amino acids. Consequently, amino acids are being mobilized from degradation of protein in muscle and other tissues to supply precursors for new protein synthesis as well as gluconeogenesis. The result is a loss of muscle mass, indicated by the MUAMC and the CHI, and decreased levels of serum proteins.

Fatty acids mobilized from adipose tissue are the major source of energy for most tissues. Because he is eating, and not in total starvation, his ketone bodies were only moderately elevated in the blood (110 μM vs. normal of 70 μM) and did not appear in the urine.

After several psychological counseling sessions, and the promise of an extended visit from his grandchild, Mr. Veere resumed his normal eating pattern.

Ann O'Rexia. Ann O'Rexia has anorexia nervosa, a chronic disabling disease in which poorly understood psychological and biologic factors lead to disturbances in the patient's body image. These patients typically pursue thinness in spite of the presence of severe emaciation and a "skeletal appearance" (Fig. 3.6). They generally have an intense fear of being overweight and deny the seriousness of their low body weight.

Amenorrhea (lack of menses) usually develops during anorexia nervosa and other conditions when a woman's body fat content falls to approximately 22% of her total body weight. The immediate cause of amenorrhea is a reduced production of the gonadotropic protein hormones (luteinizing hormone and follicle-stimulating hormone) by the anterior pituitary; the connection between this hormonal change and body fat content is not yet understood.

Ms. O'Rexia is suffering from the consequences of prolonged and severe protein and caloric restriction. Fatty acids, released from adipose tissue by lipolysis, are being converted to ketone bodies in the liver, and the level of ketone bodies in the blood is extremely elevated (4,200 μM vs. normal of 70 μM). The fact that her kidneys are excreting ketone bodies is reflected in the moderately positive urine test for ketone bodies noted on admission.

Although Ms. O'Rexia's blood glucose is below the normal fasting range (65 mg/dL vs. normal of 80 mg/dL), she is experiencing only a moderate degree of hypoglycemia (low blood glucose) despite her severe, near starvation diet. Her blood glucose level reflects the ability of the brain to use ketone bodies as a fuel when they are elevated in the blood, thereby decreasing the amount of glucose that must be synthesized from amino acids provided by protein degradation.

Ms. O'Rexia's BMI showed that she was close to death through starvation. She was therefore hospitalized and placed on enteral nutrition (nutrients provided through tube feeding). The general therapeutic plan, outlined in Chapter 1, of nutritional restitution and identification and treatment of those emotional factors leading to the patient's anorectic behavior was continued. She was coaxed into eating small amounts of food while hospitalized.

BIOCHEMICAL COMMENTS

Clinical Use of Metabolite Measurements in Blood and Urine. When a patient develops a metabolic problem, it is difficult to examine cells to determine the cause. To obtain tissue for metabolic studies, biopsies must be performed. These procedures can be difficult, dangerous, or even impossible, depending on the tissue. Cost is an additional problem. However, both blood and urine can be obtained readily from patients, and measurements of substances in the blood and urine can help in diagnosing a patient's problem. Concentrations of substances that are higher or lower than normal indicate which tissues are malfunctioning. For example, if blood urea nitrogen (BUN) levels are low, a problem centered in the liver might be suspected because urea is produced in the liver. Conversely, high blood levels of urea suggest that the kidney is not excreting this compound normally. Decreased urinary and blood levels of creatinine indicate diminished production of creatinine by skeletal muscle. However, high blood creatinine levels could indicate an inability of the kidney to excrete creatinine, resulting from renal disease. If high levels of ketone bodies are found in the blood or urine, the patient's metabolic pattern is that of the starved state. If the high levels of ketone bodies are coupled with elevated levels of blood glucose, the problem is most likely a deficiency of insulin; that is, the patient probably has type 1, formerly called insulin-dependent, diabetes mellitus. Without insulin, fuels are mobilized from tissues rather than being stored.

These relatively easy and inexpensive tests on blood and urine can be used to determine which tissues need to be studied more extensively to diagnose and treat the patient's problem. A solid understanding of fuel metabolism helps in the interpretation of these simple tests.

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REVIEW QUESTIONS—CHAPTER 3

You will need some information from Chapters 1 and 2, as well as Chapter 3, to answer these questions.

1. By 24 hours after a meal,

    (A) gluconeogenesis in the liver is the major source of blood glucose.
    (B) muscle glycogenolysis provides glucose to the blood.
    (C) muscles convert amino acids to blood glucose.
    (D) fatty acids released from adipose tissue provide carbon for synthesis of glucose.
    (E) ketone bodies provide carbon for gluconeogenesis.

2. The liver is the only tissue that

    (A) contains significant glycogen stores.
    (B) oxidizes fatty acids during overnight fasting.
    (C) oxidizes ketone bodies during overnight fasting.
    (D) converts ammonia to urea.
    (E) converts glucose to lactate.

3. In a well-nourished individual, as the length of fasting increases from overnight to 1 week,

    (A) blood glucose levels decrease by approximately 50%.
    (B) red blood cells switch to using ketone bodies.
    (C) muscles decrease their use of ketone bodies, which increase in the blood.
    (D) the brain begins to use fatty acids as a major fuel.
    (E) adipose tissue triacylglycerols are nearly depleted.

4. A hospitalized patient had low levels of serum albumin and high levels of blood ammonia. His CHI was 98%. His BMI was 20.5. Blood urea nitrogen was not elevated, consistent with normal kidney function. The diagnosis most consistent with these finding is

    (A) loss of hepatic function (e.g., alcohol-induced cirrhosis).
    (B) anorexia nervosa.
    (C) kwashiorkor (protein malnutrition).
    (D) marasmus (protein–energy malnutrition).
    (E) decreased absorption of amino acids by intestinal epithelial cells (e.g., celiac disease).

5. Otto Shape, an overweight medical student (see Chapter 1), discovered that he could not exercise enough during his summer clerkship rotations to lose 2 to 3 lb per week. He decided to lose weight by eating only 300 kcal/day of a dietary supplement that provided half the calories as carbohydrate and half as protein. In addition, he consumed a multivitamin supplement. During the first 3 days on this diet,

    (A) his protein intake met the RDA for protein.
    (B) his carbohydrate intake met the fuel needs of his brain.
    (C) both his adipose mass and his muscle mass decreased.
    (D) he remained in nitrogen balance.
    (E) he developed severe hypoglycemia.