Nutrition and the Cardiac Surgical Patient*

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Cardiac surgery is now being performed on older, sicker patients often presenting with multiple pathologic conditions. Other patients presenting for treatment of noncardiac problems are found to have cardiac abnormalities requiring operative correction. Frequently these patients are extremely ill and require lengthy preoperative hospitalization for both diagnosis and interim therapy. Although usually not apparent, protein-calorie malnutrition is common in hospitalized patients. Anthropomorphic and laboratory measurements suggest that 25 percent to 50 percent of patients hospitalized for more than two weeks are affected by some degree of protein-calorie malnutrition. Obesity does not ensure adequate protein stores. Prehospital nutrition is often marginal in these patients, and typically worsens with hospitalization due to restricted diets, restricted activity, and interruption of meals because of testing. Many patients in the hospital also receive nutritional supplementation. Both facts have important cardiac implications.

Earlier concepts that the heart is spared in malnutrition have been shown to be incorrect. Malnutrition results in a proportional loss of skeletal and myocardial muscle. Severe cardiac debility results in worsening nutrition, which might in turn produce unsuspected, but clinically significant, myocardial atrophy. Nutritional support may play a role in improving cardiac function in selected patients who are being prepared for cardiac surgery, and in patients with rapid weight loss who are at risk for sudden death due to arrhythmias.

As these patients become more prevalent, it is essential that the internist, cardiologist, and cardiac surgeon understand the interrelationships of nutrition on cardiac and systemic homeostasis. The purpose of this review is to present background on nutritional assessment and to discuss the effects of malnutrition and nutritional repletion on both myocardial morphology and cardiac and systemic physiology.

**Nutritional Assessment**

It is probable that malnutrition, with its systemic and myocardial effects, originate from a subclinical state, and detection of subclinical malnutrition might signify patients at increased risk of morbidity and mortality. Routine nutritional parameters such as percentage of ideal body weight, triceps skin-fold thickness, mid-arm muscle circumference, percentage of body fat, serum albumin, and total lymphocyte count have not been shown to agree consistently with body composition measurements and are of little value in determining the nutritional status of the individual patient. Mullen and associates concluded that serum albumin, serum transferrin, delayed hypersensitivity, and triceps skin-fold thickness could be used to predict risk of postoperative morbidity and mortality in general surgical patients, but they warned that isolated parameters should be interpreted cautiously. Abel and coworkers, in a later prospective study, evaluated nine nutritional parameters (percentage of ideal body weight, triceps skin-fold thickness, mid-arm muscle circumference, percentage of body fat, serum albumin, serum transferrin, total lymphocyte count, delayed hypersensitivity, and right atrial myocardial glycogen concentration) in 100 consecutive cardiac surgical patients with respect to the development of postoperative morbidity and mortality. They concluded that routine nutritional assessment is of no value in guiding the nutritional management of individual patients, although as a group, statistical correlations could be made between morbidity and mortality and various parameters.

Albumin is the major plasma protein and appears to be an amino acid donor for extrahepatic protein synthesis. Sixty-five to 70 percent of albumin catabolism normally takes place peripherally, and the rate of peripheral catabolism increases as a result of injury. Rich and colleagues noted an increased risk of cardiac surgery in patients 75 to 90 years old with serum albumin concentrations of less than 3.5 g/dl and noted an associated 1.8-fold relative risk of postoperative infection as well as a 2.25-fold prolonged hospitalization. Pulmonary, gastrointestinal, renal, and mental disorders were also significantly increased. Albumin, however, does not appear to be an essential element for wound healing or tissue repair, as there are reports...
of a patient\textsuperscript{12} as well as rats\textsuperscript{13} with analbuminemia showing no impairment of growth or wound healing.

In a comparative study of four plasma proteins in malnutrition, albumin had the lowest sensitivity, transferrin an intermediate sensitivity, and thyroxin-binding prealbumin-retinol binding protein complex the highest sensitivity to alterations in nutritional status.\textsuperscript{14} Because the four- to eight-day half-life of transferrin is shorter than that of albumin, and because it equilibrates rapidly due to a relatively small extravascular store, it is thought to be more accurate than albumin in assessing protein status.\textsuperscript{15} However, the synthesis of transferrin appears to be submitted to contradictory feedback according to protein and/or iron deficiency, which partly invalidates it as a reliable test for estimating protein depletion. The components of the thyroxin-binding prealbumin-retinol binding protein complex respond together in a parallel direction to protein deficiency.\textsuperscript{16} While plasma albumin and transferrin fail to respond to short-term protein or energy restriction, prealbumin and retinol binding protein are very sensitive and respond rapidly to refeeding. Prealbumin is the most sensitive indicator of nutrition. Prealbumin and retinol binding protein levels serve as useful measures of protein repletion, with increases noted as early as five to seven days after institution of parenteral nutrition.\textsuperscript{17} This is apparently related to their rapid turnover rate as well as their unusual richness in tryptophan.\textsuperscript{18} Thus, prealbumin, retinol-binding protein, and transferrin can be used to detect subclinical malnutrition and monitor the effectiveness of dietary treatment,\textsuperscript{16-18} and they could be useful in detecting subclinical malnutrition in cardiac surgical patients.

However, a recent prospective study of 221 consecutive cardiac surgical patients found that 98.5 percent had prealbumin levels within the normal range, and 79.1 percent had normal retinol-binding protein levels.\textsuperscript{19} Furthermore, there was no statistical relationship between nutritional status, delayed hypersensitivity and perioperative immune response, and the development of postoperative morbidity and mortality.

**Nutritional Factors and Cardiac Physiology**

Cardiac failure and cyanosis complicate severe types of ischemic, valvular, and congenital heart disease. These lead to malnutrition and end-organ dysfunction, such as renal and hepatic impairment.\textsuperscript{20} Cardiac cachexia is a specific form of protein-calorie malnutrition and is a consequence of prolonged circulatory failure. Seen frequently in the early years of cardiac surgery, it is now much less common. It is more frequently seen in older patients with long-standing rheumatic valvular disease with its associated congestive heart failure and edema, or with end-stage cardiomyopathy seen in younger patients awaiting transplantation. The pathogenesis of cardiac cachexia as described by Pittman and Cohen\textsuperscript{21} is secondary to congestive failure and the resulting relative tissue hypoxia, anorexia and hypermetabolism, with wasting of muscle protein and adipose tissue. The effects of hypermetabolism and stress on protein utilization are demonstrated by excessive protein requirements.\textsuperscript{22} With the increased catabolism accompanying surgery, sepsis, or profound illness, severe depletion of the visceral proteins might occur prior to further anthropomorphic changes.\textsuperscript{3} Visceral proteins are imperative for enzymatic function, osmotic pressure differentials, host immune response, and wound healing. To maintain gluconeogenesis and the body's other critical protein-requiring mechanisms, the metabolism is shifted toward muscle protein catabolism.

Cachexia causes dyspnea, gastrointestinal hypomotility, decreased gastric capacity, and further decreases in appetite.\textsuperscript{23} Anorexia is further aggravated by unpalatable and restricted diets, low in fat and salt, as well as by the administration of digitalis, opiates, and diuretics. This cachexic situation gives rise to increased heart failure and development of a positive feedback loop that ultimately results in death if not corrected. Cardiac wall compliance might decrease as a result of interstitial wall edema.\textsuperscript{23} Splanchnic congestion leads to malabsorption and protein-losing enteropathy.\textsuperscript{1} Renal protein losses are exacerbated by increased renal venous pressures. With the dyspnea of congestive failure, the respiratory muscles can consume more than 25 percent of the total oxygen consumption.\textsuperscript{24}

Voit,\textsuperscript{25} in 1866, presented work on feline starvation, and found that the heart of the starved animal was only slightly smaller than that of the normal animal. He concluded that the heart was "spared" in malnutrition, a concept widely held until Keys et al\textsuperscript{9} presented data from a large autopsy series of starved persons showing that myocardial loss is proportionate to the losses from the rest of the body. Adequate nutrition is important in maintaining normal cardiac function. Thiamine deficiency produces a peripheral vasodilatation\textsuperscript{26} resulting in high-output congestive heart failure. Cardiac contractility is reduced with certain electrolyte deficiencies.\textsuperscript{27} Fatal cardiomyopathy resulting from selenium deficiency after long-term parenteral nutrition has been reported.\textsuperscript{28} Structural and functional myocardial impairment can result from protein-calorie malnutrition.\textsuperscript{29-31} Muller and Wollert\textsuperscript{32} reported on a 52-year-old woman with cardiac failure after double valve replacement that was refractory to maximal dosages of dopamine, dobutamine, and epinephrine. The administration of 1 g/kg/min glucose and 1.5 IU/kg/min insulin reversed this situation apparently due to a resynthesis of high-energy phosphates that improved the inotropic capacity of the heart.
The myocardium plays a major role in energy homeostasis undergoing glycolgenolysis and proteolysis similar to that of the liver and skeletal muscle. Myocardial atrophy has been documented in patients suffering from cardiac cachexia, anorexia nervosa, low-energy diets, and kwashiorkor. Keys and associates also showed that with loss of approximately 25 percent of body weight after six months of a low-energy, low-protein diet, all roentgenographic dimensions of the heart are markedly decreased, and that the calculated decrease in cardiac volume is 70 percent of the loss of body weight. Myocardial atrophy correlates with vacuolization of myocardial fibers, with a decrease in the size and sometimes fragmentation of myofibrils. Kuykendall et al showed that protein depletion produced a reduction in the total cardiac mass due to a decrease in nitrogen and glycogen content with a concomitant increase in fat content. The normal mass of the heart, but not of the liver, was restored by protein repletion.

Associated with the loss in myocardial mass is a proportionate decrease in cardiac output and stroke volume. However, stroke-volume index and cardiac index either remain the same or increase slightly due to the decrease in body size. In another study, however, the cardiac index in malnourished patients was significantly less than that of patients with normal nutritional states. Kyger et al found that protein malnutrition significantly decreased both left ventricular stroke work and stroke volume when left atrial pressure exceeded 10 cm H2O. Similarly, cardiac output and aortic blood flow were significantly reduced in the protein-depleted animal when left atrial pressure exceeded 25 cm H2O. Coronary artery blood flow and heart rate did not differ significantly, although the protein-depleted group was unable to match the increased heart rate developed in the control group at higher left atrial pressures. Therefore, severe protein-calorie malnutrition leads not only to a decreased baseline myocardial performance, but also to the inability of the heart to respond to increased metabolic demands placed on it.

As myocardial mass decreases, so does the ability to generate cardiac output. However, various compensatory mechanisms are generated. Blood pressure, heart rate, blood volume, and oxygen demand decrease in the malnourished. Plasma catecholamine levels are directly affected by fat and carbohydrate intake. A hypoadrenergic state in the fasting individual leads to an accompanying bradycardia. Keys and colleagues showed that heart rate declined steadily from 56 beats per minute to 35 beats per minute at the height of starvation. Low catecholamine levels, resulting in destimulation of the renin-angiotensin-aldosterone axis, might lead to a decrease in blood pressure through both a loss of sodium retention and vasodilatation. At the height of starvation, Keys and associates showed that the blood volume had decreased by nearly 9 percent and the venous pressure had fallen by more than 50 percent. Along with diminished catecholamines, there is impairment of deiodination of thyroxine to triiodothyronine, leading to further decreases in the metabolic rate and cardiac workload.

Malnutrition can also lead to disturbances in cardiac rhythm. A popular very low-calorie “liquid protein” diet led to about 60 sudden deaths in the approximately 100,000 Americans who followed it for at least one month. Frank and colleagues reported on 36 of these patients whom on average had lost 30 percent of their body weight over four months preceding death. Thirty of the 36 had preexisting illness, and the authors concluded that electrolyte or mineral deficiencies might have been a significant cause of many of the deaths. On the other hand, Isner and associates reported on 17 previously healthy obese patients who had lost a mean of 34 percent of their premorbid weight over two to six months, and seven were still greater than 20 percent over their ideal weight. Eleven of these 17 exhibited electrocardiographic evidence of ventricular tachycardia shortly before death. Eight had QT interval prolongation, suggesting a predisposition to arrhythmias due to delayed repolarization. There was evidence of myocardial atrophy with attenuation of myocardial fibers and excessive lipofuscin production at postmortem examination. Heart weight was diminished in proportion to body weight. The cause of the QT interval prolongation remains unclear.

Lean body mass has been shown to be an important predictor of survival with end-stage heart disease. Malnutrition is a major significant risk factor in major surgery. Morbidity and mortality are substantially greater in these cachectic patients than in normally nourished patients undergoing similar procedures. Abel and co-workers showed that outcome after cardiac surgery was much worse in malnourished patients. There was a 16 percent mortality in their group of 44 malnourished patients compared with none in a matched group of well-nourished controls. Morbidity consisting of respiratory failure, pneumonia, renal failure, and sternal wound complications was also more frequent. In a retrospective study, poor nutrition was found in 28 percent of a series of 60 consecutive patients undergoing heart valve replacement. Of those with poor nutrition, 58 percent developed postoperative complications, including mediastinitis, wound infection, ventricular arrhythmias, or secondary pulmonary infection, while postoperative complications developed in only 6 percent of patients with normal nutritional states. The duration of hospital stay was also significantly longer in patients with poor nutrition.
NUTRITIONAL REPLETION

If malnutrition is present, one could argue for preoperative nutritional repletion to decrease morbidity and mortality. Anergy is often a reversible state and the early aggressive use of nutritional repletion might be an integral part of its treatment. Spanier and coworkers showed that reconstitution of the total-body cellular mass by total parenteral nutrition is regularly followed by a return of normal delayed hypersensitivity response. In many patients, however, total parenteral nutrition given over two to three weeks does not correct the total-body cellular mass or the abnormal total exchangeable sodium to total exchangeable potassium ratio. In these patients, skin test reactivity also remains abnormal, and failure to correct protein-calorie malnutrition is the likely outcome. Forse and associates showed a 43 percent conversion rate from anergy to normal reactivity during a two-week interval of total parenteral nutrition with a simultaneous improvement in body composition. There is evidence that neutrophil bactericidal dysfunction is related to abnormal nutritional status and can be improved, with an associated increased survival, by nutritional supplementation.

Management of patients with protein-calorie malnutrition is based on controlling underlying congestive failure as well as providing an adequate amount of protein, calories, electrolytes, vitamins, and minerals to enhance protein synthesis. The enteral route is preferable to total parenteral nutrition. Nutrition can reverse starvation-induced myocardial atrophy, although routine postoperative parenteral nutrition proved to be of no benefit in one study. In a study of five chronically malnourished patients gaining 12 percent of their weight, heart size increased by 25 percent as assessed roentgenographically. In addition, by echocardiography, there was a 59 percent increase in diastolic volume, a 31 percent increase in left ventricular mass, and a 90 percent increase in cardiac output. With refeeding of carbohydrate and fat, there is reactivation of the renin-angiotensin-aldosterone system as well as release of catecholamines, leading to further increases in plasma volume and blood pressure. Nutritional repletion reverses the natriuresis of starvation. Refeeding leads to hypermetabolism from the hypometabolic state of starvation.

Nutritional supplementation of malnourished patients therefore reverses the compensatory factors of starvation and might increase the short-term potential for heart failure. Reversal of cardiac atrophy and cardiac output capability might not be able to meet the rapidly increasing demands. Cardiac failure with "refeeding edema" is well described after oral alimentation of starved patients. Children with protein-calorie malnutrition fed high-protein oral diets experience significant increases in their plasma volume,
and 20 percent will develop congestive heart failure.\textsuperscript{62} In these patients, it might be useful to supply a larger proportion of energy as fat rather than carbohydrate.\textsuperscript{1} However, another study found that enteral nutrition is safe treatment for malnutrition in congenital heart disease. Twelve of 13 patients with congenital heart disease given continuous enteral nutrition displayed normal growth, and cardiac function remained stable or improved in ten patients despite the water load (146 ± 22 ml/kg/day).\textsuperscript{63}

Two to three weeks of optimal nutritional support are required to produce clinically significant changes in both body composition and cardiac function, as well as to facilitate an appropriate metabolic response to injury and stress.\textsuperscript{67} Abel and coworkers\textsuperscript{68} found that preoperative cardiac cachexia in patients undergoing valve surgery results in a poor outcome, and that a short five-day course of immediate postoperative total parenteral nutrition is ineffective in reducing morbidity and mortality. Gibbons et al\textsuperscript{69} presented data from three of their multiple valve patients who were preoperatively repleted with orally administered parenteral nutritional formulas. Based on whole-body protein synthesis and turnover rates, they estimated that at least three weeks of preoperative nutritional repletion would be necessary to reverse the underlying malnutrition.

Flancbaum and associates\textsuperscript{64} studied the diagnosis-related groups (DRG) impact of nutritional support on 80 consecutive cardiac surgical patients. Preoperatively, six of the 80 patients were nutritionally depleted. The seven patients who received postoperative nutritional support were significantly older, had a significantly longer average length of stay in both total postoperative days as well as in the intensive care unit, and had a greater incidence of both septic complications and mortality. The authors concluded that nutritional support in cardiac surgical patients warrants special consideration in view of a significantly increased hospitalization and resource utilization as compared with other patients having cardiac surgery. One must question whether the nutritional support deserves consideration rather than the category of patient who might require it.

The role of nutritional repletion of cardiac surgical patients is unclear. Whether correction of abnormal cardiac hemodynamics is all that is required is also unknown. However, because of the substantial morbidity and mortality associated with malnutrition in this setting, we believe that preoperative correction of malnutrition is prudent. Most cardiac surgery is performed either urgently or emergently, so it is usually impossible for the surgeon to preoperatively replete malnourished patients.

This article was intended to increase the awareness of the importance of nutrition with respect to both the preoperative and postoperative courses of cardiac patients, so that correction of deficiencies can be instituted as soon as possible. We evaluate nutritionally suspect patients through determination of prealbumin, retinol-binding protein, and transferrin levels. We prefer to replete our patients by the enteral route if possible. We begin supplementation at approximately 1,200 kcal/day, aiming for a total caloric intake of 30 kcal/kg/day to be achieved over three to four days. Excessive calories are avoided to prevent hepatic steatosis and its complications. Up to 30 percent of calories are supplied as fat. However, we are concerned about recent evidence suggesting that omega-6 fatty acids, the main component in intravenous lipid emulsions, are immunosuppressive.\textsuperscript{65} If the intravenous route is required, cardiac formulations are used to prevent fluid overload.

In summary, the myocardium plays a major role in energy homeostasis resulting in a loss of myocardial mass and a proportionate decrease in cardiac output and stroke volume. Immune defects are also seen with protein-calorie deficiencies and have been associated with an increased morbidity and mortality. Nutrition can reverse both the myocardial changes and the immune defects, but routine postoperative parenteral nutrition is of questionable benefit in these patients. Nutritional supplementation also reverses the compensatory factors of starvation and might increase the short-term potential for heart failure.

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