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# Nutritional considerations in the surgical patient

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In the patient having dental surgery, there are several considerations that impact and rely on nutritional status. First is the consideration that surgery inflicts a controlled injury that begins a critical and lengthy process of wound healing. The process is complex, involving multiple biochemical and cellular mediators with the aim of restoring homeostasis, form, and function. The success of the body to heal itself is critically dependent on the extent of the injury and the presence of sufficient and continued nutrition. Many patients are increasingly cognizant of nutrition and health maintenance. Self-prescription of herbs, vitamins, and alternative medicines can result in drug interactions as well as alterations in homeostasis. The nondisclosure of these compounds secondary to the patient's hesitance, the patient's lack of knowledge of the potential side effects, or to the dentist's lack of inquiry about these herbs/supplements is the second nutritional consideration. The last consideration is related to the operative site that dentistry manages. The mouth is the portal for entry of food into the body; thus, disease of the oral and maxillofacial region and/or surgery of these structures may result in impaired food intake both prior to and after surgery. The severity and duration of impairment is dependent on the disease state and the surgical intervention required to correct it. The dentist must be cognizant of methods to ensure the patient has appropriate nutritional support. Systemic conditions (eg, diabetes mellitus) can have

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significant adverse effects from inappropriate dietary intake during the perioperative period.

## Wound healing

The physiology and biology of wound healing is integrated on multiple levels. The responses are aimed at maintaining homeostasis and recruitment of resources for rebuilding the injured organs. The responses have overlapping timelines and can be categorized as (1) neurohumoral, (2) endocrine, (3) cardiovascular, and (4) biochemical at the site of injury.

Neurohumoral mediators are released in response to the stress of surgery or injury, are stimulated by the sympathetic nervous system, and begin the fight-or-flight response. This response involves the release of catecholamines (epinephrine, norepinephrine), glucocorticoids, and glucagon. This primary initiating response stimulates the cardiovascular system to increase stroke volume and heart rate and increase systemic vascular resistance to maintain perfusion pressure. Glucose, the body's chief energy source, must be available to prevent protein catabolism and fatty acid mobilization. Glucose is provided from hepatic stores undergoing glycogenolysis. The liver typically has a 24-hour to 36-hour store of glycogen stored for periods of inadequate caloric intake and responds to these hormones by manufacturing and releasing stored glucose. The endocrine response to injury involves the release of corticotropin from the posterior pituitary to stimulate cortisol release from the adrenal glands. The rise of cortisol directly stimulates muscle catabolism and the subsequent proteolysis and release of amino acids. The amino acids are used for repair and synthesis of the injured tissues and the ongoing production of cytokines including acute-phase reactants (C-reactive protein, fibrinogen). In response to the catecholamines, insulin production is decreased. The extent of stress or injury will also affect the regulation of salt and total body water to maintain perfusion pressures. The posterior pituitary will modulate antidiuretic hormone upon sensing changes in serum osmolarity. The juxtaglomerular organ of the kidney releases renin to stimulate the adrenal gland into releasing aldosterone (the major salt-retention hormone) to conserve water.

The metabolic response to surgery can be considered to occur in three phases. The initial phase (adrenergic-cortisol) lasts 1 to 3 days and is followed by the withdrawal phase (adrenergic-cortisol withdrawal), typically lasting 1 to 3 days or lasting up to weeks, which is followed by the anabolic phase that could last months (Box 1). The precise physiologic role for the production of acute-phase reactants is not fully understood but it is thought that they are produced, in part, to counter the systemic effects of tissue damage and cytokine production. Many acute-phase reactants act as antiproteases and wound-healing factors [1].

Cytokines are similar to endocrine polypeptide messengers except they are produced by a diverse group of cell lines from keratinocytes, immune

**Box 1. Three phases of the metabolic response to injury**

1. Adrenergic-cortisol (ebb)
   Glycogenolysis, lipolysis—energy source for fight or flight
   Increase in specific plasma proteins secondary to injury, surgery, or inflammation (eg, fibrinogen, C-reactive protein)
   Converse decrease in the production of the steady-state proteins (eg, albumin)
2. Adrenergic-cortisol withdrawal (flow—catabolic)
   Increased metabolism, body temperature, pulse, nitrogen loss, and muscle breakdown (release of tumor necrosis factor-α, interleukin-1 and interleukin-6)
   Lasts several days to weeks depending on termination of initial inflammatory response
3. Convalescence—anabolic stage lasts for weeks or months

cells, and endothelial cells within the region of the wound. Cytokines act in a paracrine and autocrine fashion, setting up gradients according to the cell type and location within the wound. In certain disease and injury states, some of the cytokines act systemically, very much in an endocrine fashion (eg, rheumatoid arthritis or sepsis). Cytokines are a large family of intercellular messengers and play an extremely important role in the control and manipulation of the inflammatory wound–healing cascade. Ongoing research reveals new information every week about the roles that each of the following cytokines play. Within the cytokine family, a balance is achieved by those that upregulate and manipulate inflammation (interleukin [IL]-1, IL-6, IL-8, interferon-γ, tumor necrosis factor [TNF]-α) and those that downregulate or terminate the response (IL-4, IL-10) [1–4].

### *Inflammatory cytokines*

#### *TNF-α (cachectin)*

TNF-α is one of the most studied proinflammatory cytokines. It is known to be a major determinant of the sepsis (or septic) response to major injury. TNF-α production is initiated by macrophages to ramp up the wound-healing cascade and is also produced by monocytes, Kuppfer cells, and lymphocytes. TNF-α is a key stimulant of the immune cascade due to injury or infection (endotoxin is a potent stimulant). TNF-α stimulates recruitment and maturation of cells into the wound. It has been shown to upregulate the adhesion of molecules on the endothelium, invoking a more concentrated chemotactic response from neutrophils. It directly stimulates hemostasis, increases vascular permeability, and increases angiogenesis. TNF-α also circulates systemically and, coupled with IL-1 and IL-6, alters the metabolic

state to promote the availability of nutrients and acute-phase proteins by the liver [2–4].

*IL-1*

IL-1 has many effects similar to TNF-α and is produced by the macrophage-monocyte lineage and by keratinocytes at the site of injury. IL-1 induces endothelium to secrete other proinflammatory cytokines. In vivo, IL-1 has shown to cause fever, anorexia, hypozincemia, anorexia, and release of corticotropin. It also stimulates the hepatic production of acute-phase reactants and decreases albumin synthesis. In the fresh wound, it increases immune cell chemotaxis, neutrophil activity, and upregulation of endothelial-derived adhesion molecules. It has been shown to increase keratinocyte and fibroblast proliferation and increase collagen synthesis [2–4].

*IL-2*

IL-2 is produced by T lymphocytes and stimulates T-cell activation. Therefore, it is not present until late in the inflammatory cascade. The evidence for its role is mixed and it may actually be more important in the immunocompromised host [2–4].

*IL-6*

IL-6 is produced by many different cell types in response to a variety (IL-1, TNF-α, and endotoxins) of powerful agonists. Evidence shows that concentrations of IL-6 increase in wounds as the polymorphonuclear cell (PMN) count increases. IL-6 is a powerful stimulant for fibroblast maturation and proliferation, is a potent activator of B and T cells, and is the major determinant in the hepatic regulation of the acute-phase metabolic response.

Scar formation may be a result of the impaired control of IL-6 production. Fetal wound healing is remarkable for the lack of scar formation. This unique property has been linked to the lower levels of IL-6 [2–4].

*IL-8*

IL-8 is produced by macrophages and fibroblasts within the wound. IL-8 stimulates neutrophil and monocyte chemotaxis and its concentration is highest within 24 hours of injury. It has been shown that IL-8 is produced in higher concentrations by fibroblasts from patients with psoriasis but conversely, similar to IL-6, it is found in diminished concentrations in fetal wounds. This would suggest a link between IL-8 and the hyperkeratoses of psoriasis [2–4].

*Interferon-γ*

Interferon-γ is secreted by T lymphocytes and macrophages. In autocrine fashion, it activates macrophages and PMN, increasing their cytotoxicity. Interferon-γ is believed to play a significant role in the remodeling of tissues

because it reduces wound contraction by retarding collagen production, decreasing cross-linking, and stimulating the production of collagenase. Experimental data have shown interferon-γ to impair re-epithelialization and decreased wound strength [2–4].

*Counter-inflammatory cytokines*

*IL-4*

IL-4 is produced by T lymphocytes, mast cells, and basophils. IL-4 predominantly causes B-lymphocyte proliferation, increases immunoglobulin E–mediated immunity, and inhibits the macrophage-derived cytokines TNF-α, IL-1, and IL-6. Within wounds, it stimulates fibroblast growth and collagen synthesis [2–4].

*IL-10*

IL-10 is produced by macrophages and T lymphocytes and acts in a paracrine manner to inhibit the production of TNF-A, IL-1, and IL-6 [2–4].

*Growth factors*

This diverse collection of polypeptide proteins functions to stimulate the maturation and proliferation of cells involved in healing the wound. These proteins function very much like the cytokines discussed previously.

*Platelet-derived growth factor*

This protein is initially released from the alpha granules of platelets when stimulated by injury or thrombotic events. It stimulates the activation and recruitment of immune cells and fibroblasts. After the initial release by platelets, macrophages maintain the secretion, stimulating collagen and proteoglycan synthesis.

Platelet-derived growth factor occurs as three isomers: AA, AB, BB. Each of the isomers has a different level of activity in stimulating wound healing. The BB isomer is now approved by the Food and Drug Administration for application in impaired wounds [4].

*Transforming growth factor* β

Transforming growth factor β occurs as three isomers: $\beta_1$, $\beta_2$, and $\beta_3$, which bind to three receptors (types I, II, and III). The transforming growth factor β series of chemokines are produced within the wound by platelets, macrophages, and fibroblasts. Transforming growth factor β causes the production of the extracellular matrix by stimulating the deposition of collagen and proteoglycans and by inhibiting protease activity [2,4].

*Fibroblast growth factor*

Fibroblast growth factor stimulates the proliferation and migration of fibroblasts and keratinocytes when released into the wound by macrophages

and endothelial cells. It prevents wound contraction, promotes collagen remodeling, and is essential to angiogenesis [2,4].

*Epidermal growth factor*

Epidermal growth factor is released by keratinocytes and functions in an autocrine manner to direct epthelialization. It stimulates fibroblast collagenase secretion and assists in the remodeling of the wound [2,4].

Much research continues to elucidate the role of growth factors and cytokines in healing. The incredibly complex and intertwined wound-healing cascade remains a significant challenge to researchers. No single substance has thus far been discovered that can be exploited for healing chronic wounds in all instances. The goal to gain exquisite control and manipulation over wound healing requires further research.

## Nutrition and wound healing

Patients require both an energy and a protein source. Carbohydrates, proteins, and fatty acids are sources of energy. Carbohydrates are the body's chief energy source. Glucose is the energy source of vital organs such as the brain and nervous tissue. The body requires an adequate supply of carbohydrates to prevent protein catabolism and fatty acid mobilization. Alternatively, excess glucose or hyperglycemia is not beneficial. Hyperglycemia results in decreased leukocyte function, in dehydration, and in metabolic acidosis.

Dietary fats are also an energy source for the body. Fat reserves provide a much greater reserve than that provided by glycogen stores. The body cannot synthesize certain fatty acids and must obtain these in the diet. These essential fatty acids are necessary for the synthesis of arachidonic acid, which is the precursor for prostaglandins and leukotrienes. The lack of these essential fatty acids has the potential to alter the immune response of the patient.

Wound healing requires energy. Thus, the nutritional status of the patient affects wound healing. Although protein can serve as an energy source, its primary purpose is for cellular proliferation and protein synthesis; any use of protein as an energy source is considered detrimental. Much of maxillofacial surgery is dependent on bone healing. Protein has been shown to be important in the strength of fracture repair [5]. Patients who are severely malnourished demonstrate delayed wound healing attributed to impaired protein metabolism, delayed angiogenesis, and impaired wound contraction. The malnourished patient also has an increased susceptibility to infections. This is secondary to a decrease in circulating T and B lymphocytes and impaired neutrophil phagocytosis. All of the above can result in a prolonged rehabilitative period.

Vitamins and minerals are also important in wound healing. Vitamin A is essential for epithelialization, collagen synthesis and cross-linking, and fibroblast differentiation. Vitamin A has been shown to reverse the

inhibitory effects of glucocorticoids on the inflammatory phase of healing [6]. Vitamin C is a cofactor in the hydroxylation of lysine and proline in collagen synthesis. A deficiency of vitamin C is associated with impaired wound healing and collagen synthesis. There are reports that suggest that perioperative administration of vitamin C promotes healing. Vitamin D and calcium are essential for the healing of hard tissues, and a deficit of either nutrient can contribute to poor healing of osteotomies or poor fracture repair. Vitamin E is a lipid soluble antioxidant. Antioxidants are thought to reduce damage from free oxygen radicals that are reduced during wound healing. There is a perception that vitamin E improves the cosmetic outcome of healed wounds but this perception is not supported in the literature. Indeed, there is evidence to suggest that large doses of vitamin E inhibit wound healing. Vitamin K is important in the activation of several clotting factors. A deficiency can result in a coagulopathy with persistent bleeding or hematoma formation. Vitamin K deficiency may be secondary to prolonged antibiotic usage. It is evident that the lack of these nutrients can potentially impact wound healing. Nutritional intake is dependent on several variables, one of which is taste. Deficiencies in zinc and protein impede taste bud regeneration [7]. Such a deficiency can compound the diminished sense of taste and smell that occurs as the body ages.

When a patient's energy requirements increase due to surgery, trauma, or other medical illness, caloric intake needs to increase. When there is insufficient caloric intake, the body undergoes catabolism to provide the necessary energy and substrates for maintaining the body. In the early phases of the catabolic response, muscle is the primary source of energy and proteins for homeostasis. As catabolism progresses, the major organs become targets of the ongoing catabolism, with subsequent loss of mass and structure.

Table 1 depicts the metabolic differences and similarities between simple starvation and major injury. The simple starvation (fasting and bed rest)

Table 1
Metabolic differences and similarities between simple starvation and major injury

| | Simple starvation | Major injury |
|---|---|---|
| Basal metabolic rate | No change | + |
| Acute-phase reactants | No change | +++ |
| Major fuel | fat | Mixed |
| Ketone production | +++ | +/− |
| Hepatic ureagenesis | + | +++ |
| Gluconeogenesis | + | +++ |
| Negative nitrogen balance | + | +++ |
| Muscle proteolysis | + | +++ |
| Hepatic protein synthesis | + | +++ |

+ or − = intensity or degree of change.

*Adapted from* Souba WW, Austen WG. Nutrition and metabolism in surgery: scientific principles and practice. 2nd edition. In: Greenfield, et al, editors. Lippincott-Raven; 1997. p. 44–9; with permission.

metabolic response has been equated to minor elective operations. A major injury like a burn can significantly increase the metabolic requirements and can severely deplete the body's energy and protein stores. Clinical nutrition requirements for various stressed conditions are listed in Table 2.

## Evaluation of nutritional status

Traditionally, evaluation of the nutritional status of patients has relied on a history and physical examination, measurement of weight, anthropometrics, and serum markers. Much of the sensitivity and specificity of some these measures has shown to be poor in predictive value.

The history and physical examination remains a vital source of information regarding a patient's ability to undergo and recover from the stress of surgery. Questions regarding specific food groups, use of supplements, ability to chew and swallow, dyspepsia, and bowel habits are important in developing a plan for maintaining nutritional intake [8,9].

The oldest method used to evaluate nutritional status is comparing body weight with usual or ideal body weight using the Hamwi formula:

Men: ideal weight = 106 lb + 6 lb for every inch over 5 ft

Women: ideal weight = 100 lb + 5 lb for every inch over 5 ft

Acute changes in body weight do not accurately reflect proportional changes in nutritional status or the proportions of protein, fat, or body water. The body mass index is a somewhat different measure, using body weight and the patient's height (weight divided by height squared). When the body mass index is less than 15 $kg/m^2$, there is significant increase in morbidity for patients undergoing major operations. The body mass index has drawbacks when it is related to overweight patients [8]. Not withstanding the issues previously discussed, it has been generally accepted for some time that patients who have had unintentional weight loss of greater than 10% to 12% in the proceeding 3 to 6 months will have a higher incidence of morbidity with major operations. Accordingly, a weight loss of

Table 2
Clinical nutrition requirements[a] in various stressed conditions

| State | kcal/d |
|---|---|
| Basal | 1450 |
| Postoperative (uncomplicated) | 1500–1700 |
| Sepsis | 2000–2400 |
| Multitrauma | 2200–2600 |
| Major burn | 2500–2600 |

[a] Daily average caloric needs of a 70 kg man.

*Adapted from* Souba WW, Austen WG. Nutrition and metabolism in surgery: scientific principles and practice. 2nd edition. In: Greenfield, et al, editors. Lippincott-Raven; 1997. p. 44–9; with permission.

less than 10% is considered mild, 10% to 20% is considered moderate, and over 20% is considered severe [10].

Using weight as the sole marker of nutritional status is inadequate, and even using traditional anthropometric measurements may not be sensitive to acute changes in nutrition (eg, arm muscle circumference, tricep skin-fold thickness, and creatinine height index, which requires a meat-free diet, normal renal function, and the patient to not be in a catabolic state).

These measures are limited by intraobserver and interobserver variation and right/left arm dominance. These measures may also not adequately reflect mean muscle mass in elderly patients and cannot be used to assess the acutely ill patient [8,10].

Simpler tests more indicative of the patient's current nutritional status measure muscle function using hand grip dynamometry and maximal inspiratory pressure and correlate well with predicting postoperative complications. These tests are underutilized [8,10].

### *Circulating serum proteins*

Traditionally, albumin, prealbumin, and transferrin have been used to assess the visceral protein status. These proteins are synthesized by the liver and are best used as complementary markers and not as exclusive determinants of nutritional depletion.

Albumin is the major protein produced by the liver and has a normal range of 3.5 g/dL to 4.5 g/dL. It functions as the major protein carrier and preserves intravascular oncotic pressure. Albumin has a half-life of 20 days and poorly reflects acute changes in nutritional intake or status. The degree of hypoalbuminemia, however, is the best serum marker to correlate with the 30-day mortality rate in hospitalized patients [9,10].

Transferrin is the major iron transport protein and has a circulating half-life of 8 days. It is synthesized by the liver and has a normal range of 200 mg/dL to 260 mg/dL. Iron metabolism and nutritional status affect its production. Theoretically, transferrin should be a better marker than albumin for nutritional depletion, but this has not been proven in studies [10].

Prealbumin, also synthesized by the liver, ranges from 15 mg/dL to 25 mg/dL. It transports thyroxine and retinol-binding protein and has a half-life of 2 to 3 days. Nutritional depletion is reflected by changes in prealbumin sooner than by changes in albumin. Even though changes in nutrition intake can be seen as early as 7 days in the prealbumin levels, fewer studies have validated its usefulness over albumin to predict outcome [10].

## Herbal medicines and the surgical patient

There has been a tremendous interest in alternative and complementary medicine. Self-prescription of herbs, vitamins, and alternative medicines can

result in drug interactions and in alterations in homeostasis. The nondisclosure of these compounds secondary to the patient's hesitance to disclose, the patient's lack of knowledge of the potential effects these compounds may have, or the dentist's lack of inquiry about these compounds may contribute to adverse effects.

A primary surgical concern is hemostasis. Table 3 lists herbs and supplements that when combined with anesthetic, analgesic, or anticoagulant drugs may have an effect on either the coagulation cascade or platelet aggregation.

## Surgical considerations

The dentist can be confronted with numerous situations that require dietary management. Patients must first be assessed for baseline nutritional status and then assessed for the impact that dental/medical treatment will have on their nutritional status. Most dental patients are not nutritionally depleted at baseline. The patient with a pericoronitis may have impaired dietary intake; however, the severity and duration of the deficit does not profoundly impact on wound healing or the homeostatic condition of the patient. If this patient has trismus or an associated facial space infection, then the severity of the dietary deficit may be more profound. Even in these situations, the most profound clinical finding is a fluid deficit with resultant volume depletion. The well-nourished individual can tolerate a starvation period of up to 10 days without adverse effects. Thus, this patient is best managed with an understanding of fluid hydration as opposed to nutritional supplementation.

### *Dentoalveolar surgery*

Traditionally, patients presenting for dentoalveolar surgery, including those for third molars on an outpatient basis, undergo routine abbreviated history and physical examinaitons and, if necessary, an anesthesia evaluation and indicated laboratory analyses. It is uncommon that an evaluation of nutritional status is performed on outpatients. The oral and maxillofacial surgery and dental literature is sparsely populated with studies aimed at the nutritional status of such patients.

A study evaluating third-molar surgery patients and postsurgical nutrition found that there was a 40% reduction in caloric intake present 1 week after surgery [11]. Unfortunately, this study did not have a large cohort and relied strictly on dietary analyses. No serum markers or anthropometric measures were used other than body weight.

White et al [12] performed an outcome study on recovery after third-molar surgery. This study, involving 737 patients, assessed several variables including interference with chewing and consuming a regular diet. On average, by postoperative day 5, the patients in this study reported "little" or "no" interference with chewing or consuming a regular diet [12].

Table 3
Herbs and dietary supplements and their potential effects

| Herb or dietary supplement | Potential prothrombinopenic components | Platelet aggregation inhibitors | Fibrin formation inhibitors |
|---|---|---|---|
| Andrographic plant | | X | |
| Baikal skullcap root | X | | |
| Bilberry fruit | | X | |
| Black currant seed oil | | X | |
| Bladderwrack | | X | X |
| Borage seed oil | | X | |
| Bromelain from pineapple fruit and stem | | X | X |
| Cayenne fruit | | X | X |
| Celery plant | | X | |
| Chamomile, German flowers | | X | |
| Clove oil | | X | |
| Coleus roots | | X | |
| Da huang, Chinese rhubarb root | | X | |
| Dan shen root | | X | |
| Dandelion root | | X | |
| Dang shen | | X | |
| Devil's claw | | X | |
| Dong quai root | | X | |
| Erigeron plant | | X | |
| Evening primrose seed oil | | X | |
| Feverfew plant | | X | |
| Fish oil | | X | |
| Garlic bulbs | | X | X |
| Ginger rhizome | | X | |
| Ginkgo leaves | | X | |
| Ginseng root | | X | X |
| Green tea | | X | |
| Hawthorn leaf and flower | | X | |
| Horse chestnut bark | | X | |
| Huang qi | | X | |
| Kava kava root | | X | |
| Licorice root | | X | |
| Onion plant | | X | |
| Papain from papaya leaves and unripe fruit | | X | |
| Pau d'arco bark | X | | |
| Reishi mushrooms | | X | |
| Sha shen | | X | |
| Shinpi bark | | X | |
| Sweet birch oil | | X | |
| Sweet clover plant | X | | |
| Tonka bean seeds | X | | |
| Turmeric root | | X | |
| Vitamin E | | X | |
| Wintergreen leaf oil | | X | |
| Wood ear mushrooms | | X | |
| Woodruff plant | X | | |

*Adapted from* Norred CL, Brinker F. Potential coagulation effects of preoperative complementary and alternative medicines. Altern Ther Health Med 2001;7:58–67; with permission.

Dietary considerations must also be appreciated in the patient who is having surgical procedures other than third-molar surgery. Surgery causes pain and swelling. A partially edentulous patient may not be able to wear a prosthesis for a period of time after the placement of implants, secondary to soft-tissue swelling. A patient, after multiple extractions and the placement of an immediate denture, needs to develop the oral neuromuscular skills to accommodate the appliance. A nutritionally complete liquid diet for the first days following surgery and gradual transition to pureed and then soft diet over a period of days or weeks will allow the patient to learn how to manipulate the dentures and will diminish tissue ulceration during the immediate post-treatment period [13].

*Orthognathic surgery*

Orthognathic surgery creates a more profound insult to the body. Therefore, it is anticipated that the patient may sustain a greater effect on nutritional abilities during the perioperative period. A pilot study involving orthognathic patients and nutritional supplementation was well designed but suffered from a very small sample size [14]. The study population all received a typical pureed diet. The subjects were then randomized to a control group and an experimental group that received a specialized nutritional supplement that provided 1.5 cal/mL (19.7% protein, 32% fat, 53.3% carbohydrate). The results revealed that the average weight of the patients in the control group moved into the mild-deficit realm up to 6 weeks postoperatively. The addition of a high-energy supplement to the postoperative diet in the experimental group resulted in better nutritional intake and maintenance of body weight. The midarm muscle circumference decreased to moderate deficit at 3 weeks in the control group. The analysis of visceral protein markers—albumin, transferrin, and total lymphocyte count—remained essentially unchanged over 6 weeks in both groups. These results are not startling for those who perform these surgeries. Not documented by these authors were adverse affects associated with the changes in the control group. No report of surgical site infections or wound dehiscence was made. Although not discussed directly by the authors, the patient population undergoing orthognathic surgery is invariably motivated and in relatively good health and nutritional status.

Patients may be placed into maxillomandibular fixation for several weeks after orthognathic or trauma surgery. Maxillomandibular fixation presents significant issues in maintaining sufficient caloric intake. The perioral pain and swelling makes the first week to 10 days an uncomfortable time period. Coupled with the loss of textured food, the pureed/liquid diet can be a hindrance to adequate caloric intake.

The shift to rigid fixation in oral and maxillofacial surgery has resulted in patients returning sooner to a more normal diet. These patients are also not restricted to pureed diets for as long. One study (a pilot) in the recent

literature compared nutritional status in patients undergoing maxillomandibular fixation versus those with rigid fixation [15]. This pilot study showed a significant weight difference by 6 weeks. A study from 1984 found that patients with internal fixation lost less weight and regained it sooner than patients with maxillomandibular fixation [16].

*Trauma surgery*

Nutritional management of a patient with an infection or a traumatic injury presents a different situation due to the unexpected nature of the malady. There is obviously no time or provision for optimizing nutritional status a priori. The severity of the injury and associated injuries may impair the patient's ability to adequately meet dietary requirements. Although the healthy well-nourished patient may tolerate a period of starvation up to 10 days, the stress and hypermetabolism of the patient with critical injuries or disease will have a diminished reserve (between 5 and 7 days). Nutritional supplementation in the patient with severe head injury (which may accompany maxillofacial injuries) has been shown to decrease morbidity. It is best that nutritional replacement should be achieved by 7 days [17]. A higher incidence of complications in patients with weight loss $\geq$10% with some evidence of physiologic impairment is consistent with the need to initiate early nutritional support [9].

*Cancer patient*

Malnutrition is a typical consequence of head and neck malignancies. The malnutrition may be secondary to associated habits such as alcoholism, as well as the patient's impaired ability to masticate and swallow. These patients may present with a moderate-to-significant weight loss, protein deficiency, and/or vitamin deficiencies. As discussed previously, these conditions can contribute to surgical morbidity. There may be benefit to having nutritional support prior to surgical, chemotherapeutic, or radiation intervention [3,18,19].

Eating is a fundamental pleasure. Treatment of head and neck cancer may result in significant changes that have a profound impact on patients' quality of life in regard to normal eating and their ability to satisfy their nutritional requirements. The surgical resection can result in a significant defect that can compromise the patient's ability to eat or swallow. The dentist, using both prosthodontic and surgical techniques, is an integral part of the rehabilitative team, having the ability to restore normal form and function.

Chemotherapy and/or radiation therapy produce changes that are unique to these modalities. Oral mucositis occurs during the actual treatment phase of chemotherapy or radiation therapy. Mucositis can cause severe pain and will impact on the patient's ability to eat and swallow. Changes in taste may also occur during both chemotherapy or radiation therapy. These changes

are generally not permanent, but there have been reports of persistent hypogeusia after these treatment modalities. Xerostomia is another finding associated with chemotherapy and radiation treatment. It generally resolves after chemotherapy but is persistent after radiation therapy. The dentist may be consulted to manage these conditions.

## Nutritional support

There are several modes of nutritional support that are commonly used in the ambulatory patient during the perioperative period: clear-liquid diet, full-liquid diet, pureed diet, mechanical soft diet, and regular diet. A clear-liquid diet is often used during the immediate postoperative period after a parenteral sedation or general anesthetic. The patient is frequently advanced to a more complete diet after demonstrating the ability to tolerate the diet without gastrointestinal complications. The patient may alternatively be maintained on a clear-liquid diet for the first 24 to 48 hours after a surgery to allow initial wound healing to occur. The diet is low in nutrients.

A full-liquid diet is more nutritionally complete. This diet is good for those patients who cannot chew or swallow foods. Full-liquid diets can frequently consist of milk products. Consumption of milk products requires lactase. Lactase deficiency prevents the hydolysis and absorption of lactose within the small bowel. This can potentially result in abdominal cramping, bloating, flatulence, nausea, and diarrhea. The dentist should be aware that there is a percentage of the population that is lactase deficient: Askenzem Jews, 60%; adult blacks, 20%; and almost all adult Asians [20]. Nutritional products, such as Sustacal (Mead-Johnson Evansville, IN) and Ensure (Ross Labs, Columbus, OH) are frequently part of a full-liquid diet. Products that are soy based are generally more palatable when cold and do not contain milk products.

A pureed or mechanical soft diet is generally a transitional diet. These diets provide the patient with increased consistency and food texture. A pureed diet can be administered to the patient in maxillomandibular fixation. The mechanical soft diet requires minimal chewing and can be tolerated by the patient who has oral pain and limited opening.

Box 2 is an example of dietary instructions for the denture patient.

### *Enteral versus parenteral feeding*

#### *Enteral feeding*

The last several years have seen a shift in the importance of the gut in maintaining homeostasis in the multitrauma or sick patient. As discussed previously, it is understood that patients have a limited nutritional reserve and will have a compromised outcome if nutritional support is not initiated within a specific time. Evidence has shown advantages to maintaining enteral feedings. Enteral feedings keep the gut active, preventing loss of

**Box 2**

**Diet for the first day after insertion: full-liquid diet**

- Bread/cereal group—gruels cooked in milk or water
- Vegetable group—juices, thin soups
- Fruit group—juices, blended drinks and shakes
- Milk group—milk, cheese soup, nutritionally complete supplement drinks such as Ensure, Carnation Instant Breakfast (Nestle, Glendale, CA), Sustacal
- Meat group—pasteurized eggnogs, meat broths or soups, pureed meats

**Diet for the second and third days after denture insertion: pureed diet to soft diet**

- Bread/cereal group—cooked cereals (oatmeal, Cream of Wheat [Kraft Foods, North field, IL], and so forth), milk, toast and softened bread, barley, small pastas, puffed rice
- Vegetable group—juices, well-cooked carrots, green beans, mashed potatoes, creamed vegetables, soups, V-8 (Campbells, Camden, NJ)
- Fruit group—well-cooked fruits (no seeds), canned fruits, juices, drinks
- Meat group—scrambled or soft-boiled eggs; thick, strained bean/pea soups like lentil soup; chopped beef; ground liver, chicken or beef in a sauce

**Diet for the fourth day and later: soft diet to regular diet as tolerated**

- By the fourth day or as soon as most sore spots have healed, in addition to the soft diet, firmer foods can be eaten. Most of the time, it is best to cut food into small pieces before attempting to eat it.
- The ultimate success with dentures is being able to manage sandwiches and salads of raw fruit and vegetables at some point in time.

mucosal integrity and the phenomenon of bacterial translocation. In a patient who is unable to eat, nutritional intake may be administered by way of tubes that are inserted into the lower gastrointestinal system. There are potential inherent risks associated with such feeding; however, with the new, more pliable silicone-based nasogastric tubes, the risks (eg, nasopharyngeal irritation, mucosal ulceration) have been reduced. Nasogastric or

orogastric tube feeding may only be used for a short period. For long-term enteral feeding, the patient may have a surgically inserted tube. This format encompasses a variety of techniques to pass liquid nutrition directly into various parts of the gastrointestinal tract (eg, percutaneous enterogastrostomy, duodonestomy, and jejunostomy). Regardless of the location of tube placement, these patients need to be monitored for the development of delayed gastrointestinal emptying, which may be associated with gastric regurgitation and pulmonary aspiration, ileus, diarrhea, and electrolyte abnormalities.

*Total parenteral nutrition*

Total parental nutrition is not the panacea it was expected to be when it was first introduced in the early 1970s. The general surgery and oral and maxillofacial surgery literature reveals a high incidence of complications and side effects. The major complication is catheter-related infections. The use of total parental nutrition in patients with strictly oral and maxillofacial surgery or dental needs is very limited to those with head and neck cancer operations, the multitrauma patient, or patients without a functioning gastrointestinal tract. Total parental nutrition needs to be administered to the central venous compartment (eg, internal jugular, subclavian, or inferior vena cava) due to the high tonicity of the solutions. The solutions contain dextrose and amino acids, with or without electrolytes. The associated lipid emulsions that are coadministered are derived from safflower or soybean oils. A lipid emulsion containing linoleic acid is mandatory to prevent essential fatty acid deficiency. Complications associated with the placement of central venous access include hemo/pneumo thorax, venous thrombosis, and air embolism. Potential complications associated with this nutritional regimen include dehydration and serum electrolyte abnormalities. The use of total parental nutrition requires a mandatory regimen of laboratory blood work for as long as the patient is receiving total parental nutrition in order to avoid electrolyte and hydration imbalances [21].

*Fluid hydration*

Patients who are undergoing a surgical procedure or who are ill may present with a fluid deficit. This may be secondary to preoperative fasting instructions or the inability to eat. The inability to eat may be secondary to an associated oral ailment (eg, trismus) or an unrelated condition due to hospitalization. For minor surgery in which the patient will be able to consume fluids immediately after the surgery, fluid deficit is not a major concern. For the patient who cannot resume oral intake or has diminished oral intake during the postoperative period, it is important to intravenously hydrate the patient. Even for minor dentoalveolar surgery, however, patients who have been hydrated report feeling better during the immediate postoperative period [22].

Fluid administration requires an understanding of fluid compartmentalization. Total body water exists in two major compartments: intracellular

and extracellular. The intracellular compartment represents approximately two thirds of the total body water; the extracellular compartment represents about one third. The extracellular compartment is further subdivided into intravascular (one fourth of extracellular) and extravascular fluid (three fourths of extracellular) compartments. Between these compartments, there is a constant redistribution of free water that is dictated by ionic and osmotic pressure. The primary extracellular electrolytes are sodium and chloride. These electrolytes are not freely diffusible between compartments. Any alteration in the concentration of these electrolytes in a specific compartment will result in a compensatory shift of free water.

Several crystalloid fluids are available. A physiologic choice for typical ambulatory surgery is either 0.45% (half-normal) or 0.9% (normal) saline with or without dextrose. This salt concentration is appropriate for the deficit in the adult patient. In addition, the fluid will remain in the extracelluar compartment and compensate for some of the hemodynamic changes that occur with surgery and anesthesia.

The following formula is used to determine the hourly infusion rate:

For the first 10 kg of body weight: Administer 4 mL/kg
For the second 10 kg of body weight: Administer 2 mL/kg
For each additional kilogram of body weight: Administer 1 mL/kg

For a 70 kg male, using this formula would result in a rate of 110 mL/hour.

First 10 kg: $10 \times 4 = 40$ mL/hour

Second 10 kg: $10 \times 2 = 20$ mL/hour

Each additional kilogram: (70 kg–20 kg) = 50 kg; $50 \times 1 = 50$ mL/hour

## Medical considerations

### *Diabetes mellitus*

It is estimated that greater than 5% of the United States population has diabetes. Diabetes effects many organs, and management of the diabetic patient requires that the dentist be cognizant of these end-organ effects. Sarasin and Westlund [23] provide important information on this topic.

Diabetes results from a deficiency of insulin or a lack of sensitivity to insulin, resulting in hyperglycemia. The medical management of diabetes consists of the administration of oral agents and/or insulin to correct for the insulin deficiency or insensitivity. If diabetic patients alters their dietary intake or are stressed by a dental or surgical procedure, then the balance achieved by the administration of the medication is disrupted. Surgical stress results in a state of hyperglycemia. There are significant risks associated with hyperglycemia and, therefore, patients must continue their medicine throughout the treatment period. The adverse effects secondary to

hyperglycemia are relatively slow to develop. Alternatively, hypoglycemia can result in an acute emergency.

Dental treatment can interfere with patients taking their medication and with patient ability to keep a normal diet. There are a number of reasons why patients may alter their eating patterns prior to dental treatment. Many patients want to give the impression that they maintain good oral hygiene. These patients may opt not to eat prior to coming to the dentist. Patients may be anxious and have a diminished desire to eat. Patients who are scheduled for sedation are usually instructed not to eat anything after midnight to minimize the risk of aspiration. Dental surgical procedures can impair the patient's ability to eat post treatment, which may be secondary to pain. Alternatively, the actual surgical procedure may change a patient's occlusion, impacting the ability to masticate (eg, full-mouth extraction). Analgesics are frequently prescribed to manage postoperative pain. The opioid-based agents may cause nausea and vomiting. These side effects can be compounded by swallowed blood from the oral cavity.

The dentist needs to be aware of the common manifestations of hypoglycemia: sweating, palpitations, anxiety, agitation, confusion, dizziness, and weakness. If there is any concern that the patient is hypoglycemic, then the dentist should have a glucometer and be able to check blood glucose levels while the patient is in the office. Postoperatively, the dentist needs to provide dietary instructions and, in consultation with the patient's physician, establish a mechanism for following the patient's blood glucose levels. Most diabetic patients are aware of their disease and will be able to understand the importance of ensuring an adequate postoperative diet. Nausea, vomiting, and pain, however, may effect the patient's ability to eat. The long-acting effect of medications used to manage diabetes (Table 4) and a reduced food intake can result in mortality.

### *Anorexia nervosa (eating disorders)*

Eating disorders are under-recognized as an etiology for malnutrition and affect more women than men. The prevalence of anorexia nervosa in women aged 16 to 25 years is estimated to be about 1%. This condition is seen in all major American ethnic groups including African American,

Table 4
Insulin preparations

| Action | Type of insulin | Onset (H) | Peak (H) | Duration (H) |
|---|---|---|---|---|
| Rapid | Lispro | 0.25–0.5 | 1–2 | 3–5 |
| | Aspart | 0.25–0.5 | 1–2 | 3–5 |
| Short | Regular | 0.5–1 | 2–4 | 6–8 |
| Intermediate | NPH | 4–6 | 8–12 | 18–30 |
| Long | Ultralente | 4–6 | 8–12 | 18–30 |
| | Glargine | 1 | None | 30+ |

*Abbreviation:* NPH, Neutral protein hagedom.

Asian, and Hispanic [24]. One recently published article suggested that dental surgery may contribute to the relapse of anorexia [25]. Medications such as the selective serotonin reuptake inhibitor Paxil (Paroxetine) may be used in the management of anorexia. There are significant systemic problems associated with anorexia nervosa (Table 5) that contribute to the mortality rate of 10% to 15%.

Electrolyte abnormalities that occur with anorexia nervosa can cause cardiac arrhythmias. Dental treatment may even potentiate the electrolyte abnormalities in selected situations. For example, hyperventilation (occurring in the anxious patient) and an increase in epinephrine (both endogenous and exogenous) decrease potassium plasma levels, increasing the risk of cardiac arrhythmia. Acetaminophen with codeine/hydrocodone/oxycodone are commonly prescribed for postoperative analgesia. The opioid component of these drugs is a prodrug and must be converted to the active drug. The enzyme that is necessary for this conversion is antagonized by several selective serotonin reuptake inhibitors used in the management of anorexia nervosa. Therefore, consideration must be given to using alternative analgesics.

### *Obesity*

Obesity is a disease that is increasing in prevalence. It is associated with numerous systemic abnormalities with which the dentist must be familiar prior to implementing care. The American Heart Association has classified obesity as a major risk factor for coronary artery disease. Obese individuals are prone to hypertension, myocardial ischemia and infarct, congestive heart disease, and sudden death.

The initial patient interaction begins with obtaining an accurate history. Inquiry should be made about dieting habits and medications that may have been used to facilitate weight loss. Although not currently prescribed, a patient who has used the drug combination fenfluramine-phentermine may have undiagnosed valvular heart disease. The currently prescribed diet medication Meridia (Sibutramine) interacts with meperidine. The administration of meperidine to the patient taking Meridia is contraindicated.

Table 5
Systemic problems associated with anorexia nervosa

| Problem | Result |
|---|---|
| Mitral valve prolapse | The reduction of left ventricular mass causes mismatch for valvular function |
| Cardiac arrythmias | Possible death (particularly with prolonged QT) |
| Refeeding syndrome | The sudden return to normal eating overloads cardiac system, resulting in cardiac overload and failure |
| Electrolyte imbalances | Hypomagnesemia, hypokalemia, hypocalcemia |
| Endocrine abnormalities | Amenorrhea, hypercholesteremia, osteoporosis |

QT; electrocardiogram finding pertaining to duration from Q-wave to T-wave.

Obstructive sleep apnea is an abnormality associated with sleep fragmentation, intermittent hypoxia, intermittent hypopnea, and apnea, which results in excessive daytime somnolence. Patients are also at risk for the development of pulmonary hypertension and cardiac dysrhythmias. Obesity is a causative factor associated with obstructive sleep apnea. The dentist should be suspicious of obstructive sleep apnea in the obese patient who provides a history of snoring and/or daytime somnolence. Administration of sedative as well as analgesic medications in the obstructive sleep apnea patient may have pronounced effects, compromising the patient's respiration. Opioids are not absolutely contraindicated, but consideration should be given to using nonopioid analgesics. In addition, medications that are administered may be stored in the fat and have a prolonged residual effect. Long-acting local anesthetics (eg, bupivicaine) can minimize the need for analgesics during the acute postoperative period. Pre-emptively administering an analgesic prior to the termination of the local anesthetic is also advantageous and will both minimize the intensity and duration of the post-treatment pain.

The physical size of these patients may create difficulty in positioning them in the dental chair. Additional support may be required. Traditional supine positioning has potential adverse effects by allowing the patient's weight to unfavorably displace the diaphragm into the thoracic cavity, reducing the functional residual capacity. This reduction compromises respiratory reserve in a patient who may already have a respiratory impairment. Such positioning in the patient with a history of gastroesophageal reflux will also aggravate the passive regurgitation. These factors may be of more significance if the dentist plans on sedating the patient.

## Summary

The nutritional status of the patient is integral to the care of the patient. Nutritional assessment and management begins with the initial patient contact and continues into the postoperative period.

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