Nutrition 32 (2016) 498–502

Contents lists available at ScienceDirect

Nutrition

journal homepage: www.nutritionjrnl.com

Occasional article

# Special postoperative diet orders: Irrational, obsolete, and imprudent

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ARTICLE INFO

*Article history:*
Received 28 October 2015
Accepted 29 October 2015

*Keywords:*
Low sodium diet
Low-fat/low-cholesterol diet
Special diets
Postoperative clear liquid diet

ABSTRACT

There are no indications to prescribed special diets for postoperative patients. Low-sodium and low-fat or low-cholesterol diets are examples of restricted diets, especially in patients with heart disease and atherosclerosis. These restricted diets are unpalatable. Postoperative nausea, paralytic ileus, and vomiting caused by residual anesthetic effects and opioids used for pain control further contribute to the problem. Long-term adherence to these diets is necessary to derive benefits. Prescribing regular and palatable diets in the immediate postoperative period to meet protein and energy goals is important for wound healing and is commensurate with best clinical practices. In the following, we review the pertinent literature and offer clinical evidence that routine special diet orders for postoperative patients are not necessary.

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## Introduction

Special diets are prescribed for postoperative patients. A common practice is to automatically prescribe clear liquid diets when indications are unclear [1]. Even more worrisome is the practice of prescribing low-sodium (Na) and low-fat or low-cholesterol diets to postoperative patients with known cardiac-related diseases. Patients are often told to avoid caffeinated beverages to prevent cardiac arrhythmias. These approaches, once prescribed based on intuitive clinical thinking, currently have no scientific validity. These antiquated diets are so restrictive that patients do not receive sufficient calories, protein, and critical nutrients [2], becoming an independent risk factor for increased hospital length of stay (LOS) [3]. Prescribing regular and palatable diets during the immediate postoperative period facilitates achieving sufficient protein and energy intake required for wound healing. There are no adverse outcomes by following this approach. In this article, we present the results of our literature review and offer evidence that routine special diet orders for postoperative patients are unnecessary, antiquated, and not in keeping with best practice standards.

## Sodium-restricted diet in cardiac disease

Research does not support the practice of Na-restricted diets for patients with congestive heart failure. Tested interventions examined daily Na intakes ranging from 230 to 5750 mg [4]. In the perceived absence of clear evidence, most physicians continue to follow the now obsolete practice guidelines for a heart-healthy diet with a mean daily Na intake of 2 to 2.4 g [5,6]. The benefits of a 3 g Na diet are associated with a longer event-free survival but only in patients with advanced heart failure [7].

Data show that Na restriction is harmful in heart failure [8–10] due to its detrimental effects caused by the activation of antidiuretic and antinatriuretic systems [11]. A randomized controlled study on hospitalized patients with refractory heart failure compared a group of patients who received hypertonic (3% NaCl) saline with patients who did not; both groups received diuretics. The hypertonic saline group had a shorter LOS and improvement in hemodynamic and other clinical variables.

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http://dx.doi.org/10.1016/j.nut.2015.10.017
0899-9007/Published by Elsevier Inc.

Long-term benefits occurred at 48 mo with lower mortality in the hypertonic saline group [12]. The current evidence clearly indicates the need to increase Na intake in heart failure patients, rather than decrease it.

In critically ill patients with acute decompensated heart failure, aggressive Na and water restriction had no effect on clinical stability at 3 d and is associated with significant increase in thirst [13]. Evidence has emerged from the Health ABC (Health, Aging and Body Composition) study that in older adults, Na intake was not associated with 10-y mortality, cardiovascular disease (CVD), or heart failure [14].

## Sodium-restricted diet for hypertension

Na intake is an independent risk factor for hypertension, primarily in communities where the daily Na intake is >2.3 g (or 100 mEq). Reducing dietary salt intake to 2.3 g lowers blood pressure (BP) in hypertensive individuals [15]. Salt sensitivity, or the responsiveness of BP to salt intake, varies from person to person [16–18]. One may expect BP to increase immediately in response to acute increase in Na intake, but this does not happen even in the same individual tested at different times. Although normal saline infusions (NS; 0.9% NaCl) are commonly used when resuscitating a patient with hypotension, the converse practice of intentional Na restriction to decrease BP is not practiced. Postoperative patients often are on intravenous fluids, commonly NS, containing 154 mEq of Na. Just 1 L of NS contains 9000 mg of NaCl or 3537 mg (~3.5 g) of elemental Na already above the daily limit of 2.3 g recommended by the Centers for Disease Control and Prevention [19]. Further restricting oral intake of Na in these situations is unjustified.

## Information regarding Na requirement and intake

The daily requirement for Na is 80 to 120 mEq (~5 g of table salt, or NaCl). One of the difficulties clinicians face is adjusting Na intake in grams (or milligrams), when the laboratory measurement is reported as mEq/L. One g or 1000 mg of elemental Na contains 43.5 mEq of elemental Na (1000 divided by 23, the atomic weight of Na). However, the Na content in salt, the common source of additional Na in the diet, is calculated differently due to the chloride ion in the molecule. One g of NaCl, or table salt, contains only 393 mg or 17.5 mEq of elemental Na. An understanding of the Na that patients receive both parenterally and enterally, and knowing how to convert the units from mg to mEq or vice versa, is essential to avoid dysnatremia. Both hypernatremia and hyponatremia are associated with increased risk for in-hospital death in critically ill patients [20].

## Low-fat or cholesterol-free diets for patients with atherosclerosis and coronary artery disease

The pathophysiological processes of atherosclerosis are complex and the importance of genetic susceptibility is recognized as an equally important, if not more important, component of the many contributing etiologies [21]. High levels of low-density lipoprotein cholesterol (LDL–C) and low levels of high-density lipoprotein cholesterol are major risk factors for atherosclerosis, in addition to endothelial dysfunction and various markers of inflammation. In addition to lifestyle changes, dietary modifications are important for primary and secondary prevention of atherosclerosis. A 2015 systematic review and meta-analysis that included 15 randomized trials concluded that decreasing dietary saturated fat intake does indeed reduce the risk for cardiovascular events (relative risk, 0.83; 95% confidence interval, 0.72–0.96), especially when replaced with polyunsaturated fats [22]. Consumption of trans-fatty acids, which raise LDL-C, should be kept to the minimal. However, there have been several major recent changes in our understanding of the relationship of diet to heart disease. A 2006study reported results from the Women's Health Initiative Randomized Controlled Dietary Modification Trial involving ~19,500 women assigned to follow a low-fat diet compared with ~29,300 women who continued their usual diets [23]. Only modest reduction (38% to 29%) of fat intake in the study group was achieved, but there was no protection against CVD. A meta-analysis of cohort studies also found no association between dietary saturated fats and CVD [24]. The earlier recommendation to replace saturated fats with polyunsaturated fatty acids was questioned in a recent meta-analysis of 76 studies [25].

Dietary cholesterol does raise the total serum cholesterol level but is far less important than saturated fat [26]. It is a common practice to instruct patients with heart disease to avoid eggs altogether, although this practice has no scientific validity. Despite good compliance with low-fat diets, the anticipated decrease in serum cholesterol levels was not achieved in a study of 126 male patients [27]. A meta-analysis published in 2013 involving >474 000 participants showed that there was no association between egg consumption and risk for coronary heart disease [28]. Dietary guidelines suggest minor emphasis on reducing cholesterol intake from eggs [29].

However, the process of atherosclerosis leading to major vascular occlusive diseases is very slow, starting in childhood as just fatty streaks in the endothelium of blood vessels, and progressing as the individual gets older. In an autopsy study involving ~2900 men and women ages 15 to 34 on noncardiac causes, all had fatty streaks in the aorta [30]. Advanced coronary artery atherosclerosis has been noted in 2% of men ages 15 to 34 y who died of noncardiac causes [31]. One in six teenagers living in the United States has abnormal intimal thickening using intracoronary ultrasound [32]. However, it is only in the fourth, fifth, or sixth decades that advanced lesions occur, as seen by intimal thickening, fibrosis, ruptured plaques, or calcifications [33]. In short, atherosclerosis takes decades to progress to a point of causing adverse events.

There is no justification to prescribe a low-fat or low-cholesterol diet to a postoperative patient even with known atherosclerosis-related conditions. Short-term consumption of diets that are not considered "heart-friendly" will have no adverse consequences on this lifelong process.

## Low-fat diet for patients with gallbladder and liver diseases

Many clinicians continue to foster patients' beliefs that fatty foods must be avoided once a diagnosis of gallbladder or liver disease has been made and continue this incorrect advice during the postoperative period. Intolerance to foods rich in fat is a common symptom in these conditions but this is not specific. Cholelithiasis during pregnancy often disappears after normal diet is resumed during the postpartum period [34]. Biliary sludge that may form in patients on total parenteral nutrition disappears with normal diet [35]. Although long-term dietary patterns may have some relationship to the formation of gallstones, low-fat diets do not prevent gastrointestinal symptoms in patients scheduled for cholecystectomy [36]. A patient-centric approach is recommended, which includes the avoidance of specific foods if they cause discomfort. There is no