Lifestyle and dietary modification for prevention of heart failure

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The preponderance of heart failure (HF), both systolic and diastolic, is related to long-standing risk factors and illness [1], which are in turn substantially engendered or mitigated by lifestyle practices. Systolic failure of the left ventricle may ensue from valvular heart disease, pulmonary hypertension and cor pulmonale, alcohol-induced cardiomyopathy, and a variety of conditions relatively uncommon in the United States, such as infectious myocarditis. On a population basis, however, systolic left ventricular failure is a sequela of ischemic heart disease [2]. Thus, the primary prevention of systolic HF derives largely from the prevention of coronary disease, an effort in which diet and lifestyle are of paramount value. The critical role of pharmacotherapy in secondary and tertiary prevention [3] is largely beyond the scope of this discussion.

Diastolic left ventricular failure or dysfunction typically develops in the aftermath of long-standing hypertension [2]. Other common antecedents include obesity, nephropathy, and diabetes mellitus [4]. These conditions are frequently intermingled in the insulin resistance, or metabolic, syndrome, a condition affecting as many as 50 million adults in the United States [5]. The principal causes of diastolic HF are risk factors for ischemic heart disease as well. Thus, neither the occurrence nor efforts to prevent one variety of HF is fully distinct from the other.

Increasing attention is being directed to asymptomatic ventricular dysfunction as a precursor to clinically overt HF [6,7]. During this precursor period, pharmacotherapy with such drug classes as angiotensin-converting enzyme inhibitors and β- and α-blockers (eg, carvedilol) is apt to be indicated in state-of-the-art prevention efforts [8,9]. Such late-stage prevention...
warrants attention to lifestyle, namely diet and physical activity patterns, as well. However, the greatest value of lifestyle interventions pertains to early mitigation of risk and the primary prevention of ventricular dysfunction.

**Systolic heart failure: diet in the primary prevention of systolic left ventricular failure**

Strategies for the primary prevention of left ventricular systolic failure are substantially confluent with those for the prevention of ischemic heart disease [10,11]. Assuming the avoidance of such toxic exposures as tobacco smoke, diet and physical activity is of principal interest. Other considerations of clear relevance, such as the reduction of psychological stress, treatment of mood disorders, or the avoidance of social isolation, are beyond the scope of this discussion.

Diet influences the pathogenesis of coronary artery disease in a variety of ways. The initial development of fatty streaks in coronary arteries is mediated by serum lipid levels, as well as by free radical oxidation, both of which are modified by nutrients. Progression of coronary lesions is affected by serum lipids, hypertension, hyperinsulinemia, oxidation, and possibly obesity, all of which are mediated by both macro- and micronutrient intake. Once coronary artery atherosclerosis is established, diet plays a role in determining the progression of plaque deposition and the reactivity of the endothelium, both of which may be predictive of cardiac events. Dietary manipulations have been shown to modify all of the known, modifiable coronary risk factors [12], and when extreme dietary measures are implemented, to induce regression of established lesions [13,14]. The role of diet in the management of coronary disease and risk factors is determined by the efficacy of dietary interventions and their complementarity to pharmacologic interventions of proven benefit.

Numerous studies have suggested an association between total fat intake and atherogenesis, both within and among populations. Investigative work over recent years, however, has been focused increasingly on the contribution of specific dietary fats to the atherogenic process. The relative cardiovascular benefits of total fat restriction versus modifying diet to promote mono-unsaturated and polyunsaturated fat intake relative to saturated and trans fat intake is of increasing interest [15,16].

Excess dietary fat produces predictable serum cholesterol and lipoprotein elevations (Hegsted and Keys equations; see [17]), which translate into predictable increases in the risk for cardiac events [18]. Dietary guidelines in the United States [19] have been based largely on evidence linking diet to heart disease. The principal mechanism by which fat and possibly cholesterol ingestion translates into increased cardiovascular risk is the induced elevation of serum lipoproteins, especially low-density lipoprotein (LDL). Elevations of LDL result in saturation of the receptor-mediated uptake by hepatocytes [20,21] and the consequent uptake of LDL by tissue-fixed macrophages.
This process of so-called “foam cell” formation is accelerated by the oxidation of LDL. The deposition of foam cells in the coronary intima and media induces smooth muscle cell hyperplasia and the growth of obstructing lesions [22,23].

In addition to the chronic effects of fat intake on atherogenesis, there is recent evidence that the acute ingestion of a high-fat meal may represent a cardiac stressor [24], and an interest in postprandial atherogenesis dates back to at least the 1970s [25]. Although the postprandial rise in triglycerides may contribute to the progression of coronary atherosclerosis, the magnitude of lipid changes seems insufficient to explain the observed increase in events; there are a variety of concomitant metabolic responses [26]. The acute ingestion particularly, perhaps exclusively, of saturated or trans fat may destabilize coronary plaque and impair endothelial function [24,27]. Evidence is rapidly increasing that endothelial function is a fundamental index of cardiac risk and that it is modified in response to a variety of nutritional influences.

Saturated fatty acids, those with no carbon–carbon double bonds, in particular raise total cholesterol and LDL. Foods rich in saturated fatty acids include the flesh of most domestic mammals raised for human consumption, dairy products, and several vegetable oils, notably coconut, palm, and palm kernel oils. The evidence that excessive intake of saturated fat, specifically C_{14:0}, myristic, and C_{16:0}, palmitic acid, raises serum lipids and promotes atherogenesis is decisive. Evidence linking diets high in saturated fats to cardiovascular events is convincing but is limited by difficulties in conducting long-term studies requiring assignment of subjects to dietary interventions. Current recommendations [28] call for reducing the intake of saturated fat to 10% or less of caloric intake (National Cholesterol Education Program) and ideally to less than 7% to 8%. The average US adult intake of these fats is approximately 13% to 14%. Prehistoric adaptations may be informative; Paleolithic intake of saturated fat was approximately 5% of calories [29].

In counseling patients to modify intake of saturated fat, a consideration of all sources of such fat in the diet is essential. The prevailing notion that dietary fat, particularly saturated fat, derives predominantly from red meat is only partly true. The primary source of dietary fat and saturated fat in the diets of American men is red meat; however, in the diets of American children it is milk, and in the diets of American women it is a combination of dairy products, including cheese, and processed foods [30,31]. When counseling subjects in an effort to reduce total and saturated fat intake, a reasonably detailed dietary history is essential. The contribution to total fat intake of often overlooked and unreported constituents of diet such as sauces, dressings, and spreads can be substantial [31]. The assertion by a patient that they are eating a low-fat diet because they have reduced or eliminated red meat is unreliable.

Modern food preparation techniques have greatly increased human exposure to trans-fatty acids, which occur naturally in small quantities in
milk. Trans fats are produced commercially by bombarding partially unsaturated fatty acids (ie, fatty acids with some preserved carbon–carbon double bonds) with hydrogen. The hydrogenation process saturates most of the double bonds in polyunsaturated fats. The trans isomeric configuration about the remaining double bond results in molecules that pack tightly together, limiting the fluidity of the fat, and producing a higher melting point. The stability of these fats at room temperature results in products that retain shape (eg, margarine in stick form as opposed to liquid vegetable oil) and increases product shelf life. Although they are advantageous to the food industry, trans fats influence serum lipids similarly to and perhaps even more adversely than saturated fats [32]. Many processed foods contain trans fats; they can be detected on labels by looking for “partially hydrogenated” before an oil is listed. Recent legislation will require the labeling of trans fat content of packaged foods by 2006 (http://www.fda.gov/OHRMS/DOCKETS/98fr/03-17525.htm).

Polyunsaturated fatty acid (PUFA) intake is generally believed not contribute to atherogenesis; however, the distribution of fats within this class may be of particular importance to human health, specifically in the ratio of n-6 to n-3 fatty acids. An extensive literature has developed a link between high intakes of n-3 polyunsaturates, particularly from marine sources, to low rates of heart disease [33–36]. Evidence to date suggests that increased consumption of n-3 fatty acids may lower cardiovascular risk; an anti-arrhythmic benefit in high-risk patients is conclusive [37]. Although n-6 polyunsaturates are readily available in commonly consumed vegetable oils, including soybean, safflower, sunflower, and corn, n-3 fatty acids are less widely distributed. Oils rich in n-3 fatty acid include flaxseed, linseed, marine oils, and, to a lesser degree, canola oil [35]. Fat-restricted diets may result in a relative, if not overt, deficiency of n-3 fatty acid intake, as well as a less than optimal intake of monounsaturated fatty acids (MUFA) [38–41]. A diet rich in n-3 fatty acid has been linked to reduced levels of serum triglycerides and platelet aggregation, and lower blood pressure.

The effects of monounsaturated fats and certain polyunsaturates, especially n-3 fatty acid, on cardiovascular health may be sufficiently beneficial that an intake of total fat in excess of 30% of calories is desirable, provided the fat is predominantly of these types [16]. Of note to primary care providers is that either the recommended reduction in total fat intake or the consumption of predominantly monounsaturated fat and n-3 polyunsaturated fat both represent significant dietary changes for most patients seen in the United States [42,43].

The cardioprotective effects of monounsaturated fatty acids have come to light largely through cross-cultural epidemiologic studies. Rates of heart disease are low in populations with high consumption of monounsaturated fat, even when total fat intake is consequently high, leading to an interest in the so-called “Mediterranean diet” [44]. Evidence suggests that the apparent neutral effects of monounsaturated fats on serum cholesterol are the result of
reductions in LDL, and concomitant elevations of high-density lipoprotein, both of which reduce cardiovascular risk [45–47]. A meta-analysis [48] in 1995, however, reported that monounsaturated fats lower high-density lipoprotein. Perhaps of greater importance is the apparent link between monounsaturated fat intake and inhibition of LDL oxidation [49].

Monounsaturated fats are abundant in traditional diets of the countries bordering the Mediterranean Sea. The Mediterranean diet, consisting of abundant fresh fruit and vegetables, olives, olive oil, wine, fish, and grains, particularly wheat in the form of pasta, has received increasing attention as a means of lowering cardiovascular risk [50–52]. Various aspects of this diet may contribute to cardioprotective properties. As discussed earlier, consuming n-3 polyunsaturated fat in fish may favorably affect serum lipids and inhibit platelet aggregation. Alcohol favorably influences serum lipids and raises endogenous tissue plasminogen activator [53]. Fruit and vegetable consumption is likely to be cardioprotective by a variety of mechanisms, as is consumption of grains, seeds, and certain nuts [54]. A variety of nuts and seeds are rich in monounsaturated fat, including walnuts, almonds, peanuts, and sesame seeds. The olive and avocado, both fruits, are also excellent sources of monounsaturated fat.

The relative contribution of dietary cholesterol to serum lipids is, to some extent, confounded by the highly correlated distribution of saturated fat and cholesterol in the diet. The meat of domestic mammals, dairy products, and organ meats is rich in nutrients and associated with elevated serum lipids. The contribution of cholesterol from eggs to the risk of heart disease is somewhat uncertain, because high-cholesterol diets are often high in total fat, and there is some evidence that egg consumption is unrelated to cardiovascular risk [55,56]. Shell fish, although relatively high in cholesterol content, are low in total and saturated fat and are not convincingly linked to an increase in cardiovascular risk. The recommended intake of cholesterol is up to 300 mg/d, with intake restricted to below 200 mg for patients with hyperlipidemia or established coronary disease. To comply with this recommendation, patients must eliminate or minimize their intake of egg yolks and restrict their intake of red meat, deli meats, cheese, whole milk, and milk products.

The direct role of total caloric intake in cardiovascular disease is somewhat less clear than the role of dietary fat. When caloric expenditure is high, caloric intake is believed not to represent a cardiac risk factor. Caloric intake in excess of caloric expenditure results in weight gain, and obesity is clearly associated with heart disease risk and systolic and diastolic HF.

Although the nutrients responsible for the health promoting properties of fruits and vegetables are a source of ongoing investigation and controversy, the cardioprotective influence of fruit and vegetable intake is decisive. Population-based studies consistently demonstrate health benefits of high fruit and vegetable intake [57]. This dietary pattern is strongly associated with a reduced risk of cancer, as well. The constituents of fruit that contribute to a cardioprotective effect include a variety of antioxidant micronutrients,
essential micronutrients such as vitamins and minerals, and soluble and insoluble fiber. Cereal grains have been associated with a reduced risk of both cancer and cardiovascular disease [58–61]. The intake of soluble fiber, particularly, appears to have cardiovascular benefits attributable to a hypolipidemic effect [62]. Establishing a causal relationship between specific food types and cardiovascular risk is confounded by the foods that are replaced; for example, diets high in fruits, vegetables, and cereal products tend to be low in fat, and some of the apparent benefits may derive from fat restriction [63]. The evidence for specific nutrient effects is less convincing than evidence for the effect of a produce-rich dietary pattern. Population-based studies suggest that vegetarianism is associated with less than average cardiovascular risk in developed countries.

On a population basis, separating the effects of soluble and insoluble fiber, fruit, vegetable, cereal, and fat intake is complicated by the tendency of dietary behaviors to cluster [64,65]. Diets low in fat tend to be relatively high in fiber of both types, and vice versa. Nonetheless, convincing epidemiologic associations exist between low-fat, predominantly vegetarian diets and the MUFA-rich Mediterranean diet and a low incidence of cardiovascular events [66].

To date, opinions remain divided between total fat restriction or the optimal distribution of fats as the best means of reducing the incidence and risk of coronary disease with diet [67–69]. The prehistoric human diet apparently provided approximately 20% to 25% of calories from fat, with approximately 5% from saturated and naturally occurring trans fat and the remainder from a combination of MUFA and PUFA. The n-6 to n-3 PUFA ratio, which is approximately 11 to 1 in US and western European diets, was between 4:1 and 1:1 for our ancestors [29]. Until or unless intervention studies further elucidate the optimally cardioprotective diet, recommendations consistent with current trial evidence, epidemiologic data, and what is known about the native nutritional habitat of Homo sapiens would seem prudent [70].

On the basis of such evidence and prevailing judgment [71,72], saturated and trans fat should be restricted to below 7% of total calories in all cardiac patients and is appropriate primary prevention in willing patients. Intake of fish, nuts, soy, olives, avocados, seeds, olive oil, and canola and linseed oil should be encouraged to raise n-3 PUFA and MUFA intake. Dietary fat and cholesterol reduction is best achieved by restricting intake of red meats, deli meats, whole-fat dairy products, especially cheese, cheese- and cream-based sauces and dressings, fatty spreads, and processed foods with more than trivial amounts of partially hydrogenated oils. Particular attention to detail is necessary to prevent substituting lipid-raising fats from one source for fats from other sources.

Optimal management of dietary fat intake appears capable of lowering LDL by as much as 20% and total cholesterol by as much as 30%, although lesser reductions are usually seen. Although dietary manipulation produces
benefits other than lowering lipid levels, more aggressive lipid-lowering measures are indicated for virtually all hyperlipidemic patients with coronary disease. Statin class drugs can lower LDL by up to 60%; the effects of these agents are enhanced by dietary therapy. Recent evidence suggests that dedicating an array of dietary strategies for lipid lowering, including high intake of soluble (viscous) fiber and plant sterols, can produce effects comparable to those seen with pharmacotherapy [73].

**Diastolic heart failure: diet in the primary prevention of diastolic heart failure**

The principal, modifiable causes of left ventricular diastolic dysfunction and failure are hypertension, obesity, and diabetes mellitus [11]. Each of these is a risk factor for atherosclerotic coronary disease and, thus, for systolic left ventricular failure, as well. The prevention of diabetes and obesity through diet and lifestyle is closely related and represents a means of preventing both systolic and diastolic HF, as discussed later. The prevention and control of hypertension through lifestyle is particularly germane to the prevention of diastolic HF [74] and is addressed here.

There has long been epidemiologic evidence of variations in blood pressure among diverse populations; hypertension is unusually prevalent in the United States, with approximately 50 million cases out of a population of some 265 million. Although some of this effect has been attributed to dietary factors, demonstrating causality has been precluded by the multitude of potentially confounding variables.

Migration studies have been conducted to help account for potential covariates of dietary change. Hypertension, like hyperlipidemia, is more prevalent in Asians living in the United States than in their nonemigrating counterparts [75], and similar effects of migration have been reported in other populations [76]. Similarly, although African Americans have particularly high rates of hypertension, the rate is low among native Africans living in rural settings and intermediate in Africans exposed to some aspects of Western lifestyle [77].

Among populations in the United States, hypertension is less common among the lean than the overweight and among vegetarians than among the general population [78]. Isolating the direct effects of diet on blood pressure is difficult because of the prevalence of obesity in the United States and the strong association between obesity and hypertension. From a practical perspective, however, patients benefit comparably from dietary interventions that lower blood pressure directly or indirectly as a result of weight loss. There is decisive evidence that even modest weight loss among obese, hypertensive subjects frequently results in blood pressure reduction [79].

The role of dietary sodium in the causes of hypertension has been of particular interest for some time. Data from multiple sources, including the INTERSALT trial [80], suggest that a high intake of sodium may be shifting
population blood pressure mean levels upward. The DASH-Sodium trial [81] provides evidence that restricting sodium to 1200 mg/d (one half the prevailing guideline in the United States) significantly lowers blood pressure among adults in the short term, at least, and further suggests that the relationship between sodium and blood pressure is dose-responsive over a considerable range.

Generally, diets associated with optimal control of blood pressure are similar to diets associated with a variety of other salutary effects. Vegetarianism is associated with lower average blood pressure, as is the Mediterranean diet, and the low-fat diet typical of the nonindustrialized far East [78]. The DASH trials have been instrumental in confirming the association between overall dietary pattern, with or without sodium restriction, and blood pressure [81–84]. DASH [85] findings indicate that a predominantly plant-based diet rich in vegetables, fruit, and grains, restricted in total fat, and generous in nonfat dairy effectively lowers blood pressure, with the possibility of particular benefit in African Americans. The DISC study [84] suggests that the relationship between diet and blood pressure in children is similar to that in adults.

As in the prevention and modification of other cardiovascular risk factors, the optimal diet for management of incipient and established hypertension is uncertain. Other avenues of research suggest that restricting total fat may be less beneficial than selectively restricting saturated and trans fat, while liberalizing the intake of monounsaturated fat and, particularly, n-3 polyunsaturates [34,87]. Recommendations for calorie control, abundant intake of fruit and vegetables, and restriction of saturated and trans fat intake may be made with confidence. Of note, such a diet is naturally rich in the micronutrients associated with lowering blood pressure, relatively rich in fiber, and relatively low in sodium. Which of these modifications in dietary behavior is responsible for blood pressure control is important to advance our understanding but not prerequisite to offering recommendations likely to benefit patients.

Although stage I hypertension has been effectively treated with diet in studies, two caveats should be noted. First, the compliance in a controlled trial is generally greater than in practice [88]. Second, more advanced hypertension has not been shown to respond to dietary management in the absence of pharmacotherapy. One suitable approach in efforts at managing more significant hypertension with lifestyle modification is to initiate pharmacotherapy as indicated and then to taper medications once the blood pressure is well controlled and evidence accrues that the patient is engaged in recommended dietary and lifestyle modifications.

**Specific nutrients**

Although there is decisive evidence that sodium contributes to the elevation of blood pressure [89,90], the causal role of sodium in hypertension
is less well established. Studies [91] suggest that only 10% of hypertensive subjects in the United States are sodium-responsive, demonstrating blood pressure variation with change in sodium intake. The efficacy of sodium restriction in the management of hypertension is difficult to demonstrate because it is difficult to achieve patient compliance with low-sodium diets [92] and because such diets almost inevitably introduce other changes, as well. Cook et al [93] suggest that the effect of salt restriction on blood pressure has been consistently underestimated.

Despite uncertainties, recommendations for restricting sodium to below prevailing levels in the United States can be made with considerable confidence. Intake in the United States generally exceeds the recommended limit of 2400 to 3000 mg/d. Our ancestral intake, which may indicate optimal levels, was approximately 700 to 800 mg/d, less than one fourth the average intake today [29].

Advocating a health-promoting diet will result in sodium restriction by reducing the intake of highly processed and “fast” foods. Patients should be advised of the importance of reading food labels. The sodium content of many commercial breakfast cereals is comparable to potato chips and pretzels, although the taste of salt in such products is masked by sugar. Attempting to limit sodium intake, many patients will report not using a salt shaker at all, but the salt added to food during preparation is less readily tasted than the salt shaken on just as the food is eaten. Therefore, preparation of low-salt foods and continued, albeit controlled, use of a salt shaker may be a preferred approach. As with other dietary changes, salt restriction becomes less objectionable as it becomes familiar [94]. Although the salt content of many processed foods goes unnoticed by most consumers, those acclimated to a lower sodium diet begin to taste salt more readily and to prefer lower intake levels [95,96]; acclimation to a high-salt diet has the opposite effect [97]. So-called “salt substitutes,” which replace some of the sodium with potassium or calcium, may serve as a useful aid to patients struggling to acclimate to a salt-restricted diet.

Diets rich in potassium tend to be relatively low in sodium, and vice versa, making the study of isolated dietary potassium difficult. Nonetheless, there is convincing evidence that potassium supplementation has a blood pressure-lowering effect [98]. The evidence is decisive that total dietary modification that results in increased potassium intake, and particularly in a potassium intake that exceeds sodium intake, lowers blood pressure [82]. The average intake of sodium in the United States is approximately 4000 mg/d, whereas the average daily intake of potassium is approximately 2500 to 3400 mg [94]. The INTERSALT study [80] found that blood pressure rose with age in all populations consuming more sodium than potassium but not in those consuming more potassium than sodium.

There is suggestive evidence that high dietary calcium intake contributes to lowering blood pressure. Calcium is considered a potentially important mediator of the hypotensive effects of nonfat dairy products seen in the
DASH trial [82]. A meta-analysis suggests that calcium, either in the diet or as a supplement, has a modest antihypertensive effect [99]. On the basis of an extensive literature review, however, the Canadian Hypertension Society [100] has advised against calcium supplementation as a means of either treating or preventing hypertension. The isolated effects of calcium supplementation on blood pressure appear to be modest; a dietary pattern providing abundant calcium may be of greater benefit. In the aggregate, evidence supports a hypotensive benefit of calcium intake at levels advisable on other grounds [101].

Diets rich in potassium tend to be similarly rich in magnesium. Magnesium supplementation may be beneficial in the treatment of hypertension in magnesium-deficient patients, but evidence of treatment benefit is inconclusive [100]. Evidence of modest treatment benefit has been reported [102]. Although magnesium supplementation is not advocated for the control of hypertension in the United States, a dietary pattern providing abundant magnesium is [103].

A potential benefit of dietary fiber in the regulation of blood pressure has been reported in both adults [104,105] and children [86]. The isolated effects of dietary fiber on blood pressure remain uncertain [106], because the data available to date are largely from observational studies, and the dietary pattern associated with lowering blood pressure is naturally high in both soluble and insoluble fiber. Keenan et al [107] have, however, published pilot data suggesting a hypotensive effect of oats, which the group ascribes primarily to the content of soluble fiber.

Patients with blood pressure in the upper range of normal generally develop hypertension over time [108] and should be encouraged to modify their diet in an effort to prevent such progression. Adherence to the recommended dietary pattern can be expected to lower systolic blood pressure by approximately 11 and 6 mm Hg, and diastolic pressure by approximately 6 and 3 mm Hg, in hypertensives and normotensives, respectively [109].

Diet and lifestyle in the prevention of obesity, insulin resistance, and diabetes mellitus

Obesity, insulin resistance, and diabetes mellitus are important contributors to the population burden of both systolic and diastolic HF, through independent and interdependent effects [110–112]. Lifestyle interventions to prevent HF, therefore, require attention to these conditions. Effective strategies for interrupting the causal links among these states of heightened risk offer the promise of preventing HF of both varieties. However, few topics in medicine generate so much heat and so little light as the optimal means to achieve sustainable weight loss or dependable weight control.

Central adiposity is strongly associated with insulin resistance, impaired glucose tolerance, and diabetes [5] and is likely a causal antecedent, although the definitive establishment of cause and effect in pathogenesis is consistently
elusive. Weight control is, therefore, appropriately viewed as the central challenge in diabetes prevention [113,114] and is an important goal in efforts to prevent HF) [110,115].

On the basis of advances in research that clarifies the relationship between weight and health outcomes, the National Institutes of Health has adopted a body mass index (BMI; weight in kilograms divided by height in meters squared) of 25 as the threshold for defining overweight [116]. A BMI greater than 30 but less than 35 is defined as stage I obesity; a BMI greater than 35 but less than 40 is defined as stage II obesity; and a BMI greater than or equal to 40 is defined as stage III obesity. The risks of complications from excess adiposity, including HF, may generally be considered low, moderate, and high as BMI rises through overweight to stage III obesity, but the actual risk in an individual will vary with other exposures, genetic susceptibility, and comorbidity [117]. Central obesity, indicated by a waist circumference greater than 102 cm in men and greater than 88 cm in women, is linked to the insulin resistance syndrome and other metabolic complications [118]. Generally, there is a strong association between central obesity and cardiovascular disease risk not seen with peripheral obesity. One mediating mechanism of cardiovascular risk in central obesity appears to be an association with high sympathetic tone [119–121]; this, in turn, may be related to the density of adrenergic receptors in centrally distributed and visceral adipose tissue. Although associated with metabolic complications of obesity, central fat tissue tends to be more readily lost than peripheral fat, in part because adrenergic receptors facilitate fat oxidation during catabolism. Thus, the prevention and reversal of central adiposity are both important and plausible goals in efforts to prevent HF.

The imbalance between energy consumption and expenditure that leads to excess weight gain can be mediated by either and is generally mediated by both, in the United States; relative inactivity and abundantly available calories contribute. Energy expenditure is composed of resting (or basal) metabolic rate (RMR), the thermic effect of food, and physical activity (Table 1). RMR accounts for approximately 70% of total energy expenditure, thermogenesis accounts for approximately 15%, and physical activity for approximately 15%, on average. The contribution of physical activity to energy expenditure is variable. A rough estimate of calories needed to maintain weight at an average level of activity is derived by multiplying the ideal weight of a woman (in pounds) by 12 to 14 and that of a man by 14 to 16.

A majority of Americans are sedentary [122] as a result of reduced work-related activity and limited leisure-time activity [123]. A reduction in exercise-related energy expenditure contributes to energy imbalance and weight gain. The attribution of weight gain to physical inactivity is compounded by the associations between activity and dietary patterns; data from the “Behavioral Risk Factor Surveillance System” [65] indicate that relative inactivity correlates with a high dietary fat intake, for example. Although there is
The energy expenditure of some representative physical activities

<table>
<thead>
<tr>
<th>Activity</th>
<th>METs(^a) (multiples of RMR)</th>
<th>kcal/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Resting (sitting or lying down)</td>
<td>1.0</td>
<td>1.2–1.7</td>
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<tr>
<td>Sweeping</td>
<td>1.5</td>
<td>1.8–2.6</td>
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<tr>
<td>Driving (car)</td>
<td>2.0</td>
<td>2.4–3.4</td>
</tr>
<tr>
<td>Walking slowly (2 mph)</td>
<td>2.0–3.5</td>
<td>2.8–4</td>
</tr>
<tr>
<td>Bicycling slowly (6 mph)</td>
<td>2.0–3.5</td>
<td>2.8–4</td>
</tr>
<tr>
<td>Horseback riding (walk)</td>
<td>2.5</td>
<td>3–4.2</td>
</tr>
<tr>
<td>Volleyball</td>
<td>3.0</td>
<td>3.5</td>
</tr>
<tr>
<td>Mopping</td>
<td>3.5</td>
<td>4.2–6.0</td>
</tr>
<tr>
<td>Golf(^b)</td>
<td>4.0–5.0</td>
<td>4.2–5.8</td>
</tr>
<tr>
<td>Swimming slowly</td>
<td>4.0–5.0</td>
<td>4.2–5.8</td>
</tr>
<tr>
<td>Walking moderately fast (3 mph)</td>
<td>4.0–5.0</td>
<td>4.2–5.8</td>
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<tr>
<td>Baseball</td>
<td>4.5</td>
<td>5.4–7.6</td>
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<tr>
<td>Bicycling moderately fast (12 mph)</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
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<tr>
<td>Dancing</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
</tr>
<tr>
<td>Skiing</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
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<tr>
<td>Skating</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
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<tr>
<td>Walking fast (4.5 mph)</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
</tr>
<tr>
<td>Swimming moderately fast</td>
<td>4.5–9.0</td>
<td>6–8.3</td>
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<tr>
<td>Tennis (singles)</td>
<td>6.0</td>
<td>7.7</td>
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<tr>
<td>Chopping wood</td>
<td>6.5</td>
<td>7.8–11</td>
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<tr>
<td>Shoveling</td>
<td>7.0</td>
<td>8.4–12</td>
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<tr>
<td>Digging</td>
<td>7.5</td>
<td>9–12.8</td>
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<tr>
<td>Cross country skiing</td>
<td>7.5–12</td>
<td>8.5–12.5</td>
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<tr>
<td>Jogging</td>
<td>7.5–12</td>
<td>8.5–12.5</td>
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<tr>
<td>Football</td>
<td>9.0</td>
<td>9.1</td>
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<tr>
<td>Basketball</td>
<td>9.0</td>
<td>9.8</td>
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<tr>
<td>Running</td>
<td>15</td>
<td>12.7–16.7</td>
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<tr>
<td>Running (4 min/mile pace)</td>
<td>30</td>
<td>36–51</td>
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<tr>
<td>Swimming (crawl) fast</td>
<td>30</td>
<td>36–51</td>
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</table>


All values are estimates, based on a prototypical 70 kg male; energy expenditure is generally lower in women and higher in larger individuals. MET and kcal values derived from different sources may not correspond exactly.

\(^a\) A MET is the rate of energy expenditure at rest, attributable to the resting (or basal) metabolic rate (RMR). Although resting energy expenditure varies with body size and habitus, a MET is generally accepted to equal approximately 3.5 mL/kg/min of oxygen consumption. The energy expenditure at one MET generally varies over the range of 1.2 to 1.7 kcal/min. The intensity of exercise can be measured relative to the RMR in METs.
kcal; at a constant level of dietary intake, such a deficit could be achieved by 45 minutes of jogging or 75 minutes of brisk walking per day.

A strong national emphasis on the health benefits of physical activity, as evinced by the Surgeon General’s Report [124], in 1996, has produced some clear benefits, such as increased availability of work-site exercise facilities. Overall, however, little progress has been made toward the “Healthy People 2010” objectives in this category [125]. Although the value of physical activity, per se, in promoting weight loss is uncertain, lifetime physical activity apparently mitigates age-related weight gain and is clearly associated with important health benefits [126]. The contribution of physical activity to weight maintenance may vary among individuals on the basis of genetic factors that are as yet poorly understood [127]. There is increasing and encouraging evidence that lifestyle activity, as opposed to structured aerobic exercise, may be helpful in both achieving and maintaining weight loss [128]. Such unobtrusive physical activity may be more readily accepted by exercise-averse patients.

Physical activity is among the best predictors of long-term weight maintenance [129–132]. It has been estimated that the expenditure of approximately 12 kcal/kg of body weight/d in physical activity is the minimum protective against increasing body fat over time [132].

Because approximately 70% of calories are spent on basal metabolism, even vigorous physical activity may be insufficient to control weight when caloric intake substantially exceeds the needs of resting metabolism. Although the number of calories required to maintain weight varies substantially among individuals, the degree of caloric restriction, relative to habitual intake, required to produce weight loss is more predictable. Each pound of body fat represents a repository of approximately 4000 kcal (454 g × 9 kcal/g). To lose a pound of fat requires that energy expenditure be increased by 4000 kcal or that intake be restricted by a comparable amount. To reduce caloric intake by 4000 kcal over 1 week requires a daily restriction of between 500 and 600 kcal. In a 2000-kcal diet, this restriction represents a 25% reduction in total caloric intake. Therefore, whatever the baseline caloric intake required to maintain weight, a reduction of 500 to 600 kcal/d will generally result initially in approximately 1 pound of weight loss per week. As basal metabolism declines, further reductions may be required to sustain the weight loss.

Because dietary fat is more energy dense and less satiating than either protein or carbohydrate [133], weight loss recommendations have long emphasized dietary fat restriction. Most epidemiologic evidence links dietary fat to body fat [134], and high intake of certain dietary fats appears to be linked with insulin resistance, independently of obesity [135]. Recently, the view that dietary fat restriction is warranted for weight control has been subject to both scholarly debate [136–144] and the counter-culture dissension of popular diet proponents. The presently popular weight loss diets, foremost among them the “Atkins diet,” tend to emphasize carbohydrate rather than fat restriction and to emphasize the glycemic index of foods. Recent
short-term trials demonstrating weight loss with such diets [145,146] have heightened interest even among clinicians.

Disease prevention, however, serves as a useful “reality check” in confronting the plethora of opinion regarding diet and weight control. The first consideration when recommending any dietary pattern is overall health; weight control should be pursued within the context of a health-promoting lifestyle. The basic, if not all, parameters of a health-promoting diet are well established, as discussed earlier, and consensus is considerable among experts. Such a dietary pattern, which is rich in grains, vegetables, and fruit, moderate and balanced, in combination with regular physical activity, is associated with sustainable weight loss [147]. The same pattern is associated with the prevention of diabetes [148] and, once it is established, improvement in the metabolic control of diabetes [149]. Trial and observational data and the dictates of good clinical judgment thus converge to support the same basic dietary pattern (Table 2) for the control of weight, the mitigation of cardiac risk, and the potential prevention of HF through the interruption of several causal pathways. The relative ineffectiveness of the glycemic index as a stand-alone basis for dietary counseling is widely acknowledged (Table 3).

Physical activity in the prevention of heart failure

Regular physical activity is associated with the prevention and control of virtually every known modifiable risk factor for HF. An inverse association is well established between physical activity and atherosclerotic coronary artery disease [150], and a dose response is probable over a wide range of activity levels [151,152]. Consistent physical exertion at moderate levels is integral to the prevention and control of diabetes and insulin resistance [148] and obesity [147], as noted earlier. An inverse association between activity levels and blood pressure is also well established [153].

Thus, a role for exercise is clear in the primary prevention of HF; it effectively impedes the development of HF along most of the pertinent causal pathways. Equally clear is the role of exercise in the mitigation of cardiac risk factors and even cardiac disease, once manifest. The benefits of physical activity in coronary disease are denoted by the prevailing use of cardiac rehabilitation programs after myocardial infarction. Evidence is consistently accruing to support a role for physical activity in the management of HF and the mitigation of its progression, as well [154,155]. This is an area of active research; therefore, new insights over the short term, and the rapid evolution of pertinent clinical guidelines, are probable [156].

Other lifestyle considerations in the prevention of heart failure

A substantial and briskly growing literature attests to the importance of psychological stress as a cardiac risk factor [157,158]. Although a direct causal link between mental stress and HF is not established, stress is likely
Table 2
Recommended dietary composition for health promotion and disease prevention

<table>
<thead>
<tr>
<th>Nutrient class/nutrient</th>
<th>Recommended intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbohydrate, predominately complex fiber, both soluble and insoluble</td>
<td>Approximately 55%–60% of total calories At least 25 g/day, with additional potential benefit from up to 50 g/day</td>
</tr>
<tr>
<td>Protein, predominantly plant-based sources</td>
<td>Up to 20% of total calories</td>
</tr>
<tr>
<td>Total fat</td>
<td></td>
</tr>
<tr>
<td>Types of fat</td>
<td>Not more than 30%, and preferably 20%–25% of total calories</td>
</tr>
<tr>
<td>Monounsaturated fat</td>
<td>10%–15% of total calories</td>
</tr>
<tr>
<td>Polyunsaturated fat</td>
<td>10% of total calories</td>
</tr>
<tr>
<td>Omega-3 and omega-6 fat</td>
<td>1:1 to 1:4 ratio</td>
</tr>
<tr>
<td>Saturated fat and trans fat (partially hydrogenated fat)</td>
<td>Ideally, less than 5% of total calories</td>
</tr>
<tr>
<td>Sugar</td>
<td>Less than 10% of total calories</td>
</tr>
<tr>
<td>Sodium</td>
<td>Up to 2400 mg/day</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Less than 300 mg/day</td>
</tr>
<tr>
<td>Water</td>
<td>Approximately 8 glasses (64 oz)/day; thirst is generally a reliable guide</td>
</tr>
<tr>
<td>Alcohol, moderate intake if desired</td>
<td>Up to one drink/day for women; up to two drinks/day for men</td>
</tr>
<tr>
<td>Calorie level</td>
<td>Adequate to achieve and maintain a healthy weight; this generally requires attention to portion control</td>
</tr>
<tr>
<td>Physical activity and exercise</td>
<td>Daily moderate activity for at least 30 minutes; strength training twice weekly</td>
</tr>
</tbody>
</table>

This dietary pattern is based on the confluence of data from intervention trials, observational studies, and trans-cultural epidemiologic comparisons. Further guidance is derived from the anthropology literature reporting on the “native” nutritional habitat of Homo sapiens. Also considered are the recommendations of such groups as the American Diabetes Association, the American Dietetic Association, the American Heart Association, and the Institute of Medicine. Adapted from Katz DL, Gonzalez MH. The way to eat. Naperville, (IL): Sourcebooks, Inc., in cooperation with The American Dietetic Association; 2002. p. 213.

a frequent contributor along the various causal pathways that are established. Stress control is, therefore, a potentially important component of comprehensive lifestyle interventions for the prevention of HF; a detailed discussion of pertinent methods is beyond the scope of this paper.

Along with overall dietary and activity patterns, some nutrients and botanicals may prove to be of value in the secondary prevention of HF. Arginine and taurine may have antihypertensive properties, but evidence to date is limited [159]. Arginine is a precursor in the synthesis of nitric oxide, an endothelium-derived vasodilator; a link between blood pressure and endothelial function is clear, although the direction of causality is not [160,161]. Evidence is as yet insufficient to justify amino-acid supplementation in efforts to regulate blood pressure or to prevent HF.

Garlic stimulates nitric oxide synthase [162], providing a mechanism by which it might lower blood pressure, and meta-analysis supports a modest
antihypertensive effect of garlic [163]. Coenzyme Q₁₀ (ubiquinone) has been associated with improvement in left ventricular function, quality of life, and functional status in some studies of HF [164,165]. There is evidence of reduced complications after myocardial infarction [166], improved hemodynamics after bypass grafting [167], and improved functional status and symptom relief in patients with angina [168]. Coenzyme Q₁₀ also has been shown to have antihypertensive effects [169–171].

### Table 3

<table>
<thead>
<tr>
<th>Food group</th>
<th>Food</th>
<th>Glycemic index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breads</td>
<td>White bread*</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>Whole wheat bread</td>
<td>99</td>
</tr>
<tr>
<td></td>
<td>Pumpernickel</td>
<td>78</td>
</tr>
<tr>
<td>Cereal products</td>
<td>White rice</td>
<td>83</td>
</tr>
<tr>
<td></td>
<td>Spaghetti</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td>Barley</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>Bulgur wheat</td>
<td>65</td>
</tr>
<tr>
<td></td>
<td>Cornflakes</td>
<td>119</td>
</tr>
<tr>
<td></td>
<td>Shredded wheat</td>
<td>97</td>
</tr>
<tr>
<td></td>
<td>Oatmeal</td>
<td>85</td>
</tr>
<tr>
<td>Fruit</td>
<td>Bananas</td>
<td>79</td>
</tr>
<tr>
<td></td>
<td>Apples</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>Oranges</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td>Grapes</td>
<td>62</td>
</tr>
<tr>
<td></td>
<td>Cherries</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>Raisins</td>
<td>93</td>
</tr>
<tr>
<td>Vegetables</td>
<td>Boiled potato</td>
<td>81</td>
</tr>
<tr>
<td></td>
<td>Baked potato</td>
<td>135</td>
</tr>
<tr>
<td></td>
<td>Corn</td>
<td>87</td>
</tr>
<tr>
<td></td>
<td>Peas</td>
<td>74</td>
</tr>
<tr>
<td></td>
<td>Carrots</td>
<td>133</td>
</tr>
<tr>
<td></td>
<td>Yams</td>
<td>74</td>
</tr>
<tr>
<td></td>
<td>Parsnips</td>
<td>141</td>
</tr>
<tr>
<td>Legumes</td>
<td>Lima beans</td>
<td>115</td>
</tr>
<tr>
<td></td>
<td>Barked beans</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>Chick peas</td>
<td>49</td>
</tr>
<tr>
<td></td>
<td>Red lentils</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>Peanuts</td>
<td>19</td>
</tr>
<tr>
<td>Dairy products</td>
<td>Milk</td>
<td>49</td>
</tr>
<tr>
<td></td>
<td>Yogurt</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>Ice cream</td>
<td>52</td>
</tr>
<tr>
<td>Sugar</td>
<td>Sucrose</td>
<td>86</td>
</tr>
</tbody>
</table>

The glycemic index (GI) of some foods is shown. Limitations to the use of the GI as a guide to good eating are suggested in particular by the boldface entries. Pure sugar and ice cream have a lower GI than white bread, whereas carrots have a higher glycemic index.


* Reference standard.
trials of coenzyme Q₁₀ undertaken to establish a definitive role in HF management or prevention are thus far lacking, but the weight of evidence suggests such benefits [172]. More widespread use of coenzyme Q₁₀ in cardiology and primary care practice may warrant serious consideration. The usual doses in trials range from 100 to 300 mg/d, taken orally twice per day. Such doses appear safe, with virtually no reports of significant toxicity.

Finally, as noted earlier, a role is clear for fish oil in the prevention of cardiac dysrhythmias in high-risk patients [173]. Regular ingestion of fish is associated with reduced risk of heart disease [71]. There is no clear case for fish oil supplementation in efforts to prevent HF, per se, although habitual intake of 0.5 g–1 g/d could be recommended on other grounds and might confer incidental cardiac benefit [70].

Summary

The factors that contribute most to both systolic and diastolic HF, ischemic heart disease, hypertension, obesity, diabetes, and nephropathy, are inextricably connected. Diabetes often follows the insulin resistance syndrome, in which obesity and hypertension are combined with dyslipidemia; obesity is likely causal. Diabetes and hypertension are common causes of nephropathy, which in turn is a common precursor to HF. Insulin resistance, obesity, dyslipidemia, diabetes, and hypertension are risk factors for atherosclerotic coronary disease and, therefore, left ventricular ischemia; each is a risk factor for diastolic dysfunction, as well.

Fortunately, there is a similar convergence among the salutary lifestyle factors that are likely to lend protection against heart disease [11,174]. Regular physical activity is associated with weight control, blood pressure reduction, improved insulin disposal, amelioration of serum lipid levels, and reduced risk of atherosclerosis. A diet low in saturated and trans fat, relatively higher in polyunsaturated and monounsaturated fats, abundant in fruits, vegetables, and whole grains, and consequently insoluble and especially soluble (viscous) fiber, and relatively restricted in simple sugar and refined starch is similarly associated with an array of benefits, prominently among them, the prevention of coronary events [175]. Such a diet, inclusive of nonfat dairy products, has been shown in the DASH trials to offer clear benefit in the control of blood pressure. With only slight variation on the theme of this healthful dietary pattern, and in combination with regular physical activity, a compelling role has been established by in the “Diabetes Prevention Program” [148] for lifestyle in the prevention of diabetes. Late-stage prevention trials, such as the “Lyon Diet Heart Study” [52], “Lifestyle Heart Trial” [176], and the “GISSI Prevenzione Trial” [177] suggest that the putative benefits of this lifestyle pattern in the prevention of HF continue to accrue, even if prevention measures are initiated late.

Although there are various targeted strategies for preventing HF, including pharmacotherapies (eg, angiotensin-converting enzyme inhibitors,
β-blockers, and aldosterone inhibitors) and possibly nutriceuticals (eg, fish oil, and possibly coenzyme Q10) [178,179], far greater potential public health benefit derives from interventions of more generalized relevance. The causative factors that underlie most cases of both systolic and diastolic HF are themselves substantially preventable through early lifestyle interventions [180]. Primary care practitioners should be diligent in efforts to detect and modify HF risk factors and cognizant of the prevalence of asymptomatic HF. The prevention of HF, however, could be advanced more by universal dedication among clinicians to the promotion of healthful dietary practices and regular physical activity in all of their patients and the population at large.

Although doubtless that good will comes from research directed at the further elucidation of the optimal dietary and activity patterns for heart disease prevention, we are well served by acknowledging that the essentials are already known. Primary care physicians should strive to become knowledgeable in these areas, using resources dedicated to the purpose. Providers, patients, and policy makers need to accept that the particular challenge of applying lifestyle practices to the prevention of HF is not so much the identification of “what” but the determination of “how,” in an environment that is increasingly conducive to physical inactivity and dietary excess [181]. A strategic blend of environmental reform and imparting key skills and strategies to individuals [182] offers the promise of population-wide improvements in lifestyle and health. There are populations in which HF simply does not occur, and that serves to establish the ultimate goal toward which our collective efforts should be directed.

With worsening epidemic levels of obesity and diabetes and the implicit threat of attendant changes in the epidemiology of heart disease, we are by no means out of the woods when it comes to realizing the promise of preventing HF. For that promise to be kept, clinicians, researchers, and policy makers must all accept that, although the destination may be visible, there are miles to go over difficult terrain. That this threat to the population’s health may one day be put to sleep should serve to motivate our perseverance.

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