the increasing awareness of the cost of anesthesia-assisted sedation. Although there is a general perception that reimbursement for health care services in the United States will imminently decline, it is difficult to predict the exact impact of these ever-changing reimbursement policies on endoscopy and the provision of anesthesia. The only certainty is that the utilization and reimbursement of anesthesia in endoscopy is heavily influenced by economics and regulatory issues.[2]

Several studies have reported the safety of propofol sedation as administered by endoscopists.[3-5] In a review of 569,220 cases of propofol sedation administered by endoscopists, only 0.09% (489 cases) required mask ventilation as a result of oversedation.[6] In the analysis by Rex et al,[6] substituting anesthesia professional services with endoscopist-administered propofol saved $5.3 million per patient-year saved. Despite the published evidence, the Centers for Medicare & Medicaid Services still considers the use of propofol for colonoscopy to be equivalent to deep sedation. This reaffirms propofol's black box warning to limit its administration to anesthesia professionals who are qualified to provide general anesthesia. As such, the price of anesthesia-assisted sedation is strongly tied to the cost of having an anesthesia professional provide sedation and not on the actual advantages of propofol over opiates and benzodiazepines.[2]

The objective of our article was to provide evidence-based and practical recommendations for requesting anesthesia-assisted sedation in endoscopy. As gastroenterologists, we share Dr Hirsch's sentiments about making sure that patients undergoing routine endoscopy will continue to have comfortable, safe, and effective endoscopic procedures regardless of the current pressures from cost containment and endless politics of determining reimbursements for health care services.

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http://dx.doi.org/10.1016/j.mayocp.2013.12.011

Population-wide Sodium Reduction: Reasons to Resist

To the Editor: In their review article in the September 2013 issue of *Mayo Clinic Proceedings*, Aaron and Sanders[1] stated that "high salt intake not only increases blood pressure but also plays a role in endothelial dysfunction, cardiovascular structure and function, albuminuria and kidney disease progression, and cardiovascular morbidity and mortality in the general population" and that "the body of evidence supports population-wide sodium intake reduction." What the authors neglect to mention is that reducing sodium intake has been associated with increases in renin, aldosterone, adrenaline, noradrenaline, cholesterol, and triglyceride levels.[2] It is uncertain that the net result of sodium restriction would be positive for health given these potential adverse effects. Moreover, reducing sodium intake may worsen insulin resistance,[3] increasing the prevalence of diabetes and prediabetes (already affecting approximately one-third of the entire US population[4]). Low-sodium diets could also exacerbate thyroid disease by reducing individuals' intake of iodine (obtained in most cases through ingestion of iodized salt).[5] Additionally, sodium avoidance could lead to hyponatremia, for which much of the population may be at risk due to (1) life behaviors (eg, manual labor/strenuous exercise, when replacing sweat with free water), (2) commonly used medications (eg, selective serotonin reuptake inhibitors, tricyclic antidepressants, nonsteroidal anti-inflammatory agents, antipsychotics, and thiazide diuretics), or (3) prevalent disease states (eg, liver disease, cancer, and congestive heart failure).[6] Regarding congestive heart failure in particular, what is even more worrisome is that randomized clinical trials have repeatedly suggested that restricting sodium intake to 1800 mg daily, compared with 2800 mg daily, is associated with increased rates of hospitalizations and mortality.[7-10]

Even if it were physiologically possible for individuals to sustainably reduce sodium intake—a proposition that science calls into question[11]—it is entirely possible that attempting to reduce population sodium consumption would do more harm than good. There are certainly many compelling reasons to resist the idea of population-wide sodium reduction. Moreover, recent literature suggests that markedly cutting back on sodium consumption may be much less important than increasing one's dietary potassium intake.[12]

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http://dx.doi.org/10.1016/j.mayocp.2014.01.007

In Reply—Population-wide Sodium Reduction: Reasons to Resist

A discussion of salt intake always generates a vigorous debate. In some part, controversy occurs because of the years of study generally required to determine the effect of dietary modifications on morbidity and mortality. Perhaps as a result, association studies have proliferated in this area to replace randomized controlled trials. However, the field of nephrology in particular has learned a series of "tough lessons" as a result of relying on observational data and is now emphasizing the need for randomized controlled studies to dictate clinical practice. With this background, DiNicolantonio et al bring forward several interesting observations that merit additional comment.

DiNicolantonio et al claim that reducing salt intake may increase renin, aldosterone, adrenaline, noradrenaline, cholesterol, and triglyceride levels. Although the examination of these associations of dietary sodium intake with the renin-angiotensin-aldosterone system is of interest, the primary consideration should be hard end points such as mortality and not just an association with renin levels. The potential issue of the effect of a low-salt diet on insulin levels is also mentioned, but perhaps caloric intake in our modern society should be considered the primary cause of hyperinsulinemia and not a low-salt diet. In addition, clinical outcomes—not necessarily associations with insulin levels—should be the preferred end point when determining the optimal mode of sodium management in patients who have (or are at risk for) diabetes mellitus.

DiNicolantonio et al quote a meta-analysis by Graudal et al[1] that derived data from sudden and large reductions in salt intake and ignored contrary evidence.[2] The underlying rationale for the inconsistent findings in the report by Graudal et al is that a drastic reduction in dietary sodium intake can lead to unfavorable metabolic and neurohormonal alterations, which could promote insulin resistance, lipid abnormalities, and increased cardiovascular risk through a compensatory activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system (activation that is proportional to the degree of sodium reduction).[1,3] The theory seems to be valid only for an extremely low sodium intake[4] and not for the 2300 mg/d recommended for the general population.[5]

Moderate salt reduction in the United States is unlikely to have a major impact on iodine status or exercise-associated hyponatremia. Most salt in the diet is derived from processed foods, and the salt used in food processing in the United States is typically not iodized.[6] Encouraging the food industry to use iodized salt during processing or alternative fortification strategies is more likely to yield optimal iodine status in Americans.[7,8] Although careful population monitoring for iodine status is required, countries that mandate iodization of salt used in processing and for personal consumption have found that lower salt intakes do not compromise iodine status.[9] In contrast to the statement by DiNicolantonio et al, most cases of exercise-associated hyponatremia are due to excessive water consumption rather than a lack of salt in the diet.[10-12]

Finally, in regard to the statement on congestive heart failure (CHF), our review excluded studies with CHF as the main outcome. However, a caveat in the discussion section of our article[13] stated that clinicians should be aware that patients with severe CHF take multiple medications that inhibit or block the renin-angiotensin-aldosterone system and thus may not benefit from salt restriction.[14] Less rigorous targets for salt reduction may be appropriate for certain groups of patients with CHF or multiple comorbid conditions.

It is currently impossible for clinicians to provide strictly individualized dietary recommendations to the general population because the approach would require them to have already outlined nutrigenomic interactions and underlying genetic susceptibility