The heart’s primary function is to pump blood to all parts of the body, bringing nutrients and oxygen to the tissues and removing waste products. Even when the body is at rest, it needs a certain amount of blood to achieve this function. During exercise or times when greater demands are placed on the body, more blood is required. To meet these demands, the heartbeat increases or decreases, and blood vessels dilate to deliver more blood or constrict during times when less blood is required.

When a person is diagnosed with heart failure, it does not mean the heart has stopped working, but rather that it is not working as efficiently as it should. In other words, the term “failure” really indicates that the heart is not pumping effectively enough to meet the body’s need for oxygen-rich blood, either during exercise or at rest. The term congestive heart failure (CHF) is often synonymous with heart failure but it also refers to the state in which decreased heart function is accompanied by a congestive buildup of body fluid in the lungs and elsewhere.

Since the 1950s, the gold standard of dietary intervention for people with congestive heart failure has been a low salt diet. Were anyone to do a search on Google for diet + “congestive heart failure”, they would find more than ½ a million citations, virtually all of them recommending a low salt diet. This is fully in line with the near unanimous “understanding” that low salt diets are the undisputed cure for blood pressure.

But consensus on an approach to treatment does not necessarily mean that the treatment is correct. The 1970s witnessed a similar consensus over need to reduce saturated fats in the diet, however, more comprehensive research has since shown this notion to be incorrect. Only consistent, high quality research can verify whether or not a particular therapy works. Might this be the case with congestive heart failure?

Congestive heart failure (CHF) can result from a number of causes:

- narrowed arteries that supply blood to the heart muscle, or coronary artery disease
- a past heart attack, or myocardial infarction, with scar tissue that interferes with the heart muscle’s normal work
- high blood pressure
- heart valve disease, due to past rheumatic fever or other causes
- disease of the heart muscle itself, called cardiomyopathy
- congenital heart defects
- infection of the heart valves and/or heart muscle itself (endocarditis and/or myocarditis)

Classifications of Heart Failure

The New York Heart Association developed a system that has been used for many years to provide a standardized set of criteria for the classification of heart failure based on the severity of the condition. This is evaluated by symptoms and ability to function.

Class I: no undue symptoms associated with ordinary activity and no limitation of physical activity

Class II: slight limitation of physical activity; patient comfortable at rest

Class III: marked limitation of physical activity; patient comfortable at rest

Class IV: inability to carry on any physical activity without discomfort; symptoms of cardiac insufficiency or chest pain possible even at rest.
People with congestive heart failure can’t exert themselves because they become short of breath and tired. As blood flow out of the heart slows down, blood returning to the heart through the veins starts to back up, causing congestion in the tissues. Often, swelling or edema results. Sometimes fluid collects in the lungs and interferes with breathing, causing shortness of breath, especially when a person is lying down. Most often, there’s swelling in the legs and ankles, but it can happen in other parts of the body, too. Congestive heart failure also affects the kidneys’ ability to get rid of sodium and water. This retained water increases the body’s edema.

When one system of the body is not functioning optimally, other systems may attempt to take over to make up for the problem. In the case of heart failure, several types of compensation are possible. First, the heart chambers may enlarge, and the heart may beat more forcefully to pump out more blood for the body’s needs. In time, the overworked heart muscle enlarges (much as skeletal muscles grow larger during weight training), creating increased muscle fibers with which the heart can pump more forcefully. Second, the heart may be stimulated to pump more often, thereby increasing its output. Third, a compensation mechanism called the renin-angiotensin-aldosterone system (RAAS) may be initiated. When the lack of blood volume coming from the heart (cardiac output) results in a decrease in the amount passing through the kidneys, the kidneys respond by stimulating the system to secrete hormones that prompt the kidneys to retain salt and water, and thereby increase blood volume. This is an attempt to compensate for the decrease in output of the heart. This can lead to a rise in blood pressure as the body attempts to circulate the extra fluid volume and also ensures that adequate oxygen reaches the brain, kidneys, and other vital organs.

These compensation mechanisms keep the failing heart functioning almost normally in the early stages of heart failure. If the disease progresses, however, compensation mechanisms cannot maintain proper circulation. It may take years for a heart to go through the stages of enlarging, working harder and harder and finally breaking down. In many cases, as when a person has hypertension, heart failure is preventable if the symptoms are treated adequately.

Following a long line of investigations conducted at the Albert Einstein School of Medicine in New York City, two new studies have just been published that have supported the observation that when people, who have had previous congestive heart failure problems, are placed on a low salt diet, they fare far worse than those who were kept on a regular salt diet.

The first paper, authored by Dr. Salvatore Paterna et al., published late in 2008 in the journal, Clinical Science, had the controversial title,
“Normal sodium diet compared with low sodium diet in compensated congestive heart failure: is sodium an old enemy or a new friend?”

The goal of this study was to evaluate the difference between a normal sodium (2,760 mg sodium per day) and a low sodium diet (1,840 mg sodium per day) on hospital readmissions after patients who experienced congestive heart failure were released. The duration of this study was 180 days.

When the study was concluded, it was found that the group of patients receiving the normal sodium diet had significantly fewer readmissions compared to the group that was placed on the low sodium diet. People receiving the low sodium diet were also found to have much higher levels of aldosterone and plasma renin activity, both known to negatively affect the circulatory system.

The conclusion of the study was that a normal level of sodium in the diet improved health outcomes and that low sodium intakes had detrimental renal and neurohormonal effects -- in summary, far worse clinical outcomes.

This study was quickly followed up by another from the same group, published in January 2009, in the American Journal of Cardiology. This second, much larger study, was designed to evaluate the effects of different levels of sodium in the diet together with different diuretic doses and different levels of fluid intake. Again, hospital readmissions and clinical outcomes were carefully measured in the six-month follow-up.

The researchers went out of their way to ensure that the people observed shared relevant characteristics such as age, weight, blood pressure, etc. Physicians doing the measurements were blinded as to which group they were analyzing to ensure that all the measurements were objectively made.

Measurements of heart failure, body weight, blood pressure, heart rate, standard laboratory parameters, electrocardiograms, echocardiograms, aldosterone, renin and brain natriuretic peptide were all examined at the start of the study and 180 days later. (Brain natriuretic peptide is a polypeptide hormone secreted by the ventricles of the heart, whenever it is under stress and being overworked.)

The results were not those that were expected. As highlighted above, for decades now, the gold standard of care for people who have experienced congestive heart failure has been a reduction of salt in the diet. Despite the many publications coming out of the Albert Einstein School of Medicine, patients are still placed on a low salt diet to recover from heart failure.

In fact, the Paterna results showed that the group placed on a regular salt diet proved to have far better health outcomes than the group that was placed on a low salt diet. The low salt diet caused a general worsening in renal function and lead to greatly increased rates of readmissions and mortality.

For the group placed on the regular salt diet and given one liter per day fluid, there was a 7.7% rate of readmissions and a 2% mortality rate. In the group placed on a low salt diet and given one liter per day fluid, there was a 49% rate of readmissions and a 10% mortality rate.

In the group placed on the regular salt diet, blood pressures and heart rates remained the same or decreased slightly throughout the course of the trial, while they increased in the low salt group. In the regular salt group, body weight remained the same or decreased slightly, while in the low salt group, body weight increased significantly, most likely due to fluid buildup because there was also a significant decrease in urinary output in the low salt group.

Importantly, the group on the low salt diet showed significant increases in plasma aldosterone, plasma renin and brain natriuretic peptide – all signs of a deterioration in the patients condition. No such changes were observed in the group on the regular salt diet.

The Kaplan Meier curves shown below represents the fraction of patients that have survived and were not readmitted to the hospital.
over the course of the study. Even at 30 days, the group on the regular salt diet (blue line) did considerably better than those on the low salt diet (green line). This effect was even more pronounced at 125 days.

When this data is combined with the results from the three NHANES studies carried out at the Albert Einstein School of Medicine1-4, it becomes clear that serious consideration has to be given to changing the low sodium dietary intervention gold standard for patients with congestive heart failure. Research, carefully and objectively carried out, shows that such patients will fare much better when they are not placed on a low salt diet.

We are likely to find much the same result in health outcomes with the general population. While there is no doubt that a certain portion of the population are salt sensitive and will benefit from a reduction in salt intake, other in the population will be affected negatively6-7-8. The ethical dilemma of placing certain members of the population at a greater risk in order to reduce the risk of another portion of the population is discriminatory and can be entirely avoided. The whole population will benefit far more if their diet is improved

the consumption of more fruits, vegetables and dairy products – even the salt sensitive population will lose much of their sensitivity on such a diet. What’s more, the overall burden of all diseases including cancer and diabetes in the general population will be reduced9.

It’s time we start to heed the scientific evidence rather than be driven by a compulsion to regulate a nutrient for which we already have multiple biological systems for natural regulation. Dr. J. Quick, Director of essential drugs and medicines policy for the World Health Organization (WHO), wrote in a recent WHO Bulletin: “if clinical trials become a venture in which self-interest overrules public interest and desire overrules science, then the social contract which allows research on human subjects in return for medical advances is broken.”

References

1. Paterna S ; Gaspare P ; Fasullo S ; Sarullo FM ; Di Pasquale P, "Normal-sodium diet compared with low-sodium diet in compensated congestive heart failure: is sodium an old enemy or a new friend?" Clin Sci (Lond), 2008; 114(3):221-30 (ISSN: 1470-8736).

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Further salt and health information is included on the same page beneath the references-on-salt-use/SI-references-on-salt-in-food.

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Salt Institute
700 North Fairfax Street
Fairfax Plaza, Suite 600
Alexandria, VA 22314-2040
Voice: 703 / 549-4648
Fax: 703 / 548-2194 Fax
Web Site: http://www.saltinstitute.org
e-mail: info@saltinstitute.org

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