Softened Water Need Not Be A Danger

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Whether one should or should not soften water has been an increasingly agonizing question for almost 20 years.1,2 This concern relates to repeated observations by investigators in a number of different countries and areas that high drinking water hardness appears to affect lowered heart and circulatory diseases death rates. Although we still do not understand why such a relationship should exist, some progress has nevertheless been made in helping define the relationship further—at least, so this writer thinks. Accordingly, water can be softened, and it can be so done without harming health as long as the sodium content is not raised to unacceptable levels.

Hardness is caused predominantly by calcium and magnesium concentrations in water. Morris, Crawford, and Heady,3 English investigators, on the basis of epidemiological studies were able to rule out magnesium as contributing to a health problem—the first major contribution to the study.

Then Canadian, Dr. T. Anderson,4 observed that water hardness related most closely to ischemic heart disease, or the sudden-death syndrome, and that the relationship was more likely to be a detrimental effect from something present in soft water than a protective effect from something present in hard water. Shortly thereafter, Dr. Edward Lee Russell,5 from a variety of water quality factors chose to study sodium. His conclusion was "... that 37.9 per cent of the residents of the . . . (southern California) district are served a domestic water supply containing 110 mg/l or more of sodium, an amount that would place in jeopardy all residents who have confirmed or incipient congestive heart disease, hypertension, renal disease, or cirrhosis of the liver.''

Texas A & M researchers in Dallas with support from both the City of Dallas and the Texas Water Resources Inst.6 were able to show a rise in all categories of heart-disease deaths. This rise was coincidental with a drought period during which highly mineralized water from the Red River was imported for consumption. The high death rate persisted for a year following termination of water importation.

Although calcium, magnesium, alkalinity, and total dissolved solids increased during the importation period—all of which had been linked with a protective effect against heart-circulatory deaths—the deaths increased. Further, the increase occurred at the sodium levels suggested by Dr. Russell. Figure 1 (heretofore unpublished) shows the deaths for one of the categories of heart disease for both the cities of Dallas and Ft. Worth (the latter did not import water, uncorrected for increasing populations.)

The evidence implicated high sodium concentrations. The writer wondered if, in general, sodium did not correlate inversely with water hardness, specifically with calcium. Plots of the sodium and calcium content of public water supplies in Texas show that this relationship does, indeed, exist: that most high sodium supplies are low in calcium and most high calcium supplies are low in sodium.7 The observed negative correlations between hardness and heart-disease deaths could now be hypothesized as attributable to sodium content. If sodium is, indeed, the culprit, some differences ought to be observable between cities using high- and low-sodium drinking water. Such a test was possible right in Dallas County, and with the additional help of the state department of health, this writer and B.J. Moore demonstrated a rather

Fig. 1. Deaths Attributed to Arteriosclerotic Heart Disease Including Coronary Disease, Category 420, 7th Revision International Lists 1955.

(Continued on page 23)
massive difference, but it was confined to the older population and only at a low order of statistical significance.7

The writer has been reflecting on these studies and many others and upon their various anomalies. He offers the following hypothesis. Sodium content in drinking water in the range 100-200 mg/l impairs salt-taste acuity of individuals8 who then unwittingly use more salt in their diet. This could explain both the persistence of deaths beyond the period of water import for Dallas and the low significance, yet massive, effect observed by this writer and Moore (the major contribution of sodium being from the diet, not the drinking water). The writer does not intend to dismiss the potential role that water instability may play in drinking-water-health relationships. Nor is the writer suggesting that sodium contributes to arteriosclerosis. But regardless of the predisposing factors, the mechanism of actual death is apparently a sodium-mediated event, albeit indirectly, such as a congestive heart failure.

Based on this interpretation of the water factors, certainly lime softening would constitute a totally safe procedure. It contributes no sodium, and if well operated, will produce a pathogen-free water that is highly stable. Soda-ash processes contribute sodium, and, depending upon the amount contributed and the amount already present in the water, these processes should be reviewed on a case-by-case basis. Zeolite softening at the home level is perfectly amenable to Dr. Shaper's suggestion of allowing a drinking tap to bypass the softener.9 (For economic reasons, one might also not want to flush toilets with softened water.) The main problem in the writer's view occurs with utility use of Zeolite processes. Many utilities might be well advised to convert to lime softening, or not soften at all and let the home owner soften individually. Any decision would have to be based on the amounts of sodium present in the finished waters, and it would certainly help if the agencies and associations that have responsibilities in the matter would get their heads out of the sand and develop the sodium criteria that are so badly needed.

References

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