Commentary

The Spectrum of Illness During Heat Waves

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Severe and sustained episodes of summer heat (heat waves) have a major impact on health. In temperate regions, heat waves can be surprisingly lethal. The 1980 summer heat wave killed 122 residents of St. Louis and 157 residents of Kansas City, Missouri.[1] At the national level, an increase in daily mortality was discernible with about 5300 "excess" deaths.[2] A heat wave in 1995 also had a dramatic health impact, causing approximately 2800 "excess" deaths in the United States.[2] The City of Chicago was particularly hard hit. The deaths of at least 437 Chicago residents were attributed to the heat.[3]

The study of the full range of heat-related health effects is problematic. It is easy to identify victims of heatstroke because of their dramatic clinical presentation (altered mental status and body temperature ≥105°F [40.6°C]). However, from 12% to 100% of excess deaths occurring during heat waves are not recognized as heatstroke, heat exhaustion, or any other syndrome clearly caused by the heat wave. Rather, these additional deaths are attributed to other, more common diagnoses. It is frequently impossible to determine which cases represent deaths that were triggered by the heat and which cases would have occurred anyway. However, at least some cases must be heat-triggered because the overall mortality for certain diagnoses increases greatly in the heat.[4]

Deaths due to cardiovascular, cerebrovascular, and respiratory illness have repeatedly been observed to increase during heat waves. Historically, cardiovascular disease has accounted for from 13% to 90% of the overall heat wave mortality increase, cerebrovascular disease from 6% to 52%, and respiratory diseases from 0% to 14% of the increase.[4]

Heat waves also produce increases in nonfatal illness. In this issue of the Journal, an article by Jan Semenza and co-workers describes how they determined the diagnoses seen with increased frequency during the heat by comparing the discharge diagnoses of patients hospitalized in Chicago during the 1995 heat wave with those of patients admitted during control periods. They detected a heat-wave-associated increase in hospital admissions,[5] a finding that has been noted previously.[1] However, their detailed analysis of "excess" admissions by discharge diagnosis is a new and important contribution to the literature.

Interestingly, primary discharge diagnoses among patients hospitalized during the heat wave did not correspond well with the underlying causes of death known to increase during the heat. The authors found only a small increase in cardiovascular disease as a primary discharge diagnosis, and this did not reach statistical significance. Similarly, cerebrovascular and respiratory diseases did not increase in frequency as primary discharge diagnoses.

The picture was different, however, when both primary and secondary discharge diagnoses were considered. There was a statistically significant increase of 461 additional patients diagnosed with a cardiovascular disease during the heat wave, enough to account for 43% of the 1072-patient increase in the total number of admissions. Similarly, there were significant heat-wave-related increases in cerebrovascular and respiratory diseases. The inclusion of secondary discharge diagnoses thus unmasked increases in diagnostic categories mirroring known cause-specific increases in heat-associated mortality.

Thus, the authors needed to consider all discharge diagnoses, not just those designated as primary, in order to demonstrate the similarities of fatal and nonfatal heat-related health events. This finding probably reflects differences in procedure and intent in the coding of death certificates and hospital discharges. The objective of coding the underlying cause of death on a death certificate is to identify the diagnostic entity most responsible for initiating the sequence of events leading to death. On the other hand, a discharge diagnosis may be chosen to reflect the major clinical entity diagnosed or treated in the hospital. In addition, where multiple diagnoses exist, rules for determining reimbursement may bias the choice of primary discharge diagnosis toward particular clinical entities. Finally, the diagnoses for hospital admissions (largely nonfatal illness) may reflect a milder spectrum than the diagnoses underlying fatal outcomes.

Although the study by Semenza et al. shows an overall increase in the number of discharge diagnoses of cerebrovascular disease during the heat, the number of specific diagnoses of stroke was not increased. This finding is difficult to square with multiple previous reports of increased cerebrovascular mortality during

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heat waves.[4] If cerebrovascular mortality (presumably due to stroke) is increased, it is difficult to understand why some increase in nonfatal strokes failed to occur. A further parallel analysis of both cerebrovascular deaths and cerebrovascular morbidity during the 1995 Chicago heat wave or during future heat waves would be of interest. Such studies could more fully evaluate the extent to which stroke and other cerebrovascular disease contribute to heat-related morbidity and mortality.

The striking heat-wave-related increase of 91 cases of acute renal failure observed by Semenza et al. is also noteworthy. Because of the magnitude of the increase and because of its specificity (limited to acute, not chronic, renal failure), the finding is unlikely to be due to coding errors. The authors attribute renal failure to prerenal azotemia, but this would involve a miscode, since prerenal azotemia is not true renal failure. They also offer the explanation of renal hypoperfusion in elderly persons who have inadequate left ventricular function. Although these factors may contribute, we should not forget that renal failure may also occur from rhabdomyolysis and myoglobinuria complicating heatstroke.[6] Further study of clinical records of these patients or others diagnosed with renal failure during a heat wave could do much to clarify the precise causes of the renal failure. In addition, the incidence and risk factors for acute renal failure should be studied in future investigations of heat-wave-related health effects.

One of the most striking findings of the Chicago study is the large number of patients with fluid and electrolyte abnormalities. This diagnosis was made in 1068 more patients during the heat wave than during the average control period. Taking extra fluids during the heat has been documented repeatedly as a factor that mitigates risk during the heat.[3,5] Encouraging increased fluid intake should be a major component of prevention programs.

Heat waves represent a type of natural disaster, and public health officials should take appropriate steps to limit morbidity and mortality. Air conditioning (including increasing time spent in air-conditioned places), taking extra liquids, and reducing physical activity during the heat are among the preventive steps for which the evidence for effectiveness is best documented.[3,5] Preventive efforts are most appropriately targeted toward high-risk groups, including the elderly, the poor, and persons with chronic illnesses or those unable to care for themselves.[1,3,5] Further studies of heat wave health effects can provide data needed to more fully develop and optimize strategies for prevention.

References

1. Jones TS, Liang AP, Kilbourne EM, et al. Morbidity and Mortality Associated with the July 1980 Heat Wave in St. Louis and Kansas City, Missouri. JAMA 1982; 247:3327–31.
2. National Center for Health Statistics. Electronic mortality data sets for 1980 and 1995.
3. Semenza JC, Rubin CH, Falter KH, et al. Heat-related deaths during the July 1995 heat wave in Chicago. N Engl J Med 1996; 335:84–90.
4. Kilbourne EM. Heat waves and hot environments. In: Noji E, ed. The Public Health Consequences of Disasters. New York: Oxford, 1997:245–269.
5. Kilbourne EM, Choi K, Jones TS, Thacker SB, and the Field Investigation Team. Risk factors for heatstroke: A case-control study. JAMA 1982; 247: 3332–3336.
6. Tan W, Herzlich BC, Funaro R, et al. Rhabdomyolysis and myoglobinuric acute renal failure associated with classic heat stroke. South Med J 1995; 88:1065–8.