Defining “Community” in Emergency Preparedness

TO THE EDITOR: The article by Braun and colleagues (1) underscores the need to accelerate integration of hospital disaster preparedness with community planning. The initial linkage for hospitals is with first responders: fire department, police department, and emergency medical service. Coordination of these services is usually provided by a governmental body, such as the Office of Emergency Management in New York City. It is clear, however, that, in a widespread disaster scenario, communities will need to be self-sufficient for at least the first 48 hours. This requires an expansion of the term “community” beyond hospitals and the agencies listed above. An integrated plan is needed that also includes skilled nursing facilities; chronic disease facilities; freestanding dialysis centers; correctional facilities; and, most critically, the community-based physicians. We learned on September 11, 2001, that having our physicians all rush to the hospital or to Ground Zero was not only nonproductive but also actually dangerous. In a pandemic, for example, it would be necessary to have physicians maintain office hours to triage less critically ill patients away from overburdened hospitals. Similarly, in a mass casualty event, coordination with skilled nursing facilities will expedite the rapid discharge of stable patients to provide surge capacity.

On Staten Island, New York, which has a population of 470,000 people, the Richmond County Medical Society in cooperation with the 2 hospital systems is developing linkages among all health care entities. Since communication is frequently the weak link in managing the response to a disaster, we have conducted a tabletop drill involving both hospitals, the Richmond County Medical Society, a New York State psychiatric facility, and the skilled nursing facilities. The next step is to repeat the drill and attempt to reach out to all physicians in the county. Other health care entities, such as hospice and home care, have been part of the initial planning and will be included in follow-up drills. I suggest that physician leadership drive this type of integration of resources, because the health of our communities is ultimately our mission.

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Reference

IN RESPONSE: Dr. Jarrett makes the point that integration among all medical assets in the community is a necessary step toward increasing response capacity and capability. We fully support this concept. Too often, disparate local health care organizations are not planning collaboratively for a coordinated, community-wide emergency response. Health care and response organizations expect to work together during an emergency but do not necessarily share plans or have a commonly understood framework for coordination under the urgency and uncertainty of a rapidly evolving incident, and major problems result. For example, during Hurricane Wilma, several hospitals had transportation agreements with the same ambulance companies, which became overwhelmed with requests for services (1).

As Dr. Jarrett suggests, physicians in private practice have a vital role in maintaining local access to care and preventing unnecessary influx of patients to hospitals. Accomplishing collaborative planning and drills, such as those undertaken by the Richmond County Medical Society, is important to prepare for an effective response. This planning group is similar to the emerging model of the “health care coalition” for emergency preparedness planning and response. The health care coalition is composed of health care facilities and other health and medical assets that form a single functional entity to maximize medical surge capacity and capability in a defined geographic area. It coordinates the mitigation, preparedness, response, and recovery actions of medical and health providers; facilitates mutual aid support; and serves as a unified platform for medical input to jurisdictional authorities (2).

The health care coalition is part of a tiered response-management system for integrating medical and health resources during large-scale emergencies. The federal Health Resources and Services Administration recently incorporated this tiered model into its Guidance for the National Bioterrorism Hospital Preparedness Program (3). This management framework describes a process for interfacing medical and health resources with widening levels of responders from the individual health care organization (tier 1) through the health care coalition (tier 2) to local (tier 3), state (tier 4), interstate (tier 5), and federal (tier 6) levels.

Dr. Jarrett suggests that physician leadership should drive this type of integration of resources. We disagree that common physician credentials make physicians the only uniquely qualified leaders for the initiative. Interested physicians should move beyond currently disjoined “disaster medicine” concepts to understand “medical emergency management” (4), with the scientific and professional qualifications for developing and managing complex systems. Understanding these concepts and principles will become even more important as the National Incident Management System (5) standardizes terminology and concepts across response disciplines and across the United States.

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IN RESPONSE: We thank Dr. Samaha for his comments and appreciate the point raised about the fairness of comparisons between the Mediterranean diets and a less intensively promoted low-fat diet. We think that it is fair to compare interventions with different grades of intensity as far as their context can be appropriately conceptualized. Our intervention was not designed as a tightly controlled, feeding trial. Instead, the PREDIMED study is a demonstration project conducted among free-living individuals that is similar to health-promoting lifestyle recommendations in the primary care setting. We conceptualized our intervention as the combination of enabling factors, such as providing healthy foods, and education plus counseling to achieve behavior change. The comparison group was given written instructions to follow a low-fat diet, which is common practice in primary care. However, we realize that the intervention in participants in the low-fat diet was indeed less intensive, and, because the study is ongoing, we have now designed group sessions with provision of written instructions in a similar way to what is done in the Mediterranean diet groups.

With respect to mean baseline levels of the main outcome variables, they were similar among the 3 groups. Systolic blood pressure ranged from 152 to 153 mm Hg; glucose levels in diabetic participants ranged from 7.9 to 8.8 mmol/L (142 to 159 mg/dL); HOMA indices in nondiabetic participants ranged from 4.1 to 4.2; low-density lipoprotein cholesterol levels ranged from 4.1 to 4.3 mmol/L (141 to 147 mg/dL); high-density lipoprotein cholesterol levels ranged from 1.3 to 1.4 mmol/L (45 to 47 mg/dL); and triglyceride levels ranged from 1.6 to 1.7 mmol/L (138 to 149 mg/dL). We acknowledge that the absolute magnitude of changes in individual risk factors associated with the Mediterranean diets was small. However, taken together, these changes represented a clinically significant reduction in coronary heart disease risk. Thus, compared with the low-fat diet group, the changes in 10-year absolute risk for CHD, estimated with the Framingham charts (2), were −1.7 percentage points (95% CI, −3.2 to −0.2 percentage points) and −1.8 percentage points (CI, −3.3 to −0.2 percentage points) in the Mediterranean diet with olive oil group and the Mediterranean diet with nuts group, respectively. In summary, a single behavioral intervention to improve a Mediterranean-style diet plus provision of healthy foods can induce changes in several risk markers in the short term, which represents a sizeable effect on overall cardiovascular risk. Hopefully, longer follow-up of the PREIDMED cohorts will magnify between-group differences in both food intake and risk factor changes that will eventually translate into diverse clinical outcomes.

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Note: This letter was written by the authors for the PREDIMED investigators.
Letters

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References

Estimated Glomerular Filtration Rate

TO THE EDITOR: The recent article about estimated glomerular filtration rate (GFR) by Levey and colleagues (1) is remarkable. The authors suggest that the new equation may still have some bias and especially may have less precision in patients with GFRs greater than 60 mL/min per 1.73 m². This is even more important if the GFR is greater than 90 mL/min per 1.73 m². In the paper’s Methods section, Levey and colleagues do not give the analytic coefficient of variation (CV) of their assays (Beckman Synchron CX3 [Global Medical Instrumentation, Inc., Ramsey, Minnesota] and Roche/Hitachi P module Creatinase Plus enzymatic assay [Roche Diagnostics, Basel, Switzerland]). This information is important especially for low or normal creatinine values. Indeed, the concept of critical difference is familiar to clinical biologists but should perhaps be reminded to internists.

The critical difference of a biological variable includes the analytic CV and the intra-individual CV and is defined as the smallest change in a biological result that is not due to chance (2). The critical difference is calculated as follows: 1.414 × 1.96 × (analytic CV² + intra-individual CV²) × 0.5. The intra-individual CV of serum creatinine is 4% (3). The analytic CV of serum creatinine varies among the assays used and laboratories. An analytic CV of creatinine as low as 2% is rare but conceivable (4). With these CV values, the lowest critical difference for creatinine is 12%. A creatinine value of 70.7 μmol/L (0.8 mg/dL) is, thus, not different from values between 62.2 μmol/L (0.704 mg/dL) and 79.2 μmol/L (0.896 mg/dL). However, we have illustrated (5), these differences are not negligible for GFR estimation if creatinine and GFR are normal because small creatinine changes induce large GFR variations within the range. If we take the example of a white 60-year-old man with a creatinine level of 70.7 μmol/L (0.8 mg/dL), the GFR is 98.6 mL/min per 1.73 m² with the Modification of Diet in Renal Disease (MDRD) Study equation. If creatinine values of 62.2 μmol/L (0.704 mg/dL) and 79.2 μmol/L (0.896 mg/dL) are introduced, the results of the MDRD Study equations will be 114.3 mL/min per 1.73 m² and 86.5 mL/min per 1.73 m², respectively.

The low precision of the MDRD Study equation, when GFR is normal, is also linked to the precision of the creatinine assay and to the biological variation of creatinine. This assertion is true for all creatinine-based equations. We think that an improvement of the precision of the creatinine-based equation may be illusive in a non-renal population. Clinicians should keep this fact in mind when they analyze an estimated GFR and when they longitudinally follow a serial of estimated GFRs in a patient with GFR greater than 60 mL/min per 1.73 m². It is perhaps more cautious to still give MDRD Study equation results as more than 60 mL/min per 1.73 m² and 90 mL/min per 1.73 m² without giving precise absolute values of GFR.

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References

IN RESPONSE: Dr. Delanaye and colleagues question whether the precision of the creatinine assay within the reference range is sufficient for accurate GFR estimation. The Beckman Synchron CX3 assay used during the MDRD Study had analytic CVs of 4.3% and 1.5% at creatinine values of 88.4 μmol/L (1.0 mg/dL) and 477.3 μmol/L (5.4 mg/dL), respectively (1). The enzymatic assay used to calibrate the MDRD Study laboratory to standardized creatinine had analytic CVs of 2.0% and 1.8% at creatinine values of 78.7 μmol/L (0.89 mg/dL) and 518.0 μmol/L (5.86 mg/dL), respectively, in 2004 (n = 194) and 1.6% and 1.1% at creatinine values of 88.4 μmol/L (1.00 mg/dL) and 339.5 μmol/L (3.84 mg/dL), respectively, in 2005 (n = 409). Thus, the analytic CV for the enzymatic assay is as low as or lower than that stated by Dr. Delanaye and colleagues.

We agree that the effect of imprecision in the serum creatinine assay and biological variation in GFR estimates is greater at lower values for serum creatinine (equal to higher values for estimated GFR) and that is one of several important reasons for lesser accuracy of higher GFR estimates (2). For these reasons, current recommendations are to report estimated GFR as a numerical value only when it is less than 60 mL/min per 1.73 m² and to report "greater than 60 mL/min per 1.73 m²" for higher values. We believe that this is sufficient for most clinical circumstances requiring the clinical assessment of kidney function. New filtration markers, such as cystatin C, and improvement in estimation equations may be required for more...
Accurate GFR estimation at higher values. Until then, if more accurate assessment of kidney function is required in patients with estimated GFRs greater than 60 mL/min per 1.73 m², measurement of the clearance of an exogenous filtration marker or creatinine is necessary.

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References

Successful Thalidomide Treatment of Persistent Chylous Pleural Effusion

Background: Disseminated lymphangiomatosis is characterized by proliferation of lymph vessels in soft tissues, viscera, or bones (1). Chylothorax is a serious manifestation and is typically refractory to several interventions, including chemical pleurodesis, surgical ligation of the thoracic duct, pleuropertitoneal shunt, radiation therapy, pleurectomy, chest wall resection, and antiestrogen drug therapy (2). It is associated with a poor prognosis. Interferon, which is antiangiogenic, has been used successfully in life-threatening hemangiomatosis of infancy and in some cases of disseminated lymphangiomatosis (3, 4). Thalidomide has immunomodulatory, anti-inflammatory, and antiangiogenic properties (5). In experimental models, thalidomide inhibits angiogenesis that is induced by vascular endothelial growth factor and fibroblast growth factor (5).

Objective: To describe the use of thalidomide in a patient with disseminated lymphangiomatosis and persistent chylous pleural effusion.

Methods and Findings: A 46-year-old woman presented with osteomyelitis of the left pelvis in 1996. Tiny cutaneous nodules with a yellowish content had initially appeared in 1990 over the thighs and had gradually spread to the buttocks and flanks and were surrounded by skin hyperpigmentation. Radiography and magnetic resonance imaging showed abnormal appearance of the sacrum, left iliac, pubis, T11-12 vertebrae, and surrounding fat tissue. Skin biopsy of a nodule showed lymphangiomas. Disseminated lymphangiomatosis involving the skin and bones was diagnosed. The osteomyelitis at presentation was culture-negative and was successfully treated with antibiotics. In May 2000, the patient developed a chylous pleural effusion (triglyceride concentration, 9.83 mmol/L [870 mg/dL]), which was drained by thoracentesis. Over the next 4 years, the chylous effusion recurred and required 35 thoracenteses, with removal of about 2 L of pleural fluid each time (Figure). One episode of pneumothorax occurred, requiring insertion of a chest tube. We tried several medical interventions: intramuscular injections of medroxyprogesterone acetate, 500 mg once monthly for 4 months; subcutaneous injections of interferon-α, 5 × 10⁶ U five times per week for 5 months; and a combination of the 2 treatments for an additional 4 months. In November 2003, we started thalidomide therapy, 200 mg daily, which stopped after 6 weeks because of side effects (mainly face puffiness). Three months later, we restarted thalidomide therapy, and the amount of pleural fluid removed decreased to 900 mL within a month. Thalidomide therapy was stopped after an additional 7 months of therapy in October 2004 because of side effects (face puffiness, paresthesias, and electromyographic changes). Chest radiography and computed tomography revealed thickened pleura in the base of the right lung. Skin and bone disease did not progress during this period. No additional thoracenteses were required as of November 2006. To summarize, the rate of pleural effusion accumulation during the 4 years was as follows: baseline, 1450 mL per month; progestosterone therapy, 1500 mL per month; interferon-α therapy, 750 mL per month; second baseline period, 2750 mL per month; and finally, a dramatic response to thalidomide as described.

Conclusions: In this patient, thalidomide seemed to lead to complete resolution of recurrent chylothorax, an effect that, to our knowledge, has not been previously reported. The time course is similar to that seen in children treated with interferon for hemangiomas who respond after 2 to 13 months of therapy (3). Indeed, interferon caused a partial response in our case. The similar time course supports the notion that the antiangiogenic properties of thalidomide are the basis for its beneficial effect (6). Further clinical experience and research is necessary to establish the efficacy of dosing, timing, and mechanism of action of thalidomide and its analogues (such as lenalidomide) in this rare disease.

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References
Thoracenteses (solid circles) and an episode of pneumothorax (asterisk) are shown. Drug dosages are stated in the text. The patient was followed until November 2006.


Correction

Correction: Update in Hospital Medicine

In a recent Update in Hospital Medicine (1), eligibility criteria for cardiac resynchronization therapy in Cleland and colleagues’ study (2) were reported incorrectly. Patients were eligible if they had had a left ventricular end-diastolic dimension of at least 30 mm indexed to height.

References