Hospital Resources for Pandemic Influenza

To the Editor: In their November 2007 article, Pandemic Influenza and Hospital Resources, Nap et al. evaluated hospital resources for pandemic influenza in the northern part of the Netherlands (*1*). Their results can be compared with those that I have described for the combined suburban communities of Roswell and Alpharetta, Georgia, USA (*2*). The Netherlands evaluation assumed that antiviral drugs will be available and will reduce hospitalizations by 50% and deaths by 30%. In view of the uncertainty of effective antiviral drugs and timeliness of vaccines, I did not estimate their effects. Nevertheless, several issues warrant comparison.

The plan for the Netherlands has no provisions for urgent care, i.e., parenteral fluids or antimicrobial drugs that are administered to ambulatory patients who are not hospitalized. Nap et al. may not perceive a need for enough beds to handle surge capacity. Allowing for 30% of beds to be used for patients with conditions other than influenza, they report a maximum availability of 232 beds per 100,000 population for pandemic influenza patients, and they estimate use of 72 beds per 100,000 in the pandemic model. In contrast, a maximum of 47 beds per 100,000 are available in Roswell/Alpharetta. Availability of beds in intensive care units, however, is identical for both regions, at 8 beds per 100,000 population.

The Netherlands plan calls for intensified treatment evaluation in 48 hours to withdraw care from patients who have little chance for recovery. Because most patients can be expected to have pneumonia and 2-organ failure (on average), a 50% mortality rate can be expected. In US hospitals, withdrawing care is difficult, even if mortality rates are expected to be 75% or 90% during acute illness with organ failure.

The pandemic influenza resource evaluation from the northern part of the Netherlands provides a useful contrast with at least 1 US hospital. The dramatic difference in bed availability highlights the potential challenges involved in local planning. The surge capacity limits in Roswell/Alpharetta led us to consider an alternative infusion center to provide care during an influenza pandemic.

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References

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Multidrug-Resistant *Acinetobacter baumannii* Osteomyelitis from Iraq

To the Editor: *Acinetobacter baumannii* identified in military settings is commonly multidrug resistant (MDR) (*1–3*). Tigecycline displays *A. baumannii* activity, but clinical experience is limited. We report a case of probable osteomyelitis caused by MDR *A. baumannii* and treated with tigecycline.

A 55-year-old man was transporting soldiers in Iraq when he sustained a grenade injury, in which material entered his anterior thigh and created a large posterolateral hip exit wound and an open left subtrochanteric femur fracture. He was flown to Germany; his wound was debrided, and the fracture was stabilized with an external fixator along with pins to his ilium and proximal and distal femur. A wound vacuum covered the exposed bones within the large soft tissue defect. He was stable upon transfer to our hospital 14 days after the injury; leukocyte count was 16,000/μL (reference range 4.5–11,000/μL), and erythrocyte sedimentation rate (ESR) was 44 mm/h (reference range 0–19 mm/h); blood cultures were not obtained. Plain radiographs showed an open femur fracture with gas in the soft tissue, shrapnel, and a gross deformity of the left iliac wing. ^{111}Indium-labeled leukocyte imaging confirmed increased activity in the left acetabulum, femoral neck, and surrounding soft tissue. Two days after his arrival, the external fixator (except for 1 pin in the distal shaft and 1 in the proximal femur) was removed, and an open reduction and internal fixation (ORIF) of the femur was performed. A cephalomedullary femoral rod and hip screw and 60 tobramycin-impregnated beads were placed into the hip joint; a wound vacuum was placed over the defect. A deep sample of the iliac wing was obtained, ground into a homogenate, placed aseptically on media, and observed for microbial growth; both coagulase-negative *Staphylococcus* and gram-negative rods grew in 1 culture. Both were considered pathogens of probable osteomyelitis based on exposed periosteum. Treatment with vancomycin plus ciprofloxacin was begun. After the gram-negative rods were identified as MDR *A. baumannii*, tigecycline (MIC 1.5) was substituted for ciprofloxacin (MIC>2). *A. baumannii* was susceptible to tobramycin (MIC≤2), intermediate to imipenem (MIC 8), and resistant to