Getting in line

Finally, a research paper makes the case that sick people waiting in line die at a rate similar to or slightly lower than the death rate for other sick people.[1] Will government now be able to say that queuing isn't bad for you? Let's put everyone on a waiting list to reduce the death rates for all diseases.

Richard Gruneir
Obstetrician and gynecologist
Leamington, Ont.

Reference
1. Naylor CD, Szalai JP, Katic M. Benchmarking the vital risk of waiting for coronary artery bypass surgery in Ontario. *CMAJ* 2000;162(6):775-9.

[One of the authors responds:]

I like Richard Gruneir's *reductio ad absurdum*. It is indeed frustrating that Canada's health care system has reached the point where we need to benchmark the toll of delayed care.[1] Ultimately, however, health professionals and administrators must get on with measuring and managing waiting lists, be it to contain the adverse consequences of poorly organized queues, or simply to provide better evidence to support arguments for additional resources.

David Naylor
Professor of Medicine
University of Toronto
Toronto, Ont.

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1. Naylor CD, Szalai JP, Katic M. Benchmarking the vital risk of waiting for coronary artery bypass surgery in Ontario. *CMAJ* 2000;162(6):775-9.

How long are TB patients infectious?

In their *CMAJ* paper on nosocomial tuberculosis, Kevin Schwartzman and Dick Menzies state that "if sputum or bronchial secretions are culture positive, then presumably they can still be disseminated into the air and transmitted to others."[1] This seems logical, but there is ample clinical evidence to show that once treatment with effective chemotherapy is started, the infectiousness of the patient becomes minimal within 2 weeks.

Tuberculosis (TB) is spread by the coughing up of minute droplets smaller than 2 μm. Suspension of these droplets as droplet nuclei necessitates the evaporation of any moisture in less than a fraction of a second. This causes the droplet nucleus to shrink to less than a thousandth of its original size. The concentration of anti-TB drugs in the saliva and bronchial secretions is the same as it is in the blood. With the evaporation of the moisture the dried-out tubercle bacillus in the droplet nucleus is exposed to a thousand-fold increase in the concentration of the drugs.

Schwartzman and Menzies quoted several papers by Richard Riley and his colleagues, dealing mainly with the infectiousness of untreated TB and the use of ultraviolet light in the control of infection. They failed to quote other papers by Riley and colleagues relating to the infectiousness of patients with TB once effective treatment is started.[2,3] Riley and colleagues found that the infectiousness of untreated patients with drug-susceptible organisms was much greater than that of patients on chemotherapy.

About the same time, Wallace Fox and coworkers showed that the tuberculin conversion rates of the close contacts of patients with open cavitary TB being treated with standard chemotherapy were the same regardless of whether the patients were treated in hospital or at home.[4–6] The only contacts who developed a positive tuberculin test or TB per se demonstrated a positive test either at the time of, or within 1 month of, diagnosis of the case. This implies they had inhaled tubercle bacilli before starting treatment and before the tuberculin test had time to convert. These observations made it clear that anti-TB therapy rendered patients virtually noninfectious within 2 weeks or so; it also persuaded most jurisdictions to eliminate compulsory segregation of subjects being treated for TB and removed the need for sanatoria.

Perhaps it will come as a shock to Schwartzman and Menzies to note the following statements in a highly regarded recent textbook: "for practical purposes patients can be regarded as being noninfectious two weeks after the start of treatment"[7] and "only untreated patients with sputum positive pulmonary TB are likely to be infectious."[8]

D. Ahmad
Internist
London Health Sciences Centre
London, Ont.
W.K.C. Morgan
Respirologist (ret'd)
London, Ont.

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[The authors respond:]

We agree that the infectiousness of TB patients diminishes rapidly once effective treatment is initiated. However, there is considerable evidence against dogmatic claims that patients are no longer infectious after 2 weeks of treatment.

Among the sentinel contributions of Wells and Riley was the finding that a single viable TB bacillus, once inhaled, is sufficient to produce infection.[1] Viable mycobacteria can persist in sputum for weeks after the onset of therapy,[2] and isoniazid-susceptible TB bacilli in droplet nuclei containing isoniazid were demonstrated to remain viable after 12 hours airborne.[3] Of course, mycobacteria need not survive this long to produce secondary infection if circumstances favour rapid dissemination (e.g., close proximity, no mask use, poor ventilation). These are precisely the circumstances once respiratory isolation is discontinued.

Smear-negative patients can and do transmit TB. Such patients accounted for 17% of secondary transmission in San Francisco.[4] There is also evidence that some mycobacteria are much more infectious than others. This was first suggested by Riley's finding of highly variable infection risks related to patients with similar clinical characteristics.[5] Valway reported a community outbreak where extremely high tuberculin conversion rates followed trivial contacts and demonstrated accelerated growth of the relevant isolate in a mouse model.[6] At present it is impossible to prospectively identify or differentially isolate patients harbouring such organisms.

Community studies suggested that within stable households, transmission to identified contacts (with long-standing antecedent exposure) greatly diminished or ceased once effective treatment was initiated. However, most of these studies had serious design flaws. The only randomized controlled trial of confinement versus outpatient treatment took place in India, where nearly all contacts evaluated were already infected.[7] It is inappropriate to extrapolate these data to the hospital setting. Hospitals now house sizeable numbers of patients infected with HIV, and other heavily immunosuppressed people. All of these individuals are at increased risk for infection and disease and most have never previously been exposed to TB.

The comments of D. Ahmad and W.K.C. Morgan also rest on the dangerous assumption that all infecting organisms are drug susceptible. Multidrug resistance is uncommon in Canada (1–2% of cases), but resistance to isoniazid was seen in 8.7% of Montreal cases.[8] In these patients, the response to standard therapy may be slower (or nonexistent, in multidrug resistance cases). The laboratory diagnosis of drug resistance cannot be established within 2 weeks. The release of smear-positive, drug-resistant patients onto general medical wards — after 2 weeks of "standard therapy" — has been documented to fuel nosocomial TB outbreaks in the United States, and the attendant risks cannot be overstated.[9]

Before hospitalized smear-positive patients move to general ward rooms, they must clearly respond to treatment. This entails a significant reduction in bacillary load, most reliably documented by conversion of the smear and supported by clinical parameters such as weight gain and resolution of fever. In some cases this may take 2 weeks or less; in others, much longer. Patients returning to stable households in which contacts have already been evaluated and treated (where appropriate) can indeed be discharged before smear conversion, provided there is clinical evidence of improvement and a suitable follow-up plan. As with other clinical decisions, we believe that a more reasoned approach is preferable to the indiscriminate application of a standard "recipe" — regardless of the (cook)books in which it has previously appeared.

Kevin Schwartzman
Dick Menzies
Departments of Medicine and of Epidemiology and Biostatistics
McGill University
Montreal, Que.

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