Response

To the Editor:

We appreciate Drs Shulimzon and Chatterji's concerns related to cross-contamination during flexible bronchoscopy (FB).[1] Despite monumental advancement in the technology, little attention has been paid to this potentially serious complication of FB.[2] In our opinion ignorance and circumvention are both equally responsible for the phenomenon. The former is strictly related to our reactionary enthusiasm to adopt advanced technology while detouring the fundamentals of bronchoscopy. Fellowship programs, manufacturers, and the wardens of infection prevention have collectively fallen short in educating bronchoscopists in relation to the risk of infection spread during the procedure. We as the interventional pulmonologists have conveniently overlooked the issue, considering its very low incidence rate while disregarding its underrecognition and underreporting.

We believe that the findings presented by Ofstead et al[3] are just a preview of the shortcomings of the current practice of FB. Introduction of "thin" and even "ultrathin" bronchoscopes with narrower channels is expected to make reprocessing and high-level disinfection ineffectual. Similarly, complexities and duration of advanced diagnostic as well as therapeutic procedures are likely to make the instruments more vulnerable to imperceptible damage: a possible nidus for biofilm formation. Akin to the practice of duodenoscopy, case reports of "superbugs" invading the bronchoscopes are already being quoted in the literature, further adding to our uneasiness.[4,5]

The issue of cross-contamination during bronchoscopy is many fold serious in the developing world. The cost of the instrument, its maintenance, disposable accessories, and even the reprocessing are prohibitive to safe practices in some instances.

While an option of sterilizing the instrument does exist it is impractical. Use of a disposable bronchoscope is indeed intriguing; but it is far from becoming a clinical reality for a variety of reasons.

Thus the findings by Ofstead et al caution us that it is high time for the community to exercise constant vigilance and surveillance related to cross-contamination during bronchoscopy. This could be achieved through proper education and a team approach among all the stakeholders in bronchoscopy. An implementation of scheduled surveillance comprising culture studies, bore scope examination of the instrument channel, and periodic inspection of the entire instrument by the manufacturer might aid in moderating the risk. Damaged instruments should be repaired only by an authorized vendor. Finally, the importance of strict adherence to the manufacturer's guidelines related to reprocessing and disinfection of the instrument cannot be overemphasized.

Atul C. Mehta, MD, FCCP
Thomas Gildea, MD, FCCP
Cleveland, OH

AFFILIATIONS: From the Advanced Diagnostic and Interventional Bronchoscopy Program, Respiratory Institute, and the Department of Pulmonary Medicine, Cleveland Clinic.

FINANCIAL/NONFINANCIAL DISCLOSURES: See earlier cited article for author conflicts of interest.

CORRESPONDENCE TO: Atul C. Mehta, MD, FCCP, Department of Pulmonary Medicine, 9500 Euclid Ave, A-90, Cleveland Clinic, Cleveland, OH 44195; e-mail: mehtaa1@ccf.org

DOI: https://doi.org/10.1016/j.chest.2019.03.006

References

1. Mehta AC, Gildea T. Burying our heads in the sand: cross-contamination during bronchoscopy. *Chest*. 2018;154(5):1001-1003.
2. Mehta AC, Prakash UB, Garland R, et al. American College of Chest Physicians and American Association for Bronchology consensus statement: prevention of flexible bronchoscopy-associated infection. *Chest*. 2005;128(3):1742-1755.
3. Ofstead CL, Quick MR, Wetzler HP, et al. Effectiveness of reprocessing for flexible bronchoscopes and endobronchial ultrasound bronchoscopes. *Chest*. 2018;154(5):1024-1034.
4. Humphries RM, Yang S, Kim S, et al. Duodenoscope-related outbreak of a carbapenem-resistant *Klebsiella pneumoniae* identified using advanced molecular diagnostics. *Clin Infect Dis*. 2017;65(7):1159-1166.
5. Klefisch FR, Schweizer C, Kola A, et al. [A flexible bronchoscope as a source of an outbreak with OXA-48 carbapenemase producing *Klebsiella pneumoniae*] [article in German]. *Hyg Med*. 2015;40(1/2):8-14 [English translation available online at, https://www.mhp-medien.de/zeitschriften/hygiene-medizin/open-access/.].

Non-TB Mycobacterial Infection-Bronchiectasis Nexus

To the Editor:

In the December 2018 issue of *CHEST*, Chalmers[1] asserts, "in recent years, nontuberculous mycobacterial infection (NTM) has emerged as a key etiology of

bronchiectasis in North America." More likely, NTM by these low virulence organisms is the opportunistic consequence of a predisposing pulmonary disease (PDPD). *Mycobacterium avium* complex, the most frequent NTM associated with bronchiectasis, is a congeries of saprophytic, free-living, soil- and water-dwelling bacteria, frequently present in municipal water supplies where it can be aerosolized. The nontuberculous mycobacteria PDPDs are bronchiectasis, old TB, COPD, cystic fibrosis, primary ciliary dyskinesia, and pulmonary alveolar proteinosis[2]; and it is a defining component of Lady Windermere syndrome (in which volitional cough suppression results in pooling of secretions).[3] These entities share a unifying, predisposing feature: stasis of (water-containing) secretions. (The pathogenesis of NTM in individuals who are immunosuppressed and persons with silicosis differs.) Were NTM causal, one would expect to find reported instances in which it preceded the onset of bronchiectasis[4]; to my knowledge, none exist. Based on the organisms' propensity to flourish in water, its presence in retained secretions in the PDPDs, and the observed sequence of events, an opportunistic rather than an etiologic role of NTM in bronchiectasis is more plausible.

Jerome M. Reich, MD, FCCP
Portland, OR

AFFILIATIONS: From the Thoracic Oncology Program, Earle A Chiles Research Institute.

FINANCIAL/NONFINANCIAL DISCLOSURES: None declared.

CORRESPONDENCE TO: Jerome M. Reich, MD, FCCP, 7400 SW Barnes Rd, A242, Portland, OR 97225-7007; e-mail: Reichje@isp.com

DOI: https://doi.org/10.1016/j.chest.2019.01.037

References

1. Chalmers JD. New insights into the epidemiology of bronchiectasis. *Chest*. 2018;154(6):1272-1273.
2. Chan ED, Iseman MD. Underlying host risk factors for nontuberculous mycobacterial lung disease. *Semin Respir Crit Care Med*. 2013;34(1):110-123.
3. Reich JM. In defense of Lady Windermere syndrome. *Lung*. 2018;196(4):377-379.
4. Evans AS. Causation and disease: the Henle-Koch postulates revisited. *Yale J Biol Med*. 1976;49(2):175-195.

Response

To the Editor:

I thank Dr Reich for raising the interesting "chicken or egg" question of whether nontuberculous mycobacterial infection (NTM) is a cause or consequence of bronchiectasis.[1] It is clear that nontuberculous mycobacteria are opportunistic pathogens that are able to cause disease in patients with impaired mucociliary clearance or host defense. When patients present for the first time, it is often difficult to ascertain whether the NTM has complicated existing structural bronchiectasis or whether the NTM itself has provoked an inflammatory response leading to the development or progression of bronchiectasis. Uncertainty surrounds many of our concepts of bronchiectasis pathogenesis because of the absence of an animal model or equivalent experimental model in which to test cause and effect.

Several related findings support the view that structural bronchiectasis is not a necessary prerequisite for the development of NTM pulmonary disease (NTM-PD). A recent detailed radiologic study of patients meeting the Infectious Disease Society of America/American Thoracic Society 2007 criteria for NTM-PD (n = 85) identified 38 subjects (44.7%) without bronchiectasis and primarily nodular features on CT scan, a finding that was replicated in an independent cohort.[2] Airway infection frequently precedes the development of bronchiectasis both in cystic fibrosis and in idiopathic bronchiectasis.[3] The emerging paradigm for the development of idiopathic bronchiectasis is that recurrent episodes of neutrophilic inflammation and infection in individuals without bronchiectasis (referred to as persistent bacterial bronchitis) leads to bronchiectasis.[3] The concept is therefore that impaired mucociliary clearance and host defense occurs prior to the appearance of structural bronchiectasis, and bronchiectasis is the consequence of this process rather than the primary cause.[3] The reported genetic architecture of nontuberculous mycobacterial disease, which includes involvement of ciliary genes, cystic fibrosis transmembrane conductance regulator, and connective tissue disease genes, also supports the view that these patients have impaired mucociliary clearance, which predispose to bronchiectasis, but that the impaired mucociliary clearance (and therefore potential susceptibility to NTM and other pathogens) precedes the requirement for structural bronchiectasis.[4]

For all of these reasons and others, all international guidelines for bronchiectasis regard NTM as a potential underlying cause of bronchiectasis and suggest to screen for NTM as part of initial testing.[5]