Commentary: I cannot breathe

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It was with great interest that we read the article by Mondal and colleagues\(^1\) describing their ex vivo tracheobronchomalacia (TBM) model for potential airway stent testing. Varying degrees of TBM were induced in ovine tracheas by performing either a single midanterior longitudinal incision or midanterior circumferential resections (25% or 50% along a 3-cm length). Forced expiration was simulated using a modulated closed pressure circuit. Laser generators and endoscopic video evaluation of laser-lit contours were used to evaluate changes in intraluminal cross-sectional area.

Excessive central airway collapse (ECAC) may be broadly classified as TBM, which is due to weakness of the anterolateral tracheal wall cartilage, or excessive dynamic airway collapse, which arises due to laxity of the posterior membranous tracheal wall with intact cartilage. Airway stents can be a part of ECAC management as a short-term measure to restore and maintain luminal patency and aid in diagnosis. However, existing stents have high rates of failure due to stent migration, stent fracture, and obstruction due to excessive mucus-plug formation or hypergranulation\(^2-4\) and are not amenable to permanent in vivo implantation. Airway stents composed of biocompatible materials are a promising avenue of exploration. Thus far, airway stent testing has been limited by the availability of standardized, high-fidelity ex vivo or in vivo testing models.

Mondal and colleagues\(^1\) model is in several ways superior to those previously described. Kaye and colleagues\(^5\) and Cao and colleagues\(^6\) described an ex vivo porcine model in which they used a combination of incisions, bisections, and manual crushing to induce artificial tracheomalacia across a spectrum of severity. The crushing process may permanently deform the trachea and thus distort the true extent of dynamic airway collapse. In contrast, Mondal and colleagues\(^1\) are able to demonstrate distinct, clinically meaningful grades of TBM using incisions/resections that are simpler, more precise, and more standardizable. Moreover, their use of laser measurements and computer-aided frame-by-frame analysis is an improvement on methods based largely on human observation.\(^5,6\)

However, there are several limitations of the described model. First, the midanterior incisions/resections described in this model produce ECAC that resembles only the saber-sheath and circumferential types of TBM.\(^2\) Crescent type TBM and EDAC,\(^2\) due to anterolateral and posterior wall weakness or laxity respectively, represent distinct morphological entities that are seen more frequently than anterior wall collapse. Second, the model does not account for the changes in intrathoracic pressures during the respiratory cycle that influence external tracheal tensile forces and behavior. It is also challenging for ex vivo stent testing to replicate underlying inflammatory and other pathophysiological processes affecting the native trachea in TBM that play an important role in altering mechanical properties such as compressibility and elasticity. Although the model is not perfect, we appreciate the work and sophisticated testing that can be utilized in future ex vivo and in vivo models of ECAC to aid not only stent development but also potentially device and surgical technique testing and advancement.
References


