Choosing an underfocused but highly relevant issue is a recipe for writing a remarkable article. D’Jouno and his colleagues accomplished their task while avoiding the temptation of recycling wisdom in their article [1]. For the time being, surgery in early non-small-cell lung cancer (NSCLC) and surgery-assisted oncological treatment in locally advanced NSCLC offer the best chances for cure [2]. The less-than-modest improvement in resectability rates witnessed presently makes operability ratio enhancement a key element in achieving better survivals. Consequently, more and more functionally borderline patients, who are prone to postoperative complications among which respiratory failure (RF) is the most deadly one, are offered lung resections. While RF is the primary cause of complication and in-hospital death [3] after lung cancer surgery, the cascade of events leading to the dreaded complication has more than one potential origin. Infective RF (iRF), where the first domino fall is usually a bacterial attack, presents a special subset within the gas-exchange catastrophes. It seems to be plausible to identify and annihilate the potential killers prior to lung resection. However, are the causative agents identifiable in advance and are they eradicable so as to prevent the complications? If the consequences are preventable, then why do they happen and who is responsible for not preventing them? The expression ‘preventable’ calls for abuse by medicolegal experts, health-care politicians and budget janitors. Originally, it was an honest self-exploring question in a search for improving performance, later on it was turned against us with the accusation: if it was preventable, how did it happen? The resulting pleuropulmonary sepsis might present itself as a post-resectional pneumonia. While postoperative thoracic empyema/sepsis caused iRF is a surgery-related complication, postoperative pneumonia-related iRF might share its origin with anesthesia, and the animate and inanimate hospital environment. The therapeutical challenges faced are not unlike to another animate and inanimate hospital environment. The pneumonic sepsis-related iRF might share its origin with anesthesia, caused iRF is a surgery-related complication, postoperative thoracic empyema and/or in the form of (nosocomial) pneumonia. While postoperative thoracic empyema/sepsis caused iRF is a surgery-related complication, postoperative pneumonia-related iRF might share its origin with anesthesia, and the animate and inanimate hospital environment. The therapeutic challenges faced are not unlike to another multifactorial complication, postoperative delirium [4]. The question is more complex than a simple ‘whom to blame’ game, nonetheless, because steering the RF treatment (decision making and delivering therapy) is usually taken over by intensive therapists and pneumonologists. While neither pathogenesis nor outcome is primarily a surgery-defined issue, responsibility for prevention of these unfavorable events remains in our domain. The authors’ statement on the heterogeneity in the etiology of respiratory complications highlights the importance of a systemic approach in RF etiology. One has to make a clear distinction between three different scenarios among a falling domino-like chain of events of bacterial infection-triggered processes. Above that, iRF must be interpreted in the wider context of acute lung injury. Microbial infection in iRF might be the starter, a primary mover, or it may be playing a secondary role, a pusher in case of an already ‘failing’ postoperative patient. There is a third picture, where infection attacks a patient already downhill from some other reason (multi-organ failure), where microbes are only the sad but clinically quite insignificant terminators. Not only should the prevention of iRF in the form of antibacterial prophylaxis mirror these differences, but forthcoming studies will also need clear-cut categories. The therapy of iRF is a clinical-picture-driven one, supported by bacteriological findings. The case scenarios available are: (1) bacteriologically proven, (2) bacteriologically unproven but supposedly bacterial, (3) viral, (4) mycotic in origin, and (5) other, non-specified. The main question remains open: does airways colonization (AWC) contribute to iRF and if yes, to what extent? Is AWC an indicator of vulnerability/susceptibility to infection/(nosocomial) pneumonia? Is AWC a cause and/or an effect? Theoretically, there are four possible outcomes of subsequent events, forming a matrix: (1) pneumonia (± iRF) caused by AWC (2) pneumonia (± iRF) in spite of lack of colonization, (3) no pneumonia in spite of proven presence of AWC, and (4) a negative—negative outcome. There is one more case, an out-of-matrix occurrence, in fact, the most worrying one from this point of investigations: (5) pneumonia (± iRF) other than identified AWC agent(s). In the latter case, preoperative antibiotic prophylaxis is not only unnecessary, but also gives a false sense of safety. The following decision is should we postpone surgery for a positive AWC culture?

The authors prove an expected respiratory complication rate of about 2:1 in the colonized—non-colonized groups, respectively. AWC is identified clearly as an independent risk factor for the development of iRF but not necessarily the causative organisms themselves. It is a strong indicator of the fragility of the patient and a rather unreliable guide in initiating any antibiotic treatment before culture-based results arrive if iRI occurs. Preoperatively identified AWC agents and those causing sepsis afterward might be different, but they share the same host. The iRF is frequently polymicrobial, and the studies are primarily descriptive in this regard; hence, the possible independent and common effects of the isolated bacteria are difficult to clearly elucidate [5]. We have simply too few data about the role of viral infections, especially in cytomegalovirus (CMV) in neo-adjuvant therapy patients, per se immunocompromised hosts.
This is an unexplored territory, indeed. Should we implement peroperative bronchial smears in the same manner that thrombembolic prophylaxis became a standard element of care? Is there a role for the technique of bidirectional stapled closure of bronchial stumps and consequently lower risk of spillage in the reportedly lower overall complication rate of video-assisted thoracoscopic surgery (VATS) lobectomies [6]?

Although it is unfair to disclose the killer in an invited preface to a detective story, it must be agreed with the main message of this article that there is an obvious need for an international follow-up study to assess the role AWC in the pathogenesis of iRF. However, a forthcoming project, requires participation of a clinical microbiologist, who is familiar with the different sampling techniques used in the laboratory diagnosis of lower respiratory tract infections [7—9]. A sharp distinction must be made between preoperative and postoperative AWCS where iRF is concerned [10]. The study in question needs a powerful scientific capacity and firm vision: the authors have presented themselves as potential motors of a project such as this.

References


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