The Timing of Tracheotomy
An Evolving Consensus

In 1807, Napoleon Bonaparte’s great-nephew died of diphtheria, prompting the Emperor to offer a prize for research to combat that illness, thus stimulating work on airway management. Tracheotomy was identified as useful therapy and became the standard of care. When Bouchut suggested translaryngeal intubation as a better alternative, he was condemned by an investigative committee of the Paris Academy. However, an ocean away, O’Dwyer in New York routinely maintained tracheal intubation for diphtheria for up to two weeks. Thus, not only have both techniques been used for more than a century and a half, but clinicians have argued their relative merits for a century.

Because of the complexity and severity of disease in critically ill patients, definitive studies of airway management have been hard to obtain and controversies therefore difficult to resolve. Instead, the development of a consensus has been an evolutionary process with conclusions based on the preponderance of evidence from a series of imperfect clinical and animal studies. The article by Colice and colleagues in this issue (see page 877) adds further weight to the evolving consensus that tracheotomy is not routinely medically indicated in the patient requiring a temporary artificial airway.

Although convictions have been strong, evidence has often been lacking as to the need for converting a translaryngeal intubation to a tracheotomy. A survey of critical care practice revealed that while most clinicians felt that a tracheotomy should be performed after a fixed period of translaryngeal intubation, the “ideal” time varied between one and four weeks with marked differences depending on primary specialty.

Colice et al confirm that translaryngeal intubation routinely causes laryngeal injury. Pressures at the interface of the tube with the mucosa covering the posterior laryngeal cartilages, the arytenoids and the cricoid, far exceed mucosal perfusion pressures. It is not surprising, therefore, that the authors found a 94 percent incidence of typical mucosal ulcerations along the posterior cords.

The article by Colice and coauthors reaches several important conclusions. The lack of correlation between severity of injury as seen at the time of extubation and long-term complications argues against the practice of basing a decision on when to perform a tracheotomy on direct laryngoscopic findings. The laryngeal mucosa heals remarkably quickly and even severe ulcers often resolve rapidly. Deciding to perform tracheotomies because of laryngeal pathology would have resulted in unnecessary tracheotomies.

A second important finding of Colice et al is that no patient developed progressive laryngeal scarring or stenosis during follow-up. Most studies have concluded that laryngeal stenosis is rare following intubation, with the exception of the paper by Whited which Colice discusses. This further confirmation of the rarity of the problem is comforting.

A third important conclusion is that laryngeal pathology was significantly worse in patients who had undergone a tracheotomy 24 hours previously than in those who had been extubated without a tracheotomy. While this confirms experimental and clinical evidence that tracheotomy may make laryngeal injury worse, it must be regarded with some caution since the full explanation of why the laryngeal injury worsened so quickly following tracheotomy is not demonstrated.

We know that tracheotomies have direct financial costs and medical risks. Colice et al add to the evidence that we cannot pick an arbitrary time or even use direct laryngoscopy as an indication for the medical necessity of a tracheotomy.

What, then, is the current role of tracheotomy? It appears to be in nursing the patient: ease of nursing care, patient comfort, ability to communicate, and appearance to the family. The timing of a tracheotomy should be driven by these issues, and it should be performed when the patient is medically stable, rather.
Aerosolized Pentamidine for Pneumocystis carinii Pneumonia

Aerosolized pentamidine has proven to be safe and effective in the prevention1 and treatment2,3 of Pneumocystis carinii pneumonia (PCP) in patients with the acquired immunodeficiency syndrome (AIDS). The aerosolized form of pentamidine offers the advantage of targeting delivery to the alveoli while reducing drug levels in serum and solid organs.4 This selective distribution is appropriate because the lung is the primary organ infected by Pneumocystis carinii, whereas the liver and kidney are the most common sites of systemic drug toxicity.5

Despite its pharmacologic properties and proven efficacy, aerosolized pentamidine has at least three potential disadvantages in patients with PCP.6 The first is that the amount of pentamidine reaching the lung parenchyma depends upon the number and size of particles generated by nebulizers used to aerosolize the drug. Alveolar deposition is enhanced with particles of 1-2 μm, whereas airway deposition, which may be associated with coughing, occurs with larger particles.7 Indeed, the relative effectiveness of aerosolized pentamidine in some studies8 as compared with others9 may be due to the kind of nebulizers used.

A second potential disadvantage of aerosolized pentamidine is that alveolar deposition is influenced by the rate and depth of breathing and by differences in ventilation within the lung. That more ventilation goes to the lung bases when pentamidine is inhaled in the upright position probably accounts for the finding of recurrent PCP in the upper lobes of patients receiving the aerosol as prophylaxis.8 In order to achieve more even distribution of the drug throughout all lung zones, patients should breathe at a fast rate, breathe higher doses, or periodically breathe from residual volume during aerosol administration.9

The third potential disadvantage of aerosolized pentamidine is that Pneumocystis carinii infection is not always limited to the lungs. Systemic dissemination of the organism was well-known in the pre-AIDS era,10,11 and it has been even better reported in patients with AIDS. To date, Pneumocystis carinii has been noted in the skin,12 eyes,13 ears,14 gut,15 lymph nodes,17 kidney,18 spleen,19 and liver of such patients. In this issue of Chest, (see page 949) Hagopian and Huseby describe Pneumocystis carinii hepatitis and chorioretinitis in an AIDS patient receiving aerosolized pentamidine prophylaxis. They also speculate that the true incidence of extrapulmonary infection may be higher than previously realized due to the infrequent use of autopsy among AIDS patients.

These reports of disseminated Pneumocystis carinii should prompt physicians to aggressively evaluate patients with suspected extrapulmonary involvement even if their lungs are clear of infection, as Hagopian and Huseby recommend. Furthermore, patients with extrapulmonary Pneumocystis carinii should receive systemic therapy in addition to or in place of aerosolized pentamidine. Nevertheless, the use of aerosolized pentamidine in treating and preventing PCP need not be limited, as others have suggested. Whatever its true histologic incidence, extrapulmonary infection from Pneumocystis carinii was not documented on clinical or laboratory grounds in any of the approximately 240 patients participating in a multicenter double-blind study of aerosolized pentamidine compared with trimethoprim-sulfamethoxazole (AB Montgomery, personal communication). The lack of disseminated disease in this study and the recent prophylaxis trial1 demonstrate that aerosolized pentamidine may be indicated in AIDS patients with PCP.

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REFERENCES


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