Retroperitoneal Air Dissection and Mechanical Ventilation

To the Editor:

I read with great interest the article by Powne and associates on "Retroperitoneal Air Dissection Associated with Mechanical Ventilation" (Chest 69:739-742, 1976), in which they described three patients who developed pneumoretroperitoneum during mechanical ventilation. This article requires some additional comment concerning the pathogenic mechanisms of the disorder reported. It must be emphasized (1) that retroperitoneal air dissection as an adverse effect of mechanical ventilation cannot be imagined without antecedent mediastinal emphysema and (2) that, under certain circumstances, production of mediastinal emphysema in a subject receiving mechanical ventilation may be the result of a mechanism other than "expansion rupture" of the lung, as will be outlined herein.

Pathophysiologic Considerations

Introduction of air into the mediastinum during mechanical ventilation is a phenomenon commonly attributed to "barotrauma" brought about by overinflation and "expansion rupture" of the lung by a mechanism elaborately investigated by Macklin1,2 and by Macklin and Macklin3 decades ago. This mechanism, with reference to which Powne et al explain their observations, is amply discussed in the original report and need not be reexplicated here.

Nevertheless, introduction of air into the mediastinum in subjects receiving mechanical ventilation may be due to a second mechanism quite different from the one mentioned previously. With fractures of the ribs, the parietal pleura, the visceral pleura, and the underlying lung may be lacerated. Whenever pressure in the airway rises above zero, air will pass from the injured lung into subcutaneously lacerated soft tissues via the pleural space, with or without radiographically apparent pneumothorax,4 and may, after distending the soft tissues of the chest and the neck, spread along fascial planes to enter the mediastinum at the neck. This mechanism of development of mediastinal emphysema was first described by Sauerbruch5 and was later confirmed by other authors.6,7 The mechanism is valid for subjects breathing spontaneously, as well as for patients receiving mechanical ventilation; and it may be set in action not only by fractured ribs but also by penetrating thoracic injuries from the exterior,4 especially in case of preexisting pneumothorax.7

When sufficient pressure develops, air always escapes from the mediastinum following one of three paths,6 one of which leads through the diaphragm, about the aorta and the esophagus, to the retroperitoneal tissues, with the consequence of pneumoretroperitoneum.

Discussion

Overexpansion of the lung during artificial ventilation has long been an interesting subject in critical care medicine; however, in one of the cases presented by Powne et al, one should be careful in accepting the interpretation offered in their article. I hesitate to believe that in each of the patients reported, a so-called "expansion rupture" of alveoli or small airways was the causative event for the production of mediastinal emphysema. This may be true for cases 2 and 3 but not for case 1.

Case 1 represents a patient with fractures of the second through fourth ribs posteriorly on the right. He was intubated to receive mechanical ventilation because of increasing hypoxemia. During the first 32 hours of ventilation, the peak inspiratory airway pressure rose from 28 to 45 cm H2O, presumably as a consequence of decreasing pulmonary compliance. Then therapy with positive end-expiratory pressure (PEEP) was instituted. Thereafter, four hours later, subcutaneous emphysema in the supraclavicular region was noted. Did the change from zero end-expiratory pressure to PEEP, together with poor pulmonary compliance, open a bronchopleural fistula in the damaged region of the thoracic cage and cause egress of air into the overlying subcutaneous tissues? I should think so. The close anatomic relationship between the supraclavicular soft tissues and the damaged part of the thoracic cage easily explains why the first traces of air escaping from the pleural space appeared in the supraclavicular region. Unfortunately, the report on case 1 by Powne et al is not detailed enough to answer all questions. But it may, anyhow, invite the attention of those practicing mechanical ventilation to an important, but less appreciated, aspect in the complex field of "pulmonary barotrauma."

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References

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Late Thrombosis of the Aortic Björk-Shiley Prosthesis

To the Editor:

Thrombosis of prosthetic cardiac valves has remained a serious hazard ever since artificial valves have been used. Despite the excellent hemodynamic performances of the Björk-Shiley prosthesis in the aortic position, this prosthetic valve has not been proved free of clotting problems, as demonstrated by the article of Fernandez et al1 in the July 1976 issue of Chest.

The mechanism of thrombosis of a valve is certainly a complex one; however, two major factors contribute to the problem: (1) lack of adequate anticoagulative therapy, and (2) implantation of small-sized prostheses. It is evident that five of the patients reported by Fernandez et al1 had a prosthesis of size 23 or less. Especially in the aortic position, the smaller aperture may become inefficient for flow in small-sized Björk-Shiley valves, resulting in stasis at this part of the prosthesis. This can especially happen after insufficient decalcification of the aortic annulus and, furthermore, in patients with severe subaortic muscular hypertrophy or when the prosthesis has been inserted in a slightly tilted position in regard to the aortic annulus. To allow sufficient flow on both sides of the tilting disk, it is important, whenever possible, to insert a prosthesis of size 25 or more. This is practically always feasible after careful and complete decalcification of the aortic annulus and the subvalvular area. If necessary, the aortic annulus can be split in front of the commissure between the left and right coronary cusps.

Among some 300 isolated aortic valvular replacements using the Björk-Shiley prosthesis, we have seen only two patients with a clotted prosthesis, both not taking anti-coagulant drugs. That careful anticoagulative therapy is mandatory, even with the hemodynamically excellent Björk-Shiley prosthesis, had been advocated already in early reports about the experience with this type of prosthesis.2,3

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REFERENCES

To the Editor:

It has been well established that inadequate anticoagulation is a factor that contributes to thrombosis of the aortic Björk-Shiley prosthesis. Inadequate removal of calcific deposits may also be a factor. In our experience a critical factor that has not been emphasized is the positioning of the prosthesis. In our opinion, the relation of the smaller aperture of this tilting-disk valve to the surrounding structures is of crucial importance. In all of our seven cases of thrombosed aortic Björk-Shiley prostheses, the valve was positioned with the smaller aperture toward the septum (1970 to October 1973).

Since that date, the prosthesis has been positioned