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To the Editor:

Regarding our publication on “Retroperitoneal Air Dissection Associated with Mechanical Ventilation” (Chest 69:739-742, 1976), Dr. Siemoneit has added interesting viewpoints of importance for further understanding of various forms of interstitial emphysema, a condition which has become a quite common occurrence in intensive care units.

We did indeed consider the explanation that Siemoneit discusses for the dissection of air in interstitial tissues of our patient; however, the appearance of subcutaneous air without simultaneous pneumothorax in patients with chest trauma due to fractured ribs particularly occurs in elderly patients with pleural adhesions. Our patient was a 22-year-old man without previous pulmonary disease whom we expected would have intact pleural cavities without adhesions. Dr. Siemoneit has brought to our attention that subcutaneous air can be the cause of mediastinal emphysema, rather than vice versa, which is another interesting possibility. We are grateful to him for these comments.

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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 al 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 al 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 anticoagulant 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...
with the smaller aperture towards the left coronary cusp, which minimizes stasis around the area of the hinge. As a result of this, not a single case of a thrombosed Björk-Shiley prosthesis has occurred in 270 consecutive aortic valvular replacements (October 1973 to October 1975), despite the use of all prosthetic sizes and in many cases with marginal anticoagulation.

We recognize that longer follow-up is needed, but so far our experience with the valve so oriented has been good.

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Isoniazid with Corticosteroids in Patients Infected with Mycobacterium tuberculosis

To the Editor:

Dr. Eshelman's recent communication raises several interesting questions about the role of preventive treatment with isoniazid for individuals infected with Mycobacterium tuberculosis who are receiving therapy with corticosteroids.

There is evidence that therapy with corticosteroids can exert a variety of undesirable effects on the course of tuberculous infection and disease.2-4 Unfortunately, the exact risk to the patient is not well defined. This results, in part, from the varying dosages and preparations of corticosteroids prescribed, from the wide variety of underlying disease for which therapy with steroids is given, and from the other concomitant risk factors for tuberculosis present in some of these individuals. The use of alternate-day corticosteroid regimens, which seem to leave delayed hypersensitivity intact5 and to be attended by fewer infectious complications,6 further confounds the issue.

In the absence of data clearly indicating that the risk of therapy with corticosteroids for persons infected with tubercle bacilli is related to dosage or is reduced by alternate-day therapy, we believe that current guidelines for preventive therapy of tuberculous infection7 should be followed. Patients with a positive tuberculin test who are receiving prolonged therapy with corticosteroids should be considered at increased risk of developing tuberculosis, whether the steroids are administered on a daily or alternate-day basis, and should, therefore, be considered as a priority group for receiving preventive therapy with isoniazid. This preventive approach recognizes the potential consequences of tuberculosis for these patients, as well as for persons exposed to them.

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REFERENCES


Granulomatous Pleuritis Secondary to Blastomycosis

To the Editor:

Patients with pleural effusion of unknown etiology frequently undergo needle biopsy of the pleura. The finding of granulomatous pleuritis with or without caseous necrosis is considered strong evidence for a tuberculous cause of the effusion.1,2 The following case is presented to demonstrate that diseases other than tuberculosis may be responsible for granulomatous pleuritis.

CASE REPORT

A 62-year-old woman was referred to Confederate Memorial Medical Center for evaluation of a left pleural effusion. She had been well until two months prior to admission, at which time she developed a productive cough, malaise, chills, fever, and left-sided pleuritic chest pain. At this time the patient was treated with penicillin and rapidly became asymptomatic; however, several weeks later, she noted the insidious onset of dyspnea on exertion and fatigue. A subsequent chest x-ray film showed a large left pleural effusion.

Physical examination at the time of admission revealed an afibrile chronically ill appearing woman with signs of a left pleural effusion. Several smears of sputum for acid-fast bacilli and fungi and the skin test with intermediate-strength purified protein derivative of tuberculin (PPD) were negative.

A thoracentesis yielding 1,500 ml and a pleural biopsy were performed. The pleural fluid was yellow and had a protein level of 6.6 gm/100 ml, a glucose level of 117 mg/100 ml, and a lactic dehydrogenase level of 226 milliunits/ml, with a simultaneous serum protein level of 7.7 gm/100 ml and a lactic dehydrogenase level of 192 milliunits/ml. The cell count of the pleural fluid was 3,150/cu mm, and 85 percent were small lymphocytes. The pleural biopsy revealed multiple granulomas with minimal caseous necrosis centrally. Smears

CHEST, 71: 3, MARCH, 1977

COMMUNICATIONS TO THE EDITOR 433

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