efficient way to monitor the effects of regimen changes? Unless the pulmonologist has experience taking care of diabetic patients in the office, he or she probably will not be as effective as the generalist who takes care of these patients on a frequent basis.

The same holds true for important areas of "primary care" such as prevention and screening. If a pulmonologist spends his day doing bronchoscopies and managing ARDS, do you want the same doctor advising you about prostate cancer screening or tick removal? Though pulmonologists frequently take care of the "whole patient" in the ICU, let us not assume that they can take care of the whole patient in the office unless they have the knowledge and experience to do so.

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REFERENCE

1 Block AJ. What is a primary care physician? [editorial]. Chest 1994; 105:1634

Amiodarone and the Development of ARDS After Lung Surgery

To the Editor:

We read with interest the article by Van Mieghem et al1 and the editorial comment by Drs. Mathru and McDaniel,2 which were published in the June 1994 issue of Chest. In 1985, we reported our experience with intravenous amiodarone. We used a dose of 450 mg administered over 8 h in the treatment of ten patients who had uncontrolled malignant ventricular arrhythmias.3 This treatment was successful in all cases and four patients are still surviving. While amiodarone has a negative inotropic, chronotropic, and dromotropic effect, none of our patients developed ARDS at this dose, which was considerably lower than the dose used by Van Mieghem et al.1 Their three cases of ARDS occurred in patients with total pneumonectomies rather than lobectomies, and in these patients the cardiac output remained constant. The single lung would therefore be exposed at least transiently to an even higher amount of a potentially toxic drug, and this may be a factor in the high rate of pulmonary complications. Amiodarone has been shown to control successfully atrial arrhythmias.4,5 In the cases presented, there were less instances of atrial arrhythmias in the treated patients. Lower doses of the intravenous drug or initiation of oral therapy before surgery may well prevent atrial arrhythmias and avoid the development of ARDS.

Our current method of using amiodarone in both ventricular and atrial arrhythmias is to give the patient an initial dosage of 400 mg and then 200 mg daily. If the arrhythmia recurs, then we give another increment of 400 mg daily on an as-needed-basis until the arrhythmia is controlled.

Edward P. Rose, MD, and Larry E. Alves, MD, FCCP, Belleville, Illinois

REFERENCES

2 Mathru M, McDaniel L. Primum non nocere: is the therapy worse than the disease? Chest 1994; 105:1634-36

To the Editor:

We read with interest the article in the June 1994 issue of Chest by Van Mieghem and coworkers (Chest 1994; 105:1642-45) on the development of ARDS after pneumonectomy and the possible role of amiodarone in its cause.

In 1992, we reported in Intensive Care Medicine1 about a patient who developed amiodarone pulmonary toxicity after pneumonectomy and who was treated with low-dose amiodarone 200 mg/d. In spite of this low dosage, amiodarone pulmonary toxicity (APT) developed in the patient 3 months after starting therapy with amiodarone, which required prolonged mechanical ventilation.

It was postulated that in the remaining lung in thin patients who have little adipose tissue, a drug concentrating effect might be involved causing APT and that dosages in case of pneumonectomy should be adapted to this particular situation. In the accompanying editorial,2 this was acknowledged as a possible new explanation for APT. It is possible that also the patients described in the study by Van Mieghem were thin but these data were not presented. On the other hand, the interval free period in our case was much longer than in the presented cases by Van Mieghem. This might be the consequence of the differences in initial loading strategy.

Our patient developed APT after 18 g amiodarone in total in 3 months. The initial loading dosages of the patients presented by Van Mieghem were much higher and could have contributed to the faster onset of APT.

We agree with the conclusion by Van Mieghem et al that amiodarone may be potentially dangerous especially after pneumonectomy.

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REFERENCES


To the Editor:

Drs. Alves and van der Zeijden suggest that the reduction in a lung parenchyma may be a predisposing factor for the development of ARDS when a standard dose of 1,200 mg of amiodarone is used in patients after a pneumonectomy. We agree and made the same suggestion at the end of our article (Chest 1994; 105:1642-45).