Pregnancy Complicated by Cardiac Valvular Disease and Hypertension

To the Editor:

I read with interest the case report and discussion by Dunnick et al., which appeared in the December 1991 issue of *Chest*. The authors described the management of a patient with valvular heart disease complicated by pregnancy-induced hypertension. I would like to make several comments.

First, I question the desirability of prolonging a 34-week gestation in a woman with pregnancy-induced hypertension. Clearly, this would not be of any particular benefit to the mother; its only purpose would be to permit further fetal maturation and thus improve neonatal outcome. However, the incidence of respiratory distress syndrome in a 35-week-gestation newborn is as low as 3 percent, and the neonatal mortality is still lower. Thus, delivery could have been accomplished prior to maternal hemodynamic deterioration. Further, there is no need to invoke an "arbitrary time of 35 weeks' gestation" to terminate the pregnancy; objective evidence of fetal lung maturity, the primary determinant of neonatal survival, can be reliably obtained through amniocentesis and determination of the lecithin-sphingomyelin ratio.

Second, it is well established that pregnancy-induced hypertension is associated with significant intravascular volume contraction, proportional to the degree of hypertension. Thus, the use of diuretics may be associated with further deterioration. It may be, in fact, that the diuretics performed in this patient led to a decrease in cardiac output, which impaired uteroplacental blood flow and led to fetal distress. As Gianopoulos points out in his commentary, placement of a pulmonary artery catheter would have permitted more goal-oriented therapy.

Finally, it is time to lay to rest the belief that beta-adrenergic blockade during pregnancy can lead to intrauterine growth retardation. In studies of atenolol, oxprenolol, labetalol, and propranolol, there is no evidence of any significant effect on fetal growth. Intrauterine growth retardation is far more likely to be due to the underlying maternal condition requiring beta-blockade than to the therapy itself.

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REFERENCES


Exposure of Health-Care Workers to Aerosolized Pentamidine

To the Editor:

We read with interest the report by O'Brien and Smaldone, which appeared in the June 1992 issue of *Chest*. It was alarming to read that systemic absorption of pentamidine occurred in health-care workers (HCWs) administering infrequent aerosol pentamidine (AP) treatments (4 to 50 per month) in their institute.

In Toronto we have a centralized community-based outpatient treatment facility, which has enrolled over 1,200 patients and administered over 31,000 individual treatments since its opening in May 1989. This center is served by 6 dedicated HCWs who administer up to 50 AP treatments per day, 6 days a week (ie, up to 1,200 treatments per month). Individual AP treatments are administered by the Canadian Multicentre Trial Protocol of 80 mg of pentamidine every 2 weeks following 5 loading doses over a 2-week period. We have also been concerned with the possibility of acute or chronic low-dose exposure to pentamidine.

Methods of administration are essentially similar at our institute to those reported by O'Brien and Smaldone, apart from the different nebulizer systems used (Fisons [Fisons Corporation, Rochester, NY] vs Respigrad [Marquest Medical, Englewood, Colo]). Individual treatments are, however, self-administered by patients in negative-pressure treatment rooms with external exhaust and high-volume high-efficiency particulate air filtration units. Gloves and masks are worn by HCWs only for drug reconstitution.

Spot urine samples were obtained from each of our HCWs on two separate occasions. Three patients consented to spot urinalysis immediately prior to therapy (positive control), and two health-care control subjects were enrolled (negative control) (Table 1). Samples

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Table 1—Results of Spot Urinalyses

<table>
<thead>
<tr>
<th>Sample</th>
<th>Subject</th>
<th>P, ng/ml</th>
<th>Cr, mg/ml</th>
<th>P/Cr, ng/mg/ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>HCW</td>
<td>2.62</td>
<td>1.62</td>
<td>1.625</td>
</tr>
<tr>
<td>2-12</td>
<td>HCWs</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<td>13</td>
<td>Patient</td>
<td>30.2</td>
<td>1.17</td>
<td>25.8</td>
</tr>
<tr>
<td>14</td>
<td>Patient</td>
<td>84.7</td>
<td>2.94</td>
<td>28.8</td>
</tr>
<tr>
<td>15</td>
<td>Patient</td>
<td>30.7</td>
<td>5.2</td>
<td>5.97</td>
</tr>
<tr>
<td>16, 17</td>
<td>Controls</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

P = pentamidine; Cr = creatinine; P/Cr = correction of pentamidine level for urinary creatinine concentration.

were assayed by high-pressure liquid chromatography by Dr. G. Smaldone (Stony Brook, NY), who was blinded to the source of individual samples. Only in one sample from one of our HCWs was pentamidine detected at a level of 1.625 ng/ml, approximately the same as the lower exposure levels in the study by O'Riordan and Smaldone. During the sampling day this HCW had been present during 35 individual treatments and had been present during some 780 treatments over the previous month. A subsequent sample on this HCW was negative later the same week.

We consider administration of AP via the Fisoneb system, even in a high-volume clinic such as ours, to have negligible effects on individual HCWs. We agree that low-dose environmental exposure is inevitable, and that occasional higher levels due to environmental splatter should be expected, although they can be minimized.

More important, we believe that breath-activated nebulizer systems, such as the Fisoneb system, are preferable because their very design significantly reduces environmental spillage, since they turn off after inspiration ceases or if coughing occurs.

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1 O'Riordan TG, Smaldone GC. Exposure of health care workers to aerosolized pentamidine. Chest 1992; 101:1404-09

To the Editor:

We thank Dr. McIvor and his coworkers for their interest in our study and for sharing their data.

Analyzing random HCW urine specimens (assayed at our laboratory) from a large treatment center (up to 1,200 treatments per HCW per month), they found only one positive urine test for pentamidine in an HCW. There are three possible explanations for the differences in the two studies:

1. The Toronto group administered all treatments with the Fisoneb system, an ultrasonic nebulizer that only produces aerosol when a patient holds down a switch on the device. In our article we suggest that the most likely explanation for high-level intermittent exposure of HCWs to pentamidine is "disconnected nebulization" (ie, when a patient removes the device from his mouth in order to cough, he may neglect to switch it off). Because of the design of the Fisoneb, such disconnected nebulization may be less likely to occur.

2. Another important difference between the studies is the design of the treatment rooms. We used a treatment room with an exhaust system that operated at 60 cu ft/min. In contrast, McIvor et al. had a specially designed treatment room that exhausted air at 450 cu ft min during treatments. The frequency of air exchanges may be important. In our initial report of positive results in urine specimens from in HCWs, levels were higher at Stony Brook, where fewer treatments were administered but where the frequency of air exchanges was aloz when compared with a Miami center, which had a more effective air exchange system but at which workers performed a much larger number of treatments.

3. In our study, HCWs supervised the treatments. In the Toronto study, the HCW was not in the room during treatments, which were self-administered by the patients. In general, because the possibility of disconnected nebulization is greatest during the absence of HCWs from the treatment room, it is usually recommended that HCWs not enter a room until two air exchanges have taken place following completion of an unsupervised treatment. This would take 30 min with our system. In Toronto, the more efficient exhaust system would markedly reduce the waiting time for two air exchanges. Thus, patient-administered treatments are likely to be much less hazardous to HCWs in Toronto than they would be under our system.

While the Toronto group should be congratulated on the effectiveness of their system in avoiding high exposures of HCWs to pentamidine, the conditions under which our HCWs administer pentamidine are, we believe, more typical of clinical practice than the conditions under which the Toronto group practices. Nevertheless, the simple precautions we advocated in our article can reduce the likelihood of high exposures with our system. We agree that chronic low-level exposure to pentamidine is inevitable and that the long-term consequences, if any, of this exposure are unclear at the present time.

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REFERENCES


Adenoid Cystic Carcinoma Mimicking a Dermoid or Hydatid Cyst

To the Editor:

A 75-year-old nonsmoking male patient was admitted with the complaints of backache and clear, watery discharge on two occasions during coughing. Chest radiography revealed a mass lesion occupying almost all of the left hemithorax. A multiloculated cyst with solid components and calcified foci was observed on computed tomography of the thorax (Fig 1). Surgery was performed on the basis of the preliminary diagnosis of hydatid or dermoid cyst. Adenoid cystic carcinoma was found in the pathologic examination.

Many cases of adenoid cystic carcinoma arising from the bronchial tree have been reported.1-3 Few reports have described calcification.