correct when they note that while group allocation was random-ized, no attempt was made to “match” the groups. We presume this means ‘matching’ for perceived risk factors. However, it is important to note that there were no significant differences between groups in the preoperative data, which were specifically documented to analyze the perceived risk factors, eg, history and symptoms of pulmonary disease, smoking history, spirometry, and arterial blood gas analyses (Chest 1994; 105:741,742). Hence, although we did not deliberately “match” the groups for such factors, the process of randomization was successful in achieving groups who had similar perceived levels of risk.

We are not certain what Smith and Fowler mean in the sentence concerning the “ . . . lack of detail about the different levels of independent variables . . . .” We chose to consider oral temperature, chest x-ray film scores, and arterial blood gas analyses as separate sets of repeated measures data. Within the repeated measures analysis of variance, time by group interactions were considered and found to be nonsignificant. As there was no indication that any significant differences existed between groups for any of these variables at any time, we could see no reason to consider combinations of these variables. Although various combinations of such data have been used in the past to define pulmonary complications, the clinical importance of such complications has been poorly addressed (Chest 1994; 105:74). Instead, while reporting the results of these data, we chose to consider only those patients identified by the cardiologist as having clinically significant postoperative pulmonary complications. The statistical tests used in the analyses are described in the Statistical Analysis section (Chest 1994; 105:742).

As far as the reliability of the measured variables is concerned, it should be clarified that spirometry was not considered as a response variable. Spirometry was only performed preoperatively to detect abnormalities of pulmonary function, and, thus, to assist in determining if the groups were evenly “matched”. The intra-examiner reliability of the radiologist who performed the chest x-ray film measurements was established since this required a subjective judgement by the radiologist. As only one senior radiologist was involved in reading the chest x-ray films, we did not consider it necessary to measure inter-examiner reliability. We also did not consider it necessary to establish the reliability of the measurements of oral temperature and arterial blood gases. The radiologist chose not to define the grades of minimal, moderate, and major abnormality as he considered that it was difficult to define precisely the extent of the abnormality as being, for example, subsegmental, segmental, or lobar using just a posteri-or or anteroposterior film but no lateral film. Smith and Fowler questioned the use of the “ . . . appropriateness of using oral temperatures and graded chest x-ray films as measures of change . . . .” We believe these are quite valid measurements but agree, as noted earlier, that abnormalities of these may not represent clinically significant pulmonary complications.

It is not clear what is meant by the “history threat” nor the “events” mentioned by Smith and Fowler. As the improvements in the parameters measured for the control group were similar to those for the two treatment groups, this suggests that the postoperative care over this time is one of natural improvement, whether or not prophylactic breathing and coughing exercises are given. Thus, the control group “controls” for natural change. As noted in the Materials and Methods section of the article (Chest 1994; 105:742), events such as the mobilization program followed the normal protocol whenever possible. No other events of note occurred to patients during the study period.

It is unclear what Smith and Fowler mean by the term “mortality rate.” As far as “real” mortality is concerned, one patient who had an inoperative cerebrovascular accident subsequently died as a result of the cerebrovascular accident. Considering experimental mortality, seven patients were withdrawn during the study because of the need for mechanical ventilation for more than 24 hr or neurologic complications. Neither the actual mortality rate or the experimental mortality could be attributed to the presence or absence of prophylactic chest physiotherapy.

The more definitive forms of chest physiotherapy cited by Smith and Fowler include unilateral basal expansion, the forced expiration technique, and the active cycle of breathing technique. We were surprised at the inclusion of unilateral basal expansion in the list of definitive techniques, considering the lack of evidence that these exercises are of any benefit.1 The techniques used in our treatment groups (Chest 1994; 105:741) included periods of deep breathing, quiet breathing, and huffs, all of which are components of the forced expiration and active cycle of breathing techniques. It is important to point out that since the overall incidence of clinically significant postoperative pulmonary complications for routine coronary artery surgery patients is so low, it is probably not cost effective to perform prophylactic chest physiotherapy for all patients, no matter what sort of therapy is used.

In conclusion, we believe the study was well designed, appropriately analyzed, and has important implications for the management of patients after routine coronary artery surgery.

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REFERENCE


Pulmonary Involvement in Patients With HFRS

To the Editor:

A respiratory illness with high mortality was recently reported in the southwestern United States. Serologic studies implicated the hantaviruses as the causative agent. Nucleotide sequence analysis revealed the associated virus to be a new Hantavirus.1,2 In the last 10 years, 32 patients with hemorrhagic fever with renal syndrome (HFRS) have been diagnosed in our center.3 The causative agent also belongs to hantaviruses and is closely related to the prototype Hantaan virus.4 Even though respiratory illness is not among the main clinical manifestations of the disease, a number of symptoms and findings in physical examinations as well as radiologic findings in chest x-ray films were evident.

In particular, 12 patients (37.5%) had dyspnea and 7 patients (21.8%) had a dry cough. Two of the 12 patients with dyspnea developed pulmonary edema, which was ascribed to fluid overload. One of these patients with oliguria and a serum creatinine of 1,273 μmol/L recovered fully after hemodialysis, while the other, a 71-year-old man, succumbed because of concomitant infection and heart failure. In the physical examination, diminished breath sounds and rhonchi or crackles were evident in most patients. The most common radiologic findings in chest x-ray films are shown in Table 1. Fifteen of the 25 patients had more
Table 1—Abnormal Findings in Chest X-Rays

<table>
<thead>
<tr>
<th>Findings</th>
<th>Patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Signs of poor inspiratory effort</td>
<td>28/32</td>
<td>87.5</td>
</tr>
<tr>
<td>Interstitial edema</td>
<td>14/32</td>
<td>43.75</td>
</tr>
<tr>
<td>Subsegmental atelectasis</td>
<td>12/32</td>
<td>37.5</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>2/32</td>
<td>6.25</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>2/32</td>
<td>6.25</td>
</tr>
<tr>
<td>Total abnormal findings</td>
<td>28/32</td>
<td>87.5</td>
</tr>
</tbody>
</table>

than one abnormal chest x-ray film finding either on the initial or subsequent films. We conclude that pulmonary manifestations are not uncommon in HFRS infection due to classic Hantaan virus.

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REFERENCES

What Causes CD4+ T-Lymphocytopenia?

To the Editor:

Turett and Telzak1 raise some interesting points in their recent article entitled “Normalization of CD4+ T-Lymphocyte Depletion in Patients Without HIV Infection Treated for Tuberculosis” appearing in the May issue of Chest; however, several issues need to be addressed. First, they report on three patients with CD4+ T-lymphocytopenia; yet, only two of these patients actually meet the criteria established by the Centers for Disease Control for CD4+ T-lymphocytopenia,2 as patient 1 failed to show a CD4+ T-lymphocyte count less than 300 cells/mm3 on even one occasion, and thus the patient cannot be considered to have CD4+ T-lymphocytopenia.

Another point that needs to be addressed is the authors’ comparison of their findings with previous reports of CD4+ T-lymphocyte depletion in other disease states. They report that CD4+ T-lymphocyte depletion was reported in the absence of HIV infection in patients with both cytomegalovirus infection3 and cryptococcal infection.4 However, the report of CD4+ T-lymphocyte depletion in cytomegalovirus disease made no mention if the patients studied were, in fact, HIV infected.4 How can Drs. Turett and Telzak state unequivocally that these patients were not HIV infected, when at the time of the study, in 1980, the HIV antibody test was not yet developed? With regard to the report4 of CD4+ T-lymphocyte depletion in a patient with cryptococcal neoformans infection, this patient had low CD4+ T-lymphocyte counts, with an inverted CD4+CD8 ratio. This was not present in the patients reported by Drs. Turett and Telzak and probably represented a selective depletion in CD4+ T-lymphocytes, which is probably an entirely different process than the generalized decrease in T-lymphocytes that the present authors observed in their patients.

What appears more likely is that the patients reported in the present article had a generalized reversible, depression in T-lymphocytes, a finding which has already been shown to result from tuberculosis5,6 as well as from other infectious processes, such as lepromatous leprosy7 and histoplasmosis.8 In all these reports, there was a normalization of all lymphocyte populations with therapy.

In addition, their statement that tuberculosis is a reversible cause of unexplained CD4+ T-lymphocytopenia is misleading to the extent that the generally accepted criteria for the diagnosis of this condition excludes processes which are known to cause transient lymphocyte depletion.2 Following their reasoning, if tuberculosis is a possible cause of unexplained CD4+ T-lymphocytopenia, so is histoplasmosis, lepromatous leprosy, and several other infectious processes.

I believe that Drs. Turett and Telzak do raise some interesting points in this article; however, I believe that it is misleading to classify tuberculosis as a possible cause of unexplained CD4+ T-lymphocyte depletion considering what we know concerning this disease process. The authors have simply described a reversible, generalized decrease in T-lymphocytes, which has been shown in the past, to occur with tuberculosis, as well as several other disease processes.

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REFERENCES
2 Centers for Disease Control. CD4+ T-lymphocytopenia in patients without evident HIV infection: United States. MMWR 1992; 41:578-79
6 Singhal M, Banavaliak JR. Peripheral blood T-lymphocyte subpopulations in patients with tuberculosis and the effects of chemotherapy. Tubercle 1989; 70:171-78

To the Editor:

We thank Dr. Gettler for his letter regarding our article “Normalization of CD4+ T-lymphocyte Depletion in Patients Without HIV Infection Treated for Tuberculosis” (Chest 1994; 105:1355-37), and we would like to respond in detail to his comments, some of which are inaccurate.