COMMUNICATIONS

TO THE EDITOR

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Pneumopericardium following Laparoscopy

To the Editor:

The report by Nicholson and Berman1 entitled “Pneumopericardium following Laparoscopy” and claiming to report this association for the first time, seems to me to contain a serious error due to an incorrect interpretation of the chest x-ray film. The evidence is grossly insufficient to support their claim that pneumopericardium was present. The authors state that to their “knowledge pneumopericardium has not been documented previously” following laparoscopy and they too have failed to document this condition. Their statement that “x-ray films of the chest—documented the presence of pneumopericardium” is definitely open to question. The patient certainly had pneumoperitoneum, pneumomediastinum and subcutaneous emphysema, but it is very doubtful that pneumopericardium was present.

The thin lines paralleling the cardiac silhouette and mediastinum, indicated by arrows in Figure 1 of the article, are interpreted by the authors as indicative of air in the pericardial sac, but these lines are highly characteristic of pneumomediastinum and are due to lateral displacement of the mediastinal pleura by dissection of air in the mediastinum.2-3 The paramediastinal line on the right appears to extend higher than the usual superior anatomic limits of the pericardium and curves laterally rather than medially as the pericardium might be expected to do if it reached this point,4 lending further support to my contention that the air is in the mediastinum and not in the pericardium. A lateral and a lateral decubitus x-ray film of the chest would have been helpful in differentiating the two conditions in question,3-5 but apparently these films were not taken.

On the basis of the evidence presented, one is forced to conclude that the first documented case of pneumopericardium complicating laparoscopy has yet to be reported.

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REFERENCES
1 Nicholson RD, Berman ND. Pneumopericardium following laparoscopy. Chest 1979; 75:805
2 Teplick JD, Haskin ME. Roentgenologic diagnosis (Vol. 1, 2nd ed.). Philadelphia: Saunders, 443-444
3 Fraser RC, Pare JA. Diagnosis of diseases of the chest, Vol. III. Philadelphia: Saunders 1979:1812

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To the Editor:

The acerbic pronouncements of Dr. Buechner do serve to remind us of the difficulty that may be experienced in trying to distinguish mediastinal from pericardial air, a problem well recognized before.1 This stems, in part, from the fact that except in the region of the hila and directly behind the sternum, the mediastinal pleura follow the contours of the pericardial sac2-4 (and his reference 4, 1140-45). Unfortunately, Dr. Buechner’s references do not help to clarify the problem. I could not check his reference to the second edition of Teplick and Haskin, but the third edition (1976), the earliest to which I had access, did not resolve this.

That the patient had mediastinal air was never in question and was clearly stated in our report. In addition, we believe this patient had air within the pericardial sac. Unfortunately, the location of the upper arrow in our illustration has a misleading appearance in the published reproduction that was not apparent in the original photographs. The arrows were meant to point to the air, as indicated in the figure legend, not to the pericardium. The upper right arrow appears to be pointing to a heavy line that does indeed curve laterally and is likely the right mediastinal pleural reflection. If Dr. Buechner were to follow the fine line of the right pericardium, located lateral to the air space indicated by the second arrow on the right, he would see that it curves medially just above the topmost arrow. Similarly the line representing the left pericardium curves in to meet the cardiac silhouette below the level of the aortic knob and at the same level as the line on the right. Both these levels are consistent with the anatomic descriptions of Gray (his reference 4) and others.6-8 These two lines join to form the upper limits of the pericardial sac referred to as the recessus aorticus pericardii.7

Contrary to Dr. Buechner’s supposition, lateral films were taken. They were omitted from the report because the editorial policy of the journal restricts case reports to two figures. We felt the two figures we selected most appropriately illustrated the features of this case. The lateral film showed a crescent of air between the right ventricle and the sternum coursing upwards and posteriorly along the curvature of the right ventricular outflow tract. This duplicated the location of the pericardial sac as seen in midline sagittal sections.8

Thus, in the presence of recognized mediastinal emphysema, most of which had already tracked up to the neck, this patient had a large localized collection of air clearly delineating the anatomic position of the pericardial sac. The resulting picture is similar to that produced by diagnostic pneumopericardium and to illustrations of pneumopericardium that have been published by others (see original references). It seems to us reasonable to conclude that this air was, in fact, located within the pericardial sac.

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REFERENCES
4 Netter FH. Ciba collection of medical illustrations, Vol 5:
Tuberculosis in Oriental Immigrants

To the Editor:

We read with interest the article by Byrd et al.1 concerning tuberculosis in Oriental immigrants. Although the authors correctly point out that immigrants from Asia do have higher rates of drug resistance than the indigenous US population, there are several statements in that article which we feel deserve comment and clarification.

In the ongoing US Public Health Service primary drug resistance (PDR) study,2 21 percent of cases considered of Asian origin were found to be excreting organisms resistant to one or more of the ten drugs tested. However, not all these persons were immigrants, as implied by Byrd et al; an unknown proportion of our study participants of Asian heritage were American born. Therefore, our study data cannot be used to assess resistance rates among immigrants.

As stated in our article, the importation of drug-resistant strains from other parts of the world is a likely explanation for the high PDR rates observed among persons of Hispanic or Asian backgrounds. However, in areas where large numbers of persons of Asian and Hispanic descent have settled, PDR rates are also high among white and black subjects. Although we can hypothesize that most drug resistance in these areas has been imported from other countries, our data do not prove that this is the case.

Byrd and associates also imply that the state of Hawaii has partially solved the problem of imported drug resistance with a policy toward the admission of patients with tuberculosis which differs from that of the other 49 states. This is incorrect. Persons with "active" disease are not allowed to enter any state in the United States without special waiver. A more plausible reason for the lower resistance rate observed by Pien et al3 in Hawaii can probably be attributed to differences in criteria for drug resistance. Pien et al used the following drug concentrations (in µg/ml) to determine resistance: streptomycin 10, isoniazid 1, ethambutol 8, and rifampin 5. By comparison the drug concentrations used by Byrd et al were much lower—streptomycin 2.5, isoniazid 0.1, ethambutol 5, and rifampin 1. By using relatively high drug concentrations, Pien et al screened out strains with a low degree of drug resistance. On the other hand, Byrd et al, by using low concentrations, increased the likelihood of finding resistant strains.

As Byrd and colleagues point out, their inability to obtain drug susceptibility test results for 40 patients who had become culture negative by the time they entered the hospital, tends to inflate the resistance rate among their patients. It is difficult to believe that "all dependents found to have tuberculosis while living overseas in their home country are referred" to Scott Air Force Base for evaluation. Since many patients with tuberculosis are managed as outpatients, we would wonder if there might not have been other patients with susceptible organisms who responded to treatment and were not sent to Scott Air Force Base.

Although drug resistance is a problem among persons of Asian descent, the magnitude of the problem may not be as great as that implied by Byrd and coworkers. Preliminary data on drug resistance among Indochinese refugees in San Francisco indicate that drug resistance rates are between 15 and 20 percent (Austin Brewin, M.D., City and County of San Francisco Department of Public Health, personal communication). These data are similar to those observed in the earlier US Public Health Service study and other data published by Canadian workers.4

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REFERENCES

To the Editor:

I appreciate the comments of Dra. Snider and Farer regarding our publication on drug resistance in tuberculous Oriental immigrants. We had, in fact, incorrectly assumed that all the patients classified as "Asians" in the United States Public Health Service (USPHS) study1 had emigrated from the Orient. In light of this fact, one may speculate the incidence of drug resistant disease may have been even higher than 20 percent in the immigrant subgroup had their numbers been diluted by nonimmigrant Oriental subjects with sensitive organisms.

Regarding our study, it was specifically noted in the subtitle of our paper, as well as in the discussion, that our military-dependent patients clearly constituted a subgroup of Oriental immigrants, and that their incidence of drug-resistant disease might not be representative of all Asian immigrants. The unpublished data on the Vietnamese "boat people" cited by Drs. Snider and Farer is also based on a subgroup principally of Chinese extraction and generally of a different economic class than the majority of ethnic Vietnamese. Therefore, these data likewise should not be offered as representative of all Oriental immigrants.

It is indeed possible that some tuberculous Oriental Air Force dependents have entered the United States without being seen at our hospital despite very clear Air Force immigration regulations in effect at the time of this study. To our knowledge, however, no additional Oriental patients have appeared on the annual tuberculosis control report forms required of every Air Force medical facility in the United States.

In our paper, we noted the fact that patients arriving at our hospital already culture-negative might indeed inflate resistance rates. However, as we pointed out, even if this subgroup were considered to have sensitive organisms, the incidence of resistance to one drug would still be 41 percent and to multiple drugs 23 percent.

The variation in criteria used to define resistance does