Table 1—Pulmonary Function Data

<table>
<thead>
<tr>
<th></th>
<th>October</th>
<th>November</th>
<th>October</th>
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</thead>
<tbody>
<tr>
<td></td>
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<tr>
<td>Vital capacity (L)</td>
<td>1.36 (37)*</td>
<td>2.26 (80)</td>
<td>3.57 (96)</td>
</tr>
<tr>
<td>Total lung capacity (L)</td>
<td>3.3 (62)</td>
<td>4.07 (76)</td>
<td>5.18 (97)</td>
</tr>
<tr>
<td>DlCO (ml/min/mm Hg)</td>
<td>9.81 (49)</td>
<td>17.53 (67)</td>
<td>19.78 (69)</td>
</tr>
<tr>
<td>Exercise tolerance (kpm)</td>
<td>200</td>
<td>300</td>
<td>1000</td>
</tr>
</tbody>
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*Nos. in parentheses represent percent predicted

Failure of Disposable Domes

To the Editor:

In the article, "Failure of disposable domes to prevent septicaemia acquired from contaminated pressure transducers" (Chest 74: November, 1978), Buxton et al contradict the title and opening statements. In all the tests that were conducted, the results proved conclusively the integrity of the membrane. The problem area is identified in the eight-day surveillance study of the 27 patients. The only patients that were contaminated were from the surgery department thereby eliminating ICU where pressure monitoring is performed with the same disposable domes. Of the four patients that were contaminated, three had contamination at the radial artery stopcock, but not at the dome and the fourth had contamination at the dome but not at the radial artery stopcock. If the E cloaca had penetrated the membrane, the complete system would have been contaminated. As the radial artery stopcock was being used as a sampling port, the contamination was probably caused by direct contact.

The conclusion that the domes failed to prevent contamination is invalid since all of their findings substantiate the dome integrity.

Alan Smart, Bentley Laboratories, Irvine, California

To the Editor:

Mr. Smart's letter raises two important issues. First, do disposable domes prevent septicaemia attributable to contamination of arterial pressure monitoring systems? Second, if they do not, what is the mechanism of monitoring-circuit contamination?

Our article, as well as another report indicate that these devices, which we grant are useful, cannot be relied upon to prevent monitoring-circuit contamination or associated septicaemia. In the outbreak we reported, Enterobacter septicaemia was epidemiologically and microbiologically traced to contaminated patient-monitoring circuits. All of the transducer sensing plates we tested were contaminated with the infecting strain.

Our study did not prove, however, how contamination entered the patient monitoring circuit. There were numerous potential mechanisms for contamination. We could not document a defect in the membrane of the domes in the tests that we conducted, but no domes used on affected patients were available for evaluation; the numbers of domes examined were too small for us to be certain, with high statistical reliability, that the domes were free of defects; and we were unable to duplicate the actual conditions of their use. We agree with Mr. Smart that direct contact with a contaminated object may have played a role. The data suggest, however, that direct contact contamination of the disposable dome was more likely than contamination of the arterial line site. Systematic cultures of transducer sensing plates, fluid in the dome, and arterial lines were obtained from only one system; malignant disease. A syndrome sometimes associated with cavitating tumors. Clin Radiol 27: 211-222, 1976


6 Libshitz HI, Banner MP: Spontaneous pneumothorax as a complication of radiation therapy to the thorax. Radiology 112: 119-201, 1974


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CHEST, 77: 3, MARCH, 1980

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the results showed contamination of transducer and dome fluid, but not of fluid from the arterial line. The three patients whose radial artery catheters yielded positive cultures did not have their disposable domes sampled (at the time these samples were obtained, the potential link to the domes was still unrecognized, and the dome with a positive culture was the only one cultured). We presume, on the basis of these few observations, that contamination was introduced from transducer to dome and that the contamination then traveled from dome to arterial line site. Because of the serious illnesses that resulted from that contamination, we were obliged to initiate immediate control measures as soon as these observations were made, and we therefore had no opportunity to extend the observations to a larger number of patients.

With regard to his other comments, Mr. Smart is misinformed. At the time of the outbreak, the disposable dome transducers were used only in the operating rooms, not in the intensive care unit. It is unlikely that contamination of the radial artery stopcock was induced by the sampling technique, as no positive cultures were obtained from any other sampling site. Samples of the other sites were performed in the same manner and at the same time as the radial artery samples. We believe that disposable domes are useful adjuncts in infection control of monitored patients. We strongly emphasize, however, the obvious fact that their mere use does not magically remove the threat of septicemia in monitored patients. We urge that hospital personnel routinely disinfect transducers, take care to assemble monitoring circuits aseptically, and change domes and circuits at regular intervals. Disposable domes should not be resterilized or reused. Should septicemia occur in a monitored patient, the monitoring circuit should be suspected as the source of infection, discontinued if possible, or changed completely if monitoring must be continued. Finally, we urge transducer manufacturers to provide users with information regarding acceptable methods to decontaminate transducers.

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1 Retaillian HR: Infection control with invasive pressure monitoring devices. APIC Journal 7:13-17, 1979
2 Center for Disease Control: Sterilization and Disinfection of Hospital Supplies. Morbidity and Mortality Weekly Report, August, 1977, p 266

Hypertrophic Cardiomyopathy

To the Editor:

In the clinical conference on hypertrophic cardiomyopathy Chest (74:659-670, 1978), Dr. Goldstein states that: "Mid-systolic closure of the aortic valve can occur in both hypertrophic cardiomyopathy and discrete subaortic stenosis and, hence, is not particularly useful in differentiating these two diseases" (p 661).

This statement is inaccurate. In the obstructive form of hypertrophic cardiomyopathy, the aortic valve reacts with a mid-systolic partial closure to the diminishing flow through it.1 In discrete subaortic stenosis, the aortic valve reacts to the jet passing through the opening in the membrane, not to the amount of stenosis. Thus, the typical echocardiographic picture of the aortic valve in discrete subaortic stenosis (Fig 1) is an abrupt partial closure which occurs earlier in systole than the one found in obstructive hypertrophic cardiomyopathy2 and is even seen in mild cases. This is followed by a coarse flutter of both aortic valve leaflets, which lasts to the end of the systole. This kind of flutter is not seen in hypertrophic cardiomyopathy. Furthermore, it is most probably the cause of the development of aortic insufficiency in these patients, irrespective of the amount of obstruction.3

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REFERENCES

To the Editor:

The statement in question from our article in Chest was meant to indicate that the presence of mid-systolic closure of the aortic valve does not, in itself, discriminate between