agree that attempts to minimize the variability of FIO₂ are helpful, especially if the hospital air-oxygen system pressures vary. The gas exchange monitor evaluates the variability of FIO₂ and gross variability (1% or more) is automatically detected and the measurement result rejected. In addition, we use routinely a pressure regulator between the compressed air wall source and the blender (Wilkerson) to reduce the pressure constantly to the lowest level of the pressure variability in the hospital system. Using this approach, the coefficient of variation of FIO₂ is negligible: a random sample from 30 measurements in mechanically ventilated ARDS patients with an FIO₂ ranging from 38.7 to 67.6% had a mean coefficient of variation for FIO₂ of 0.2 ± 0.1% (SD).

Since the physiologic changes in VO₂ are the major source of variability, it is not surprising that the variability of the measured VO₂ is indeed very small, when the physiologic variability is controlled. In the same sample of ARDS discussed above, the overall variability of VO₂ was 3.3 ± 1.2%, which includes both the physiologic variability and the variability due to FIO₂. Similar results have been obtained by others.

We conclude that VO₂ can be measured reliably in a 30-min measurement, even if random variability of FIO₂ is present and if the physiologic changes in VO₂ are minimized. The variability of VO₂ should be evaluated, and in stable conditions it is less than 5%. As a practical rule, the magnitude of a true difference in VO₂ that can be detected with a 95 to 97.5% power using 30 min measurements is equal to the standard deviation of the VO₂. Under these conditions, the variability in FIO₂ is relatively unimportant. If shorter measurement periods are desired, the variation in FIO₂ becomes more important, and the power in detecting small true changes decreases. If measures to reduce the variation in FIO₂ to about 0.2% are unsuccessful, the formula proposed by Dickerson et al may be used to evaluate the magnitude of the potential error.

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REFERENCES
1. Norman GR, Steiner DL. Biostatistics—the bare essentials. St Louis, Mo: Mosby-Year Book 1994; 24

Respiratory Syncytial Virus (RSV) or Rous Sarcoma Virus (RSV)?

To the Editor:

In the article entitled “Recovery of Viruses Other Than Cytomegalovirus From Bronchoalveolar Lavage Fluid” by Connolly and colleagues (CHEST 1994; 105:1775-81) in the June 1994 issue of CHEST, the abstract of the article mentions the finding of one case of respiratory syncytial virus among viral cultures from 1,199 BAL specimens. In the text, however, it is stated, “... and Rous sarcoma virus were each cultured once...” and

... the patient with Rous sarcoma virus also showed...”pp1778-79 There is no allusion to respiratory syncytial virus anywhere in the text.

The finding of respiratory syncytial virus in a patient with AIDS is interesting and has only rarely been reported. However, the finding of Rous sarcoma virus, an oncogenic retrovirus causing sarcomas in chicken, is even more interesting. I am looking forward to learning which virus was recovered from the patient.

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When Should We Perform Bone Marrow Biopsy in Patients With Miliary Tuberculosis?

To the Editor:

We agree with Dr. Lindhardt’s careful review of our manuscript (CHEST 1994; 105:1775-81). We meant that the virus we were recovering from the lower respiratory tract was “respiratory syncytial virus.” We agree that the Rous sarcoma virus would be highly unusual to be obtained from the lung, and it was not what was recovered in these patients.

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Dr. Kinoshita and colleagues recently published an interesting article in the Journal (CHEST 1994; 106:990-96). These authors emphasized the value of bone marrow biopsy to confirm tuberculosis in miliary pulmonary lesions.

Bone marrow biopsy, as liver biopsy, allow in some emergency cases a rapid diagnosis of miliary tuberculosis. Bone marrow biopsy had the advantage that it could be performed even with thrombopenia or coagulation disorders. However, published data are questionable.

Gelb et al¹ studied 109 patients with miliary tuberculosis; bone marrow biopsy allowed the diagnosis of 2 of 2 cases (100%). Nevertheless, bone marrow was involved in only 5 of 21 cases (24% necropsies).

Kim et al² reported on 38 patients and performed bone marrow biopsy in 22 patients. Granuloma was found in 22% of the cases, caseating in 9%. Bone marrow biopsy lead to the diagnosis in only 14% of cases.

Maartens et al³ noted in 109 cases that 82% had granulomas, caseating in 27%. All these data are lower than those obtained with liver biopsy.

Lombard and Mansel⁴ studied hematological changes associated with miliary tuberculosis. All the patients had tuberculous infiltration of the bone marrow. All the patients with bone mar-