tuberculosis. The authors conclude that transbronchial biopsy cultures are not helpful, based on evaluation of only six patients. Meaningful conclusions cannot be drawn from such a limited sample.

In summary, I believe that the conclusions of Miro and coworkers are supported neither by published reports nor by the data they present. Additional studies are needed to address the contribution of transbronchial biopsy to the diagnosis of tuberculosis in patients with HIV infection.

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To the Editor:

We are grateful for Dr. Barnes's interest and comments on our article and appreciate the opportunity to respond to his letter.

The lung is a frequent target of AIDS-related opportunistic infections, including diseases caused by bacterial, viral, protozoal, mycobacterial, and fungal pathogens. Because of this diversity and the frequency of multiple, concurrent infections, fiberoptic bronchoscopy remains a cornerstone of the diagnostic strategy in these complex cases.

At our institution, when HIV-related pulmonary infection is suspected based on clinical history and examination, our practice is to perform a diagnostic bronchoscopy within 24 to 48 h of admission unless a diagnosis has been established by other means. This is justified in order to obtain a rapid and accurate diagnosis and to institute appropriate therapy. Thus, the procedure is often performed prior to availability of final results from other tests, such as the PPD skin test, serial sputum acid-fast bacilli (AFB) smear analysis, and HIV serology. We sought to determine which bronchoscopically obtained specimens were sufficient for detecting pulmonary tuberculosis. We did not intend to compare the diagnostic utility of sputum analysis alone with that of bronchoscopy for diagnosis of pulmonary tuberculosis.

We agree with Dr. Barnes that bronchoscopy is not indicated for diagnosing pulmonary tuberculosis if sputum AFB smear analysis is already confirmative. However, in dealing with a population including many HIV-positive patients, there is often a clinical suspicion of pulmonary infection other than tuberculosis. In fact, a concurrent diagnosis of Pneumocystis carinii pneumonia (PCP) was found in 19 percent of our high-risk patients. This is consistent with previous reports, including a study by Dr. Barnes and his colleagues.1 They found concurrent PCP in 4 of 15 AIDS patients (27 percent) with Mycobacterium tuberculosis infection. This high frequency of concurrent infections underscores the importance of a thorough diagnostic evaluation. In our study, although only 18 of the 27 patients in the high-risk AIDS group satisfied Centers for Disease Control clinical criteria (opportunistic infections or malignancies), in all patients bronchoscopy was indicated based on an elicited history of high-risk AIDS behavior and/or other clinically typical signs.

As Dr. Barnes points out, granulomatous histopathologic change detected in transbronchial biopsy (TBB) specimens provides a rapid presumptive diagnosis in the non-AIDS patient population. We similarly found a 63 percent incidence of granulomatous changes in biopsy specimens from our non-AIDS patients. However, among the high-risk group, this typical histopathologic pattern was generally absent; it was detected in only 2 of 22 (9 percent) biopsy samples analyzed.

Finally, we agree that our limited sample size of six TBB specimens submitted for culture limits conclusions regarding the microbiologic contribution of this sampling tool. However, preliminary data in a prospective study from our institution similarly showed that although TBB was culture-positive in 11 of 16 (69 percent) specimens submitted, it was never the sole source of microbiologic confirmation.2 Additional information evaluating the risk-benefit ratio and complication rate from this procedure in this specific patient population would be useful in guiding clinical management.

In summary, we greatly appreciate the comments made by Dr. Barnes, but contend that our conclusions regarding the limited additional value of obtaining TBB for the diagnosis of pulmonary tuberculosis in HIV-related pulmonary disorders is supported by our data. We acknowledge that our study was retrospective in nature and, therefore, represents only the initial step in evaluating the precise role of TBB in this patient population.

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Ovarian Hyperstimulation Presenting as Acute Hydrothorax in Early Intrauterine Pregnancy

To the Editor:

Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic complication of ovulation induction by clomiphene and exogenous gonadotropins.3 The severe form of OHSS is characterized by massive bilateral ovarian enlargement with multiple cyst formation, ascites, hydrothorax, hemoconcentration, azotemia, cardiac tamponade, and often thromboembolic phenomena. It is life-threatening in severe cases.4

A 28-year-old woman presented to the maternity unit on January 2, 1992, with a 4-day history of increasingly severe right upper quadrant and lower abdominal pain associated with nausea. The pain was dull in character and was associated with dyspnea on
minimal exertion, wheezing, a cough productive of clear white sputum, and a pressurelike sensation under the right rib cage and midsternal area. There was no history of fever, chills, night sweats, or hemoptysis. She denied recent travel, prior pneumonias, or exposure to tuberculosis. She had a history of chronic sinusitis with postnasal drip but was otherwise in good health. A workup for infertility 4 years previously revealed polycystic ovarian disease. Ovarian stimulation was being given in the form of clomiphene citrate (Clomid) and human menopausal gonadotropin (Pergonal). Other medications included amitriptyline, asetimizole, and amoxicillin/clavulanate potassium for recent urinary tract infection. She was a nonsmoker and denied any history of alcohol abuse.

The physical examination revealed a heavyset woman with hirsutism, in mild distress with dyspnea and abdominal pain. The oral temperature was 36.5°C; the respiratory rate was 22 breaths per minute; the blood pressure was 130/80 mm Hg; and the pulse was 90 beats per minute. Her weight was 288 lb. Jugular venous distention was absent, and the trachea was central. There was no lymphadenopathy. There was dullness and decreased fremitus and breath sounds at the right lung base with egophony at the superior margin of the area of dullness. There were no adventitious sounds heard. Cardiac examination showed slight enlargement on percussion, but findings were otherwise normal. The abdomen was obese with striae gravidarum and nonnarrow bowel sounds. There was no clubbing, cyanosis, pedal edema, or calf swelling or tenderness.

The chest radiograph showed cardiomegaly and a large right pleural effusion (Fig 1). Pelvic ultrasound revealed a gestational sac consistent with a 4-week intrauterine pregnancy, markedly enlarged ovaries, and fluid in the cul-de-sac. The left ovary and right ovary measured 125 mm × 70 mm and 162 mm × 93 mm, respectively, with multiple large ovarian follicles. Abdominal ultrasound showed multiple gallbladder calculi. The kidneys were of normal size. The CBC count and routine blood chemistry studies were normal. Arterial blood gas analysis was not performed. The chrorionic gonadotropin level was 1,212 mIU/ml. The diagnosis of grade IV severe OHSS was thus confirmed.

Upon admission the patient was put on complete bed rest with relief of pain by intramuscular meperidine hydrochloride and low-dose diuretic therapy. Frequent vital signs, daily body weights, intake and output, CBC count, and serum chemistry were closely monitored. With this conservative approach, she lose 2.3 kg, with resolution of her symptoms. A repeat chest radiograph showed a minimal residual right pleural effusion and minor atelectasis. After a 5-day hospital stay, she was discharged home in an asymptomatic state. Follow-up chest radiographs revealed complete resolution of pleural effusion.

This patient presented with features of severe OHSS secondary to ovulation induction. The two major components of OHSS are massive bilateral ovarian enlargement secondary to multiple cysts with stromal edema and acute fluid shifts due to the sequestration of fluid from the intravascular space to the third space.6 The clinical course is self-limited in nonpregnant women, but the duration may be longer with a more severe expression in pregnancy since the ovaries are restimulated by endogenous chorionic gonadotropin production.4 Thoracentesis is usually not necessary unless respiratory embarrassment is present. Knowledge of this syndrome is important to avoid unnecessary diagnostic maneuvers. Awareness of life-threatening complications, including thrombotic stroke, venous thrombosis, massive pericardial effusion, and adult respiratory distress syndrome, is necessary in dealing with OHSS.

![Figure 1](https://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21672/)

**Figure 1.** Chest radiograph demonstrating a large right pleural effusion and cardiomegaly.

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**Response of Pulmonary Nocardiosis to Ceftriaxone in a Patient With AIDS**

To the Editor:

The infrequency of Nocardia infection in cases of AIDS (not above 0.3 percent) is surprising, and it is exceptional for Nocardia infection to be the form of presentation of AIDS. We recently had the opportunity to treat a heterosexual man who had no history of intravenous drug abuse and who was a carrier of HIV antibodies. He presented with pulmonary infection by Nocardia, which responded excellently to ceftriaxone.

The patient was a 35-year-old man with a history of frequent contact with prostitutes. He reported having had a constitutional syndrome, fever, and hemoptysis 3 months before admission. Examination showed a temperature of 39°C, moderate malnutrition, multiple buccopharyngeal whitish plaques, and a crepitant stertor in the right pulmonary base. A chest radiograph taken on admission showed multiple-cavity infiltration in the right lung. Treatment with erythromycin, 4 g/d, was initiated. On the seventh day after admission, ceftriaxone, 2 g/d, was added to the treatment because of a general worsening of the clinical picture. Forty-eight hours after initiation of treatment with ceftriaxone, the patient had no fever, and his clinical condition had clearly improved.

Transtracheal needle aspiration of the right lung revealed the presence of necrotizing granulomas; acid-fast and Löwenstein smears were negative. Culture isolated *Nocardia asteroides*. Enzyme-linked immunosorbent assay and Western-blot technique showed HIV-positive antibodies. Study of lymphocytes in peripheral blood using flow cytometry showed the following values: CD4, 58 × 10^3/L; CD8, 219 × 10^3/L; CD4/CD8 ratio, 0.26.

The patient left the hospital 25 days after admission, and ceftriaxone was replaced by trimethoprim and sulfamethoxazole, since they can be administered orally. Three months after leaving the hospital the patient was asymptomatic, and thoracic radiography was normal.

Nocardia infection is usually introduced via the lungs. Consolidation with cavitation is the most frequent radiologic pattern.7 This