grounds, and are likely to go untreated until the results of blood cultures are available. This emphasizes the importance of performing blood cultures in patients with suspected sepsis.

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The Reply:

In Gray and Pedler's analysis, one third (6 of 18 cases) of anaerobic bacteremias occurred in patients who had no clinically obvious source of infection; 4 of these cases occurred in febrile, neutropenic patients. In our study, only 5 of the 40 cases were of uncertain origin, 2 being in febrile, neutropenic patients. The differences seem striking, but the numbers of cases are small, and the relative proportions of bacteremias from uncertain sources are not statistically different ($p = 0.08$; Fisher's exact test, two-tailed). Although this trend may reflect a real difference, the patient populations and medical practices in the two study locations may be different in ways that would affect the local epidemiology of anaerobic bacteremia.

Our colleagues' judgment that seven of their patients would not have received antianaerobic therapy without the benefit of the blood culture result led them to reaffirm the importance of culturing blood for obligate anaerobes. We found that the results of anaerobic blood cultures rarely influenced patient management, although the empiric use of extended-spectrum antibiotics (e.g., ticarcillin-clavulanate or imipenem/cilastatin) in some of our cases leaves us uncertain as to whether the inclusion of antianaerobic activity was by design or by default. In part because of the use of these antibiotics, there were no cases in our study in which the isolation of an anaerobe was both unexpected and influential in management. Nevertheless, while there may be specific clinical circumstances in which dedicated anaerobic cultures can reasonably be omitted from the blood culture routine, we agree that the clinical situations emphasized by Gray and Pedler, i.e., fever with neutropenia and fever without an obvious source of infection, are circumstances in which anaerobic blood culture techniques are clearly indicated.

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ACUTE MYELOGENOUS LEUKEMIA

To the Editor:

I enjoyed reading the recent review of acute myelogenous leukemia (AML) by Mastrianni *et al* [1]. However, I disagree with some of their statements, as follows:

(1) Although it is difficult to estimate the incidence of AML, the 10,000 cases per year figure is not supported by their reference. The American Cancer Society estimates that 11,300 cases of granulocytic leukemia will be diagnosed in the United States in 1992 [2]; however, only about 77% of these cases will be AML (the rest is accounted by chronic myelogenous leukemia) [3]. The estimated incidence of AML is thus closer to 8,700 cases per year.

(2) Allogeneic bone marrow transplantation is somewhat riskier but still potentially lifesaving and feasible in patients over 45 [4–6]. Many centers (including ours) perform transplants in such patients. The blanket statement "the procedure is used only for patients under 45 years old" is inaccurate, and adherence to such a rule may prevent curative therapy in many patients.

(3) It is misleading to label p53 as an antioncogene (i.e., a gene that prevents malignant transformation when present) in AML. The study by Smith *et al* [7] quoted in the article actually showed a positive correlation between p53 protein expression and secondary plating efficiency, an indicator of poor prognosis in AML [8]. We have recently reported that inhibition of p53 RNA with an antisense oligonucleotide suppresses *in vitro* growth of fresh AML cells [9]. It would be more accurate to say that p53 is involved in leukemogenesis, and that reversal of this action may be clinically useful in the future.

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