more hematopoietic reserve and less severe disease. Although overall survival and leukemic transformation rates were equivalent between E1996 randomization groups, crossover of the supportive care arm to active treatment after 4 months limits our ability to draw conclusions about longer-term safety of ESAs in MDS, and the study was also powered so that only quite dramatic survival differences could have been detected (ie, 80% power to detect a 46% difference in hazard rate).

Several recent retrospective studies suggested that ESA treatment may be associated with a survival benefit in MDS, but these encouraging retrospective analyses are potentially confounded by patient selection bias.7,8 It is clear that larger prospective studies with primary safety endpoints, including both overall survival and progression-free survival, are needed. However, as for Hotspur’s metaphorical dangerous nettle in Henry IV, Part I, actually conducting such studies to pluck the flower of safety is a prickly business, because of logistical hurdles, patient preferences, and entrenched practice patterns. An ambitious industry-sponsored controlled study of epoetin alfa in MDS, EPO-ANE-3018, began enrolling patients a few months ago (the accrual goal is 450 patients), but results will not be available for at least a few years. Therefore, E1996 represents an important contribution—not because it definitely answers all important ESA safety questions, but because it suggests that ESA exposure is relatively safe in MDS at least in the very short term—and because for several years to come, the E1996 results will remain virtually the only controlled data available in this area.

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REFERENCES
The purpose of risk stratification is to assign treatment according to risk of relapse or death. Probably children with \( t(1;11)(q21;p23) \) rearrangement need less treatment while those with the other rearrangements need better treatment. Unlike \( MLL \)-rearranged leukemia in adults,\(^7\) allogeneic stem cell transplantation is no better than chemotherapy in children.\(^7\) Better therapy is likely to involve agents that impair targets normally controlled by \( MLL \) such as clustered \( HOX \) homeobox genes or novel enzymatic activities of fusion proteins.\(^7,8\)

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T(11q23): brand necessary

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