This established classification of the acute erythroleukaemias is based partly on the old FAB criteria and also upon morphological, cytochemical, and immunophenotypical criteria. All bone marrow aspirates demonstrate ≥ 50% erythrocytic precursors, with erythroid dysplasia. Dysplasia of the granulocytic and megakaryocytic cell lines may or may not be present. The M6a subtype is defined as ≥ 30% blasts of the non-erythrocytic component (FAB exclusion criteria); the M6b subtype is defined as ≥ 30% pronormoblasts of the erythrocytic elements; and the M6c subtype has ≥ 30% blasts and ≥ 30% pronormoblasts of the aforementioned exclusion criteria. Because the dysplastic changes may, at times, make definitive characterisation of the blasts as erythrocytic versus non-erythrocytic difficult, the morphological features must always be confirmed by cytochemical stains, immunohistochemical stains, and/or flow cytometric analysis.

These three separate subtypes must be distinguished from one another to provide useful prognostic information for the clinician and the patient. When treated with the standard myeloid protocol, the M6a and M6c subtypes demonstrate a very high remission rate, whereas most patients with the M6b subtype remain refractory to treatment. Notably, patients with the M6b subtype remain in remission for a significantly shorter time than the M6a group. Mean survival for these subtypes is: M6a, 31.4 (SD, 32) months; M6b, 3.13 (SD, 4.2) months; M6c, 10.5 (SD, 12.7) months.

The malignant clonal cell of origin manifesting as acute erythroleukaemia of any subtype appears to be a multipotential stem cell, which shows varying degrees of erythrocytic and granulocytic lineage maturation. Therefore, the three distinct subtypes of acute erythroleukaemia are not three separate diseases, but rather represent a spectrum of the same disease. The poor remission rate and short survival characteristic of this disorder are dependent upon: (1) a high pronormoblast to myeloblast ratio within diagnostic bone marrow aspirates, (2) a high proliferative index, (3) “unfavourable” cytogenetic aberrations, and (4) a high incidence of P-glycoprotein expression (the multidrug resistance phenotype).

**References**


**CORRECTIONS**


Detection of the CD56+/CD45− immunophenotype by flow cytometry in neuroendocrine malignancies. Bryson GJ, Lear D, Williamson R, et al. J Clin Pathol 2002; 55: 335–7. The quotation on page 335 was inadvertently cut out of the first paragraph (it should have remained there as the second sentence) and should begin with CD56 not CD99.