The Poster Session of June 20th, chaired by Dennis Quaglino and Franz Schmalzl, was concerned with a number of clinical and cytopathological aspects of myelodysplasia (MDS).

*S. Nardelli et al.* reported on the occurrence of familial MDS, associated with congenital malabsorption, in three sisters. Treatment with iron, folic acid, vitamín B\textsuperscript{12}, and digestive enzymes produces transient improvement, but bone marrow abnormalities persisted unmodified.

*F. Bailly et al.* concerned themselves with problems of differential diagnosis in young patients presenting with mild pancytopenia and increased bone marrow cellularity. The authors stated that bone marrow biopsy is always necessary to rule out aplastic anemia, but that it is not always adequate. Biochemical studies and CFU-GM cultures are not discriminative while the presence of nonrandom cytogenetic abnormalities associated with hypoplastic bone marrow favors a diagnosis of preleukemia rather than one of aplastic anemia.

*A. Tichelli et al.* studied 146 patients with severe aplastic anemia. They discussed the advantages of allogeneic BMT (34 patients) and pointed out the complications associated with treatment with antilymphocytic globulin (111 patients). With the latter, MDS and PNH are increasingly observed. However, despite these drawbacks ALG remains the only alternative if BMT is not feasible.

*A. M. Manel et al.* describe the clinical and hematological features in acquired myelodysplasia in childhood. In bone marrow smears dyserythropoiesis was often marked, but Prussian blue staining never showed more than 15\% positivity. Dysgranulopoiesis was less marked than usually observed in adults. Excess of blasts was seen in five cases. Cytogenetic abnormalities were found in seven out of ten cases, including monosomy 7 in two cases, trisomy 21 in two cases, and 5q- in one case. CFU-GM cultures showed decreased number of colonies (3/6) or the presence of microclusters (3/6).

*P. M. Carli et al.* reported the incidence of MDS from a population registry, limited to hematological malignancies, in the Departement of Cote d’Or, France. Over 7 years (1980–1986) 92 cases of MDS were recorded among 997 cases of hematological malignancies (9\%). There were 41\% RAEB, 24\% CMML, 22\% AISA, 7\% RAEB-T, 2\% RA, and 4\% acute MDS with myelofibrosis. Incidence rate increased with advancing age. Rates were similar until age 65 in men and women, but above that age rates were higher in
men than in women. The distribution of cases was similar in urban versus rural areas.

D. T. Bowen and A. Jacobs observed sideroblastic erythropoiesis (45%–80% ring sideroblasts) in four nonanemic patients. Peripheral blood cultures showed generally poor growth of BFU-E. Ferrokinetic studies showed in one patient 68% ineffective erythropoiesis. Two patients had a normal karyotype, and one had a constitutional 16p+ abnormality. The authors suggest these cases may represent early forms of myelodysplasia.

T. Miyazaki et al. reported erythrocyte ferritin investigations in patients with refractory anemia in MDS. The main findings obtained were the following: (a) red cell lysates in RA contained larger amounts of ferritin than normal subjects; (b) the levels of acidic isoferitin were higher than those of basic isoferitin in refractory anemia; and (c) red cells from RA patients contained normal or slightly higher values of iron compared to normal subjects.

D. M. L. Jupe et al. reported the association of HbH disease with MDS. The authors identified seven such patients over the past 8 years by employing Brilliant cresyl blue staining and electrophoresis at pH 8.6. These patients appear to have a poor tolerance to moderate degrees of anemia.

P. Felman et al., among atypical cases of CML, identified six cases presenting a distinctive nuclear anomaly, consisting in an extremely exaggerated clumping of chromatin in mature leukocytes, associated with loss of segmentation. Ultrastructural studies and flow cytometric DNA analysis, performed in some cases, led the authors to believe that the nuclear anomaly may reflect a faulty distribution of chromatin within the nucleus. No Ph1 chromosome was detected in the four cases available for study, but a clonal 12p anomaly was observed in one case. The authors suggest that cases displaying this nuclear feature take place among MDS beside CMML, with which it shares may clinical and biological similarities.

J. Baumann et al. used a color TV high resolution image system to compare the blast cells of MDS in the preleukemic stage with those in the fully blastic period. Computerized pattern recognition revealed no significant differences in nuclear and cytoplasmic architecture in the blast cells of the two groups, suggesting a derivation from a single clone. This finding is in contrast with the results of the authors in AML, where blast cells in relapse after complete remission show significant structural differences, suggesting a derivation from different cell clones.

P. Stöger et al. carried out quantitative microspectrophotometric measurements of polymorphonuclear neutrophils in smears of peripheral blood and bone marrow after staining for peroxidase and chloroacetate esterase. The authors observed in most patients with MDS conspicuous variations in enzyme activity indicating profound alterations of neutrophil maturation.

R. Lafuente et al. reported on platelet peroxidase in platelets and in the very immature megakaryocyte precursors in the peripheral blood of ten healthy subjects and in 63 patients suffering from chronic myeloproliferative syndrome (CMPS) and MDS. PPO activity was detected in all platelets of