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Production of Dysplastic Platelets in Chronic Myeloproliferative Diseases

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In a recent study of circulating megakaryoblasts in chronic myeloproliferative diseases (CMPD), Matolcsy and Majdic [1] considered theoretical concepts of platelet production to explain the presence of dysplastic platelets that can accompany these disorders. An additional theory, also deserving consideration, is the formation of platelets within the lungs [2, 3].

Tavassoli and Aoki [4] demonstrated by electron microscopy that megakaryocytes (MKs) with their cytoplasm intact can leave bone marrow to enter the blood. This is consistent with the isolation of MKs with copious cytoplasm from central venous blood [5, 6]. MKs with intact cytoplasm are rarely found in central arterial [5, 6] and peripheral venous [7, 8] blood. These reports indicate a loss of MK cytoplasm across the lungs. A mechanism has been proposed [3, 9] by which platelets are formed from the physical fragmentation of MK cytoplasm within the pulmonary circulation.

Tinggaard Pedersen and Laursen [10] have reported increased numbers of MKs in cubital venous blood of patients with CMPD including a greater proportion of MKs with intact cytoplasm in some patients with myelofibrosis. The latter finding was explained by the presence of active haemopoietic tissue in the peripheral part of the upper extremities of these patients.

In CMPD with myelofibrosis abnormal MKs may enter the circulation from active and reactivated bone marrow. The MKs travel in central venous blood to the heart from where they are propelled towards the pulmonary capillary bed. It is at this site that their cytoplasm undergoes fragmentation to release dysplastic platelets, if the theory of pulmonary platelet production is correct. Within this framework the 'cyto-plasmic membrane inclusions' of MKs observed by Matolcsy and Majdic [1] are possible weak spots at which the fragmentation process could occur.

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