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Platelet Volume and Aspirin-induced Inhibition of Aggregation

To the Editor:

After ingestion of acetylsalicylic acid (ASA) there is inhibition of the release reaction and thus of platelet aggregation in response to collagen and adrenaline (second wave). This inhibition may persist for many days and is said to correlate with platelet turnover measured by the $^{51}$Cr method. Similarly, the duration of the inhibition of lipid peroxidation in platelets, also induced by aspirin, is related to the $^{51}$Cr platelets' survival. Platelet size has also been reported to vary with platelet age and hence survival; large platelets are said to be young platelets, which decrease in size as they age in the circulation; however, this is by no means generally accepted.

We measured the persistence of the ASA-induced inhibition of platelet aggregation and platelet volume (for method see Ref. 5) to determine if the two parameters were related in ten healthy control subjects, seven patients examined 1-21 days after a myocardial infarct, and five women in the third trimester of pregnancy. Both groups of patients were chosen because they had previously been shown to have large platelets. All subjects gave informed consent and their platelets were shown initially to respond to collagen (Hormonchemie 0.5 U/ml final concentration) and to give a double wave of aggregation with adrenaline (Evans 5 $M \times 10^{-5}$). On day 0 each volunteer was given 600 mg of ASA. On day 1 aggregation by collagen and the second wave of aggregation induced by adrenaline were completely inhibited in every volunteer. These tests were repeated daily until both aggregation results had returned to normal. Occasionally there was a difference of one day between the recovery times of the two aggregating agents; the mean figure was then reported.

The recovery time in days was plotted against the mean platelet volume (MPV) in fl averaged from all the daily results (Fig. 1). It will be seen that the recovery time was inversely correlated with the MPV. People with large platelets recovered quickly and vice versa. Patients tended to have larger platelets and recover sooner than the controls.

It has twice been reported that the addition in vitro of only 5%-10% of normal platelets to aspirated platelets will give normal aggregation. If platelet survival is normally 7-10 days, then about 10% of new platelets are formed on day 1 after all the ASA is cleared from the
plasma. But, in fact, aggregation remains abnormal for many days. Thus the persistence of the ASA-induced inhibition is presumably due to an aspirin effect on megakaryocytes. Thus the observed duration of ASA-induced inhibition cannot be taken to measure directly platelet survival or turnover; nevertheless, it must presumably be related to the survival time.

A short recovery time then presumably indicates a rapid turnover, with a disproportionate number of young platelets. Thus these findings apparently support the hypothesis that when there are many young platelets the MPV will be large. Although there are no doubt many exceptions, nevertheless it would appear that in appropriate situations (and these have not yet been delimited) the finding of a large MPV may suggest an increase in platelet turnover.

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5q- Acute Myelogenous Leukemia

To the Editor:

Recently, we have described five patients with refractory anemia, a slight excess of myeloblasts in the bone marrow, and an apparently identical chromosome marker appearing only in the bone marrow and identified as a deletion of the long arm of a No. 5 chromosome.1,2 These patients are still being followed, their clinical picture remains unchanged, the abnormal 5q—clone is still confined to the bone marrow, and no additional changes are present. We now describe five new patients with the same 5q—chromosome anomaly, but with an otherwise different clinical and cytogenetic picture.

The first patient was a 59-yr-old male of Italian extraction who had vague gastrointestinal symptoms for many years. During his first admission in 1967 no specific lesions could be demonstrated, but the bone marrow was not investigated. Over the years his complaints remained unchanged except for increasing fatigue. During the past summer he was admitted because of anorexia and a weight loss of 10 kg. This time he was found to have moderate anemia with a hyperactive and sideroblastic erythroid series, poorly lobulated megakaryocytes, and an excess of myeloblasts (19%) and monocyteid cells (12%) in the marrow. The peripheral blood showed thrombocytopenia and 10% immature cells. Cytogenetic investigations showed that in the bone marrow, as well as in the unstimulated peripheral blood, a cell clone was proliferating, characterized by the 5q—marker chromosome, plus additional numerical and structural anomalies, involving chromosomes No. 3, 6, 9, 15, and 17-19. The same anomalies were consistently found on four repeat studies, of which one was done a few days before he died of subacute myelogenous leukemia.

The second patient, a female born in 1897, was never seriously ill, but her more recent medical history was unknown. She was admitted in extremely poor physical condition, and died from septic shock five days later. She had classical acute myelogenous leukemia, and the cytogenetic investigations of the bone marrow and the peripheral blood showed the 5q—marker plus additional chromosome changes, involving chromosomes 7, 8, 13, 17, and 22.

The third patient, a female born in 1905, also presented with myelogenous leukemia and was known to have had neutropenia and recurrent infections for at least 1 yr. In the marrow, as well as in the unstimulated peripheral blood, the 5q— anomaly plus a supernumerary chromosome 21 were present. Treatment with 6-mer-
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