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Letters to the Journal

Letters are welcomed and will be published as space permits. Like other material submitted for publication, they should be typewritten, double-spaced, should be of reasonable length, and will be subject to the usual editing.

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ACUTE MYOCARDIAL INFARCTION IN THE PRESENCE OF THROMBOCYTOPENIA

To the Editor:

In a recent communication (*Canad. Med. Ass. J.*, 85: 621, 1961), Mustard emphasized the importance of platelets in the initiation of blood clotting, and suggested their possible central role in atherogenesis. If true, this would assign to platelets a uniquely critical role in coronary atherosclerosis, coronary thrombosis and myocardial infarction. While the idea may have merit, it is to be emphasized (as, in fact, Mustard himself so indicates) that atherogenesis and myocardial infarction are complex processes involving the mutual interaction of several factors. Platelets, then, do not necessarily "tell the story".

A case in point recently came to our attention. The patient, a 70-year-old hypertensive white female, was admitted to hospital in pulmonary edema with frank clinical, electrocardiographic and chemical (transaminase) evidence of acute anteroseptal myocardial infarction. For the preceding two or three years, she had had angina pectoris and recurrent generalized ecchymoses. A previous hematologic workup was said to have revealed idiopathic thrombocytopenia. At the time of her present admission, the platelet count was 40,000.

This case would suggest either that platelets are not critical in the pathogenesis of coronary atherosclerosis and thrombosis, or that their role in these processes is quite independent of their number.

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To the Editor:

Drs. Schoenfeld and Goldberger describe a case of atherosclerotic vascular disease with the clinical complication of myocardial infarction in a woman 70 years of age with an assumed three-year history of thrombocytopenia. They suggest that this evidence may mean that platelets are not critical in the pathogenesis of coronary atherosclerosis and thrombosis. They seem to have forgotten one rather important point; that this woman's atherosclerosis, like most other human atherosclerosis, probably started about the time she was born, not three years ago when she became thrombocytopenic. All the available evidence would indicate that a woman of 67 would have extensive atherosclerosis. In view of this, I fail to see how one can develop any argument for or against the role of the platelets in atherogenesis based upon the evidence they have presented.

When atherosclerosis is moderately advanced, intimal hemorrhage itself can play a vital role in the evolution of this disease. As for the complications, most pathologists recognize that thrombosis, intimal hemorrhage and/or vessel narrowing can be important factors. Thus I fail to see how their evidence argues for or against the role of the platelet in thrombosis. They actually present no evidence that their patient had a coronary artery thrombosis, only that she had signs of a myocardial infarction.

Aside from these weaknesses in their contention, the obvious question is, What does a platelet count of 40,000/c.mm. represent? It gives no reflection of the rate at which the platelets are disappearing from the circulation. If the platelet survival in this patient was short (i.e. platelet half-life ½ day), then the platelet turnover would be about 28,000/c.mm. of blood per day. If the platelet half-life were even shorter (as it frequently is in thrombocytopenia), the turnover value would be even greater. As we have reported elsewhere, platelet turnover in normal subjects lies between 20,000 and about 60,000/c.mm. of blood per day. Thus the evidence which Drs. Schoenfeld and Goldberger present gives absolutely no idea about the nature of platelet survival in their patient. The low platelet count could just as readily have been due to increased destruction as to decreased production. One cause of increased destruction is, of course, thrombosis. Perhaps the thrombocytopenia was due to too much thrombus formation rather than too little.

I raise these points to emphasize that if a single case is to be used as an argument for or against a particular hypothesis it requires extensive investigation before even tentative conclusions can be drawn. This apparently was not done in the case reported by Drs. Schoenfeld and Goldberger.

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TWINS OF COMBINED LARGE WEIGHT AT BIRTH

To the Editor:

Regarding Dr. F. Farlinger's letter and inquiry (*Canad. Med. Ass. J.*, 86: 242, 1962) concerning total birth weights of twins, it has been stated (*J. A. M. A.*, 163: 1661, 1957) that *Northwest Medicine* has recorded (37: 137, 1938) that in *Lancet* (2: 1029, 1884) there is a report of twins with total weight of 35 lb. 8 oz.

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