Discussion

Our results show that the increase in mean platelet volume after acute infarction is due not to an excess of large platelets but to a deficit of small ones, which is evidence against the hypothesis that myocardial infarctions are precipitated by excessively large circulating platelets. Our results also suggest that the increase in mean platelet volume does not predate the myocardial infarction because the reduction in numbers of small platelets appeared to start at the time of the myocardial infarction. This reduction was less at one month and was almost back within the normal range at 18 months, suggesting recovery from the effects of the myocardial infarction.

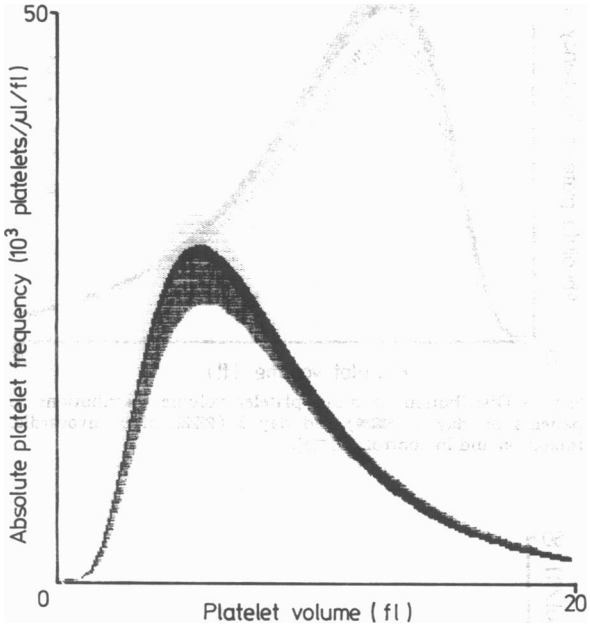

FIG 4—Distribution of means of platelet volume distributions in patients with (■) or without (□) reinfarction after index myocardial infarction. All curves ±1·5 SD.

The patients who reinfarcted after one month had fewer small platelets at one month than those who did not reinfarct, but this difference was not significant. We suspect that this reflected a difference in the severity of their infarcts, which caused both a greater fall in numbers of small platelets and also an increased risk of further infarction. It is, however, conceivable that the decreased numbers of small platelets led to a greater risk of reinfarction but that too few (15) reinfarcted to produce the relevant difference at a significant level.

We did not examine the raw platelet volume data as they were produced by the Coulter counter at each patient's first admission. We did, however, compare the histograms, fitted curves, and curves back plotted from recorded variables for all the patients at their 18 month appointment. We think that the differences were not important, particularly as mean platelet volume itself was calculated from the fitted curve rather than from the raw histogram data. This is, of course, open to the criticism that any differences between histogram and fitted curve would be much more likely to show up at the first measurement after infarction than 18 months later (increase in mean platelet volume being greatest then) and that we failed to notice a deviation of the raw histograms from the fitted curve. (The Coulter counter is supposed to warn the operator if the histogram deviates appreciably from the best log normal curve it can fit. It did not do so for any of our patients at any stage, but it has been reported that it gives this warning very much less often than it should do.[6])

Possible explanations for the reduced numbers of small platelets without reduction in the larger ones include the following: (1) All platelets may be generally consumed but larger ones replaced more rapidly. Although larger platelets were once considered to be younger ones, this is now questioned.[4] When the marrow is stressed, however, the average size of rapidly produced platelets is larger,[7] and we may therefore have observed the result of a partially compensated thrombocytolytic state. (2) Only small platelets may be consumed. As platelets are activated small ones may possibly become exhausted and be removed from the circulation before larger ones. (3) There may be an unrecognised control system for platelet count that takes more account of circulating large volume platelets than of small. If so, a pre-existing reduction in numbers of small platelets might be accentuated by lack of feedback control for these platelets. This theory is partially supported by some evidence in our study that numbers of large platelets appear to be more tightly controlled than numbers of small ones: within all our patient groups the standard deviation of the absolute platelet frequencies at 20 fl was a smaller fraction of their mean (roughly 6%) than at 5 fl (roughly 10%). (4) Bone marrow production of small platelets may be reduced.

If any of the first three explanations were correct it would be in agreement with the finding that the duration of platelet survival is shortened in patients with ischaemic heart disease.[8 9] Determining the relative survival times of small and large platelets in normal patients and in those with acute myocardial infarction would be of interest as this would help to decide between the various explanations.

In conclusion, the reported rise in mean platelet volume after myocardial infarction appears to be due to a deficit of small platelets rather than an increase in large ones. These changes are probably the result rather than the cause of myocardial infarction.

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