turn is largely determined by the response of the sphincter and dilator muscles to autonomic agonist drugs.

In a group of 112 patients with spontaneous acute glaucoma 65 (58%) showed evidence of systemic autonomic dysfunction with standard autonomic function tests (Valsalva's ratio, variation of heart rate during deep breathing, immediate response of heart rate to standing and lying, and decrease in systolic blood pressure in response to standing),[14] compared with a prevalence of 7% in a control group matched for age and sex without glaucoma. Also, the pupils of diabetics are partially denervated and show a supersensitivity to topically applied autonomic mediators—both sympathetic and parasympathetic.[15 16]

Perhaps the observed association between diabetes and acute glaucoma described above is a consequence of autonomic dysfunction within the anterior segment of the eye. Because of this dysfunction some anterior segments develop a heightened response to autonomic mediators, endogenously released or exogenously applied, and the diaphragm of the iris and lens moves forwards and closes the angle. The shallower the anterior chamber at the outset the greater the probability of this occurring.

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Selective consumption of large platelets during massive bleeding

CRAIG B THOMPSON

Abstract

To see whether selective consumption of either small or large platelets occurs during haemostasis in vivo the mean platelet volume was studied in six patients who developed thrombocytopenia after trauma. The mean platelet volume at the onset of thrombocytopenia was significantly lower than that on admission ($p < 0.01$): selective loss of large platelets had occurred.

Introduction

Recent reports have shown that mean platelet volume is increased in patients after acute myocardial infarction.[1 2] It has been suggested that this increase may contribute to the myocardial infarction as large platelets are more active than small ones in assays of in vitro aggregation. Sewell *et al* suggested, however, that the increase is the result of selective consumption of small platelets in vivo.[3] Despite in vitro evidence that large platelets are selectively consumed during platelet aggregation there is no direct evidence that similar selection occurs in vivo.[4 5] To test whether selective consumption of either large or small platelets occurs during in vivo haemostasis I studied the mean platelet volume in patients who developed thrombocytopenia after trauma.

Department of Medicine, University of Washington School of Medicine, Seattle, Washington, United States of America
CRAIG B THOMPSON, MD, department of medicine

Correspondence to: Dr C B Thompson, Fred Hutchinson Cancer Research Center, 1124 Columbia Street, Seattle, Washington 98104, USA.

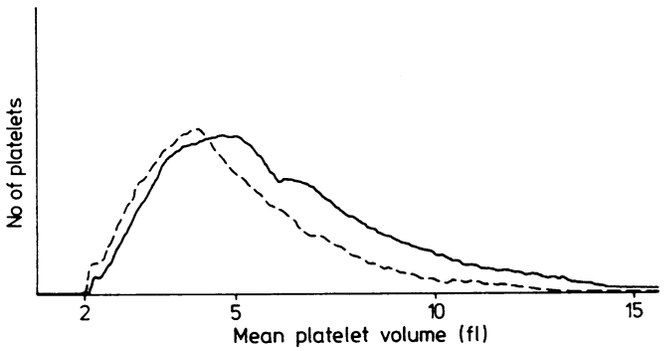
Patients and methods

I studied six patients who were admitted as emergencies after major trauma or rupture of an aortic aneurysm and who initially had a normal platelet count and mean platelet volume, but developed thrombocytopenia within 24 hours after admission. Patients with a history of recent major illness or laboratory evidence of acute ethanol intoxication were excluded.

Complete blood counts, including platelet and mean platelet volume, were obtained (Coulter S+ counter) using blood samples anticoagulated with edetic acid.[2] Each sample was measured twice, the first analysis being done immediately on receipt in the laboratory to determine the platelet count and the second at least two hours after collection to confirm that the mean platelet volume had stabilised. No appreciable variation in the platelet count was observed between the two determinations. The patients' charts were reviewed to ensure that they had been receiving antiplatelet drugs. Initial blood samples were obtained before the patients received any blood products. Blood volume was maintained by giving either packed red blood cells or modified whole blood free of cryoprecipitate and platelets (on average 10 (SD 2) units, range 8-13 units). None of the patients received a transfusion of platelets before developing thrombocytopenia, but all patients received platelets for generalised vascular oozing within 24 hours after admission.

On admission the mean platelet count was 283 (SD 78) $\times 10^9$/l (range 218-398 $\times 10^9$/l) and mean platelet volume 9·0 (0·5) fl (range 8·5-9·8 fl), well within the normal ranges for the laboratory. All the patients developed thrombocytopenia (mean platelet count 95 (30) $\times 10^9$/l, range 61-146 $\times 10^9$/l) during the 24 hours after admission, necessitating transfusion of platelets. The mean platelet volume at the

onset of thrombocytopenia was 7·8 (0·5) fl (range 7·3-8·7 fl), which was significantly lower than that on admission (paired t test $p < 0·01$). Comparison of plots of platelet volume on admission and at the onset of thrombocytopenia confirmed the selective loss of large platelets (figure). Four patients survived; their platelet counts and mean platelet volumes had returned to within the normal range before they were discharged.



Distribution of platelet volume on arrival at hospital after motor vehicle accident (——; platelet count 398×10^9/l) and after development of thrombocytopenia during massive blood loss (- - - -; platelet count 101×10^9/l).

Discussion

This study shows that during massive bleeding selective loss of large platelets can occur, apparently due to the selective consumption of large platelets.[4 5] The changes observed are unlikely to have been the result of changes in thrombopoiesis because of the short time over which the thrombocytopenia developed. The changes are also unlikely to have been due to the circulation of transfused platelets as all patients received blood products deficient in platelets.

This finding has several implications. Firstly, the thrombocytopenia associated with massive trauma and blood loss may be more severe than the platelet count alone would suggest because of selective consumption of the larger, more haemostatically effective platelets. Secondly, it is unlikely that selective consumption of smaller platelets could account for the increased mean platelet volume in patients after acute myocardial infarction. This is more probably the result of the production of larger or "stress" platelets.[2 3] Thus measurement of the mean platelet volume during periods of sustained consumption of platelets may provide insight into the balance between the selective use of larger platelets and the bone marrow's ability to replace them by producing larger, "stress" platelets.

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100 YEARS AGO

A great many letters have reached us on the above subject—so many that it is impossible for us to find space for half of them. We have therefore, determined to deal with the question in this place. Some of the letters before us are written in a spirit we cannot commend, but we have every reason to believe that the writers represent the opinions of a small minority of the medical officers in the military service of the State. It is clear to us that the gentlemen to whom we refer have mistaken their profession. They should have gone to Sandhurst or Woolwich Academy, not to Netley. We do not say that they are ashamed of their profession, but we do say that they are either oblivious or ignorant of the fact that the consideration and status afforded to medical officers in the army depends not on their relative military rank, but on the reputation they make for themselves as skilful surgeons and physicians. Failure in the above all-important particulars will never be made up by any purely military titles, however imposing. Putting aside the small class of claimants who seek distinction more as soldiers than as members of the healing art, we recognise the fact that the position of army medical officers is somewhat anomalous. They are now, after a long struggle, entrusted with command over their own men, and in their own hospitals. The duties they have to discharge are in a high degree onerous and responsible; and, in addition to the dangers peculiar to their calling, they share in those of the field, which, in popular opinion, are supposed to be confined entirely to so-called combatant officers. The enormous mortality among the medical portion of the force lately employed in the Soudan is a painful but honourable illustration of the reality of service-risks to which they are exposed, risks to which high-class insurance-offices are sufficiently awake. If, then, the most modest and reasonable portion of the medical officers of the British and Indian Armies feel that the titles by which they are known do not suffice to impress the public with the composite nature of their position, their duties and responsibilities, we do not think the fact should excite surprise; and, although we do not put forward any scheme by which the injustice can be rectified, we think it is an injustice, and one which should be rectified without awakening the jealousy or wounding the susceptibilities of the purely combatant ranks of the service. We could wish that honours, "the cheap rewards of nations," were given to Army Medical Officers with a less grudging hand. Not in the Army only, but in civil life also, it has been observed that the present Chief of the Medical Staff of the Army, who has twice been charged with the great responsibility of fitting out and organising the health-arrangements of two successful expeditions, is still without any public recognition of the admirable manner in which this difficult duty was discharged. This, to say the least of it, is bad policy. Dr. Crawford has no doubt the approval of his own conscience. He knows he has well discharged his arduous duty; this reward the Government and the nation he has served well can neither give nor withhold, and he owes no thanks to men in office for it. If this is true of our strictly so called "Home Rulers," what are we to say of the Government of India? Nothing less than this, that, of all existing governments, the rulers of this great dependency of the Crown have been the most backward and grudging in doing honour to their medical servants, without whose labours it is not too much to say India could not have been held, except at a cost in human life and suffering that England would not have tolerated. Scores—nay, hundreds—of men have gone to their graves unhonoured by the Government they served. "The Exalted Order of the Star of India" was instituted expressly to reward good service; the Companionship has only twice been given to an Indian medical officer; and in the case of the distinguished officer who is the solitary medical Knight Companion, it is certain that, unless the recipient of the honour had served on the staff of His Royal Highness the Prince of Wales, on the occasion of his memorable visit to India, this decoration would never have adorned his breast. On the authority of a member of the then Government of India who was present, we record the following fact. When the first list of members of the Order was submitted to Sir John Lawrence, afterwards Lord Lawrence, and then Viceroy of India, the name of a medical officer of distinction was included in the list. "What!" said this great administrator, "a Doctor!" as he snatched up a pen; and, with a face red with passion, he erased the name. This is the unwise and unworthy spirit that prevails in high places in Calcutta and in the India Office to this day.

(*British Medical Journal* 1885;ii:351.)