

logically important parameter under conditions approximating those in vivo. This parameter is the resistance of whole blood to flow in microscopic channels, and the simplicity of the published technique stimulated other workers to assess its clinical usefulness. Several parameters have been found to affect the flow of whole blood through small channels in vitro<sup>1</sup> and our recent work<sup>2</sup> was designed to quantify the contribution of some of these to retarding this flow. Among these factors are leucocyte concentration, haematocrit, plasma viscosity and red cell deformability. The first three of these are readily quantifiable and easily manipulated experimentally. Red cell deformability, however, is a picturesque concept but is not a quantitatively defined parameter. Changes in red cell deformability can only be defined as a residual change in (say) whole blood filterability after all other influencing factors have been quantitatively obviated.

On this basis we could find no evidence to support the concept of changed red cell deformability in patients with peripheral vascular disease from our whole blood filtration data. Direct comparison of filtration data between claudicant and normal subjects showed the whole blood filterability to be reduced in the former group, but the extent of reduction could be accounted for by changes in other parameters, notably the leucocyte count. Contrary to Drs Dormandy and Ernst assertion, the data in our paper show that the described relation between leucocyte count and filterability persisted for leucocyte counts below and through the normal physiological range ( $5-12 \times 10^9/l$ ), as well as into the pathological range. The increases in leucocyte count alone were sufficient to explain the reduction in whole blood filterability in our claudicant patients. With no residual differences in whole blood filterability demonstrable between the two subject groups, no differences in red cell deformability could be shown. Of course, the patients we selected may not have had reduced red cell deformability, but this cannot be assessed without an independent measure of this parameter.

That blood flow properties change in patients with peripheral vascular disease is unquestioned. That any of this change is attributable to altered red cell deformability in vivo has yet to be proved.

MJ ALDERMAN  
AA MORLEY  
RG RYALL

*Departments of Haematology and  
Surgery,  
Flinders Medical Centre,  
Bedford Park,  
South Australia 5042*

#### References

- <sup>1</sup> Buchan PC. Evaluations and modification of whole blood filtration in the measurement of erythrocyte deformability in pregnancy and the newborn. *Br J Haematol* 1980;**45**:97-105.
- <sup>2</sup> Alderman MJ, Ridge A, Morley AA, Ryall RG, Walsh JA. Effect of total leucocyte count on whole blood filterability in patients with peripheral vascular disease. *J Clin Pathol* 1981;**34**:163-6.

#### Megakaryocytes in serous effusions

We read with interest the paper "Megakaryocytes in pleural and peritoneal fluids: prevalence, significance, morphology, and cytohistological correlation" by Kumar and Naylor.<sup>1</sup> The authors searched specimens of pleural, peritoneal, and pericardial fluids for megakaryocytes. However, they did not find any in pericardial fluid. In two patients with agnogenic myeloid metaplasia (AMM) foci of haematopoiesis on serous surfaces were found at necropsy. In patient 1 the diaphragmatic pleura and heart were affected by AMM but there was no comment about the presence of pericardial effusions or foci of haematopoiesis on the pericardium.

We have recently had the opportunity of studying a patient with AMM and persistent pericardial effusion. The pericardial fluid contained more than 10 megakaryocytes per slide. Death occurred fifteen days after pericardiocentesis; foci of haematopoiesis on pericardium were found at necropsy and were the cause for the megakaryocyte-containing effusion.

The pericardium, therefore, must be added to those rare serous locations of extramedullary haematopoiesis found in AMM. Although less frequent than pleural and peritoneal such pericardial location may have clinical and diagnostic relevance.

J VILASECA  
JM ARNAU  
N TALLADA  
A SALAS

*Departamentos de Medicina  
Interna y Anatomía Patológica de la  
"Ciudad Sanitaria de Valle de  
Hebron," Barcelona, Spain*

#### Reference

- <sup>1</sup> Kumar NB, Naylor B. Megakaryocytes in pleural and peritoneal fluids: prevalence, significance, morphology and cytohistological correlation. *J Clin Pathol* 1980;**33**:1153-9.

#### Evacuated tubes for monitoring heparin treatment

I read with interest the paper by Professor Heyns and his colleagues of the Orange Free State University, South Africa, entitled "Unsuitability of evacuated tubes for monitoring heparin therapy by activated partial thromboplastin time" which appeared in the January issue.<sup>1</sup>

Although the conclusions of the authors were valid three to four years ago, they are certainly not true today for the Becton, Dickinson Vacutainer brand Blood Collection System.

In 1977, Becton, Dickinson and Company recognised the difficulties one could encounter in the use of evacuated blood collection systems to monitor heparin concentrations in patients by means of the activated partial thromboplastin time (APTT), and set up a programme of product improvement to overcome the problem.

The problem and subsequent solution, was to lie in the stopper formulation. The isoprene-stoppered tubes exhibited sporadic shortening of APTT's on heparinised plasmas and this was attributed to the extraction of divalent cations (calcium and zinc) from the surface of the stoppers. This serious clinical limitation to the use of the Vacutainer brand Blood Collection System motivated the Vacutainer Systems Division to commission a new neobutyl rubber stopper formulation which was compatible with heparin treatment monitoring procedures.

The new improved Vacutainer brand tube with neobutyl rubber formulation stopper was extensively tested in their own laboratories by the in vitro addition of known quantities of heparin, and in hospitals on patients receiving heparin intravenously. These studies showed no significant differences between aged neobutyl rubber formulation stoppered Vacutainer tubes and prepared controls.<sup>2,3</sup>

Three of the lots of Vacutainer brand tubes used by Professor Heyns and his colleagues (lots 7L071, 8A063, C814L022) were manufactured with isoprene stopper materials discarded by Becton, Dickinson and Company in October 1978. Con-