

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 vitro¹ and our recent work² 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

- ¹ 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.
- ² 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.¹ 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

- ¹ 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.¹

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.^{2,3}

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-

sequently, their results are concurrent with those found by Becton, Dickinson.

It should be noted that all lots of Vacutainer brand tubes with neobutyl rubber formulation used in the published study (lots 9B052, 9C055, 9D149, 9B127, 8B622) compared favourably with the siliconised glass reference method.¹

In summary, the conclusions formed by the authors are valid for the obsolete isoprene-stoppered Vacutainer brand and Venoject tubes. Since 1978, the Vacutainer brand citrate tubes for coagulation studies have had a continuous update programme including the use of special rubber formulations to eliminate erratic results and heparin inhibition, chemical buffering of the citrate molarities for maximum glass/rubber/solution compatibility and a non-soluble tube wall coating to minimise blood/glass activation. Vacutainer brand citrate tubes are supplied with sterile interior. These new improved

Vacutainer brand citrate tubes are the result of a continuous product improvement programme and have all been meticulously documented with normal plasma and that of patients treated with oral and intravenous drugs. The control method used in these evaluations is blood drawn into a polypropylene syringe and aliquoted 9:1 into polypropylene tubes containing freshly prepared citrate solutions.

Copies of the references cited, and further information on the Becton, Dickinson Vacutainer brand Blood Collection System may be obtained directly by writing to me.

J CUMMINGS
Vacutainer Systems UK,
York House,
Empire Way,
Wembley,
Middlesex HA9 OPS

Vacutainer is a trade-mark of Becton, Dickinson and Company. Venoject is a trade-mark of Terumo Corporation.

References

- 1 Heyns A duP, van den Berg DJ, Kleynhans PHT, du Toit PW. Unsuitability of evacuated tubes for monitoring heparin therapy by activated partial thromboplastin time. *J Clin Pathol* 1981; 34: 63-8.
- 2 Becton, Dickinson & Company. Technical Report. Performance characteristics of improved B-D Vacutainer brand tubes containing 3.8% sodium citrate. March, 1977. (Data on file.)
- 3 Becton, Dickinson & Company. Technical Report II. Performance of B-D Vacutainer brand tubes containing buffer 3.8% sodium citrate. August, 1977. (Data on file.)

Book reviews

Synopsis of Pathology. 10th ed. WAD Anderson and TM Scotti. (Pp 804; illustrated; £14.75.) YB Medical Publishers Ltd. 1980.

Significant changes are evident in this new (soft-backed) edition of "Anderson's Synopsis". Most are improvements; the book is enlarged to almost the same page format as the *J. Clin. Path.*, with double columns, permitting the inclusion of even more information in a readable way and allowing the more numerous illustrations and tables to fit more comfortably. However, the quality of printing and of the illustrations is not quite up to the previous high standard. The first 278 pages, on general pathology, are rather dated; throughout the book there is an emphasis on descriptive pathology rather than the scientific basis of disease processes. The character of the book remains unchanged; the approach is definitely North American and, to the medical student reader, the coverage continues to be encyclopaedic rather than synoptic.

IC TALBOT

Methods in Hematology. Vol 1. Iron. Ed JD Cook. (Pp 180; illustrated; £17.) Churchill Livingstone. 1980.

A glance at the distinguished Editorial Board which guides this new series shows why their first thought should have been "Iron". It is difficult to provide definitive methods in a field which is still evolving but the editor has had the benefit of some outstanding contributions. The chapter on serum ferritin assay will be particularly helpful both in routine haematology practice and in research. There are also practical contributions on the measurement of serum iron and tissue iron stores. Other chapters on iron absorption, erythrocyte protoporphyrins, and a unique contribution on electron microscopy will, as the editor says in an extensive preface, be more relevant to research studies. Sadly the chapter on ferrokinetics is somewhat dated. Nevertheless it is generally an advantage to have an expert personal opinion rather than the artificial compromise of a committee. This series should find a place at the bench in every haematology laboratory.

I CAVILL

Renal Pathophysiology. 2nd ed. A Leaf and RS Cotran. (Pp 410; illustrated; £7.95.) Oxford University Press. 1980.

This book gives a concise account of the circulation and physiology of the kidney followed by clear, up-to-date sections on fluid and acid-base regulation, the pathophysiology of upsets in potassium, sodium, calcium, magnesium and phosphate, and oedema, diuretics, and renal failure.

Chapters concerning the morphology, pathogenesis and clinical implications of glomerulonephritis, vascular disease, hypertension, and pyelonephritis, and other tubulo-interstitial diseases are highly condensed and could usefully be expanded. Illustrations, though few, are of high quality.

This is a compact, readable account of a complex field which should be useful to undergraduates and postgraduates, though the latter should supplement it with more detailed text.

HM CAMEROON