Mean Milk pH and Colony Counts of Specific *E. coli*

Milk		Count after Incubation (organisms/ml)					
Group	pH	1 hour	2 hours	3 hours	4 hours	5 hours	6 hours
I	6.58	5.1×10^3	9.3×10^3	4.6×10^4	8.6×10^4	5.3×10^5	3×10^6
II	7.20	5×10^3	8×10^3	3×10^4	7×10^4	1.6×10^5	4.6×10^5
III	7.40	3×10^3	5×10^3	9.3×10^3	2×10^4	4.6×10^4	7.8×10^4
P (Group I & II)		>0.1	<0.02	<0.05	<0.05	<0.01	<0.01
P (Group I & III)		>0.1	<0.01	<0.01	<0.01	<0.01	<0.01

water was added to untitrated samples. Viable *E. coli* counts were made on 6 ml of milk at the reconstituted pH and at pH values of 7.20 and 7.40. The specific *E. coli* O111/B4 inoculum was prepared from a 12-hour actively growing culture in tryptone water. Each milk sample was inoculated with approximately 2×10^3 organisms and incubated for six hours at 37°C. Counts were made hourly in triplicate on blood agar plates by the method of Bullen *et al.*¹

The results shown in the table indicate a bacteriostatic effect of titrated milk which is more marked after six hours and most obvious at a pH of 7.40. The bacteriostatic effect appears to be pH-related as it was originally produced in milk after an addition of electrolyte-free THAM (trometamol) as well as sodium bicarbonate.

We trust that the disagreement regarding these findings will not detract from the main point of the original article—namely, that the ability of titrated milk to colonize an infant's bowel with lactobacilli rather than *E. coli* may provide some protection against gastroenteritis.—We are, etc.,

V. C. HARRISON
G. PEAT

Department of Paediatrics and Child Health,
University of Cape Town,
Cape Town

¹ Bullen, J. J., Wilson, A. B., and Cordner, K., *Journal of Pathology and Bacteriology*, 1961, 82, 383.

Traveller's Ankle

SIR,—Many people have had to give up long journeys by coach or aeroplane on account of the ankle swelling and discomfort caused. Passengers in cars or trains seem to be immune, and it would appear that it must be the unrelieved knee flexion that is to blame. Similar conditions, as in a crowded air-raid shelter or over-long spent with the knees tightly flexed over the rail of a desk chair (shelter leg; steamer leg) occasionally gave rise to deep venous thrombosis. These cases were often attributed to imagined kinking of the popliteal vein with obstruction. But no evidence of any such obstruction can be seen when the knee is flexed during phlebography. Alternative hypotheses are therefore proposed.

Veins extensively intercommunicate and offer a variety of alternative routes to the venous blood. When blood flow through them is at a minimum there must often be segments of vein of which the contents are completely stagnant. The veins in which this would be expected most to occur in the "coach" position would be the numerous wide sinusoidal veins in the calf muscle. These are indeed the veins in which deep venous thrombosis most commonly starts.

The usual cause of ankle oedema is locally raised intracapillary pressure. This rises some 80 or 90 mm Hg on changing from a recumbent to a standing position, or about 60 mm

Hg on adopting the "coach" position. But activating the calf pump reduces this by about 50%. This gives any oedema that has formed an opportunity to absorb. Even the little movement carried out in a train or a car is evidently enough. I therefore suggest that sufferers from "traveller's ankle" should exercise their toes and ankles quite briefly, say every half-hour. This simple manoeuvre could also hope substantially to reduce the incidence of deep venous thrombosis in association with operation, with childbirth, and even with "medical" illness. To the question "Why does not spontaneous thrombosis occur at night?" the answer was surely implicit in a recent television programme on sleep; for it seems that during natural sleep we move a great deal without knowing it.

Obviously no single doctor is going to have enough material for significant analysis; but if those who try my suggestion care to write to me about the result I will write again to this journal in 12 months' time.—I am, etc.,

H. DAINTREE JOHNSON

Department of Surgery,
Royal Postgraduate Medical School,
London W12

Prevention of Pulmonary Embolism

SIR,—Though we share most of the views on pulmonary embolism expressed by Mr. F. S. A. Doran in his letter (9 June, p. 612), we must question his statement that the measurement of fibrin/fibrinogen degradation products is impractical. The methods he mentions, gel chromatography and the serial dilution protamine sulphate test, are indeed tedious and difficult. Fibrin/fibrinogen degradation products may, however, be easily and accurately measured using the latex test¹ and the tanned red cell haemagglutination immunoassay.² Excellent commercial kits for these estimations are available from Burroughs Wellcome. Commercial preparations of lyophilized staphylococci are also available from Sigma Chemicals or Hoechst Pharmaceuticals for the staphylococci clumping test,³ which can easily be performed in 10 minutes by any technician. We have found that 60-70 such tests can be completed in two hours.

We recommend that Mr. Doran try one of these simple tests. We are sure that he will find them an invaluable diagnostic aid in pulmonary embolism.—We are, etc.,

D. R. B. JONES
I. M. C. MACINTYRE
C. V. RUCKLEY

General Surgical Unit,
Western General Hospital,
Edinburgh

¹ Garvey, M. B., and Black, J. M., *Journal of Clinical Pathology*, 1972, 25, 680.
² Merskey, C., Kleiner, G. J., and Johnson, A. J., *Blood*, 1966, 28, 1.
³ Leavelle, D. E., Mertens, B. F., Bowie, E. J. W., and Owen, C. A., *American Journal of Clinical Pathology*, 1971, 55, 452.

Erythrocyte Sedimentation Rate in Hyperlipidaemia

SIR,—I believe that my colleagues and I¹ have already published the clue to the question posed by Professor L. E. Böttiger and others (23 June, p. 681) as to why the E.S.R. is raised in many hyperlipidaemic subjects. These authors correctly hypothesize that it is a reflexion of hidden vascular disease. We were able to show that the level of serum fibrin monomer, which is the first breakdown product of fibrinogen and an indicator of in vivo thrombin formation, is directly related to the beta-lipoprotein level in diabetic subjects. In another study (to be published elsewhere) we have also found that serum fibrin monomer levels are high in youngish subjects experiencing their first coronary thrombosis, and furthermore that platelet factor 4 levels, a real indicator of in vivo platelet breakdown,² are related to serum fibrin monomer levels and also to the serum cholesterol.

It seems to be a matter of opinion as to which factors are really important in determining the E.S.R. I am convinced that Lipinski *et al.*³ were correct when they demonstrated that it is the level of serum fibrin monomer that determines the E.S.R. and that variations in other proteins, "agglomerins," and lysolecithin are not the basic explanation.

I therefore envisage a sequence⁴ whereby hyperlipidaemia will lead to enhanced platelet aggregation, with platelet factor 4 release causing in vivo thrombin formation, which will mean increased fibrin monomer levels. In turn the increased fibrin monomer could lead to increased fibrinogen synthesis, as had indeed been shown for fibrin degradation products which, when increased, lead to increased fibrinogen synthesis.⁵ This would be the explanation of the increased fibrinogen levels and increased fibrinogen synthesis rates in atherosclerotic subjects, and very likely would also account for the observation that fatty acid levels can influence fibrinogen synthesis.⁶—I am, etc.,

E. N. WARDLE

Department of Medicine,
Royal Victoria Infirmary,
Newcastle upon Tyne

¹ Wardle, E. N., Piercy, D. A., and Anderson, J., *Postgraduate Medical Journal*, 1973, 49, 1.
² Fuster, V., Kazmier, F. J., Cash, J. D., Bowie, E. J. W., and Owen, C. A., *Mayo Clinic Proceedings*, 1973, 48, 103.
³ Lipinski, B., Worowski, K., Mysliwiec, M., and Farbiszewski, R., *Thrombosis et Diathesis Haemorrhagica*, 1969, 21, 196.
⁴ Wardle, E. N., British Diabetic Association Summer Meeting, Newcastle upon Tyne, 1973.
⁵ Bocci, V., and Pacini, A., *Thrombosis et Diathesis Haemorrhagica*, 1973, 79, 63.
⁶ Pilveram, L. O., and Pickart, L. R., *Journal of Atherosclerosis Research*, 1968, 8, 155.

Consultant Surgeons and Vasectomy

SIR,—Mrs. Marjorie Waite's thorough and fascinating paper (16 June, p. 679), deals with various aspects of vasectomy, including estimates of the number of operations performed in 1970. This information would be much more interesting and meaningful if it could be related to known demand for the operation; no attempt is made in the paper to assess this.

Our experience is relevant here. This is a practice of five doctors, providing general medical services wholly to N.H.S. patients. We offer vasectomy free of charge to registered patients, and have done so since