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 2×10^2 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.,

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¹ Bullen, J. J., Wilson, A. B., and Cordiner, 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.,

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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.,

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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,
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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

January 1971. Besides being free of charge, the service is readily available without a waiting list. Under these circumstances, from our 13,000 patients, we have in the first year performed 100 vasectomies; in 1972 there were 75 and this year so far the demand is again at the rate of 100 per year.

Projected throughout the country these figures suggest a continuing demand for vasectomy, approaching half a million per annum. Mrs. Waite's paper underlines the still considerable lag in meeting this demand.—I am, etc.,

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SIR,—I was most interested to read Mrs. Marjorie Waite's paper (16 June, p. 629) and I should like to comment on the section headed "Inpatient or Outpatient?"

In the 1,000 cases followed up by the Simon Population Trust Vasectomy Project, published in 1969¹ no significant differences were reported in respect of minor post-operative complications (haematoma, infection, etc.) between men operated on under a local anaesthetic and sent home the same day and those operated on under a general anaesthetic and retained overnight in hospital. The work of the former Simon Population Trust Sterilization Project is now continuing with the same staff under the title the Crediton Project. The vast majority of our applicants opt firmly for a local rather than a general anaesthetic.—I am, etc.,

L. N. JACKSON
Hon. Director,
The Crediton Project

Crediton, Devon

¹ Simon Population Trust, *Vasectomy: Follow-up of a Thousand Cases*. Cambridge, S.P.T., 1969.

Persistent Fertility after Vasectomy

SIR,—As is now well known, one of the main snags of vasectomy is the need to keep on examining successive specimens of semen until sperm can no longer be found. Some men continue to show sperm in their ejaculate as long as 18 months after vasectomy. It is true that these sperms are usually non-motile, and many authors consider that such non-motile sperms can safely be regarded as being dead, and if only non-motile sperms are found in the ejaculate they consider it reasonably safe to inform the patient that the object of the operation has now been accomplished. If the vas is irrigated towards the urethra with sterile water or various solutions, the sperms in the ejaculate become non-motile more quickly and several workers have used these solutions in the hope of achieving virtually instant sterility.

We wish to introduce a note of caution, for it appears that things are not so simple. Tyler *et al.*¹ have been using revived frozen human semen for artificial insemination with success, but these thawed-out sperms are never motile. There are other substances (some of them antibodies) which can make sperms non-motile without killing them. These may be present in seminal plasma and can be washed off the sperms, which then become motile again.²

One way to make sure that sperms are dead and not merely motionless is to see if

they take up vital dyes such as trypan blue. We killed fresh sperm by incubating them with 0.25% chlorhexidine in 70% ethyl alcohol and then added trypan blue: all the sperms took up the dye. When we incubated samples of semen (in triplicate) from different patients with distilled water in vitro, though all the sperm became non-motile, only 30% took up the vital dye, suggesting that as many as two out of three were not dead. Similarly, when the same experiment was repeated using aqueous 1/5,000 chlorhexidine, though all the sperms were rendered immotile only 70% took up the vital dye.

As these in vitro studies cast doubt on the efficacy of these irrigating solutions we carried out a few pilot studies in vivo, injecting 5 ml of either sterile distilled water or 1/5,000 aqueous chlorhexidine towards the seminal vesicle after vasectomy had been performed. Of the six patients injected with distilled water, two still had motile sperms 48 hours later and only 50% of the sperms took up trypan blue. Of the six patients injected with chlorhexidine, three still had motile sperms in the ejaculate at 48 hours and only 70% of the sperms took up trypan blue.

We are pursuing these studies to see whether these or similar irrigations can speed up the process of achieving sterility after vasectomy, and it is quite possible that two or three weeks after vasectomy some such irrigation may have achieved its effect. But we do urge surgeons to be very cautious in their interpretation of non-motile sperms in the semen. The significance of a few non-motile sperms months after vasectomy is also doubtful, and further studies are now in progress to compare the motility of these sperms with their ability to take up vital dyes.—We are, etc.,

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¹ Tyler, E. T., *Proceedings of the 3rd European Congress on Sterility*, Athens, 1972, p. 228A.

² Halim, A., Antoniou, D., Leedham, P. W., Blandy, J. P., and Tresidder, G. C., *Proceedings of the Royal Society of Medicine*, 1973, 66, 373.

Regulation of the Profession

SIR,—The Special Representative Meeting at Folkestone approved, with some amendments, the draft report of the B.M.A. Working Party on evidence to be submitted to the Merrison Inquiry into the Regulation of the Medical Profession (*Supplement*, 23 June, p. 137). This draft was first seen by the majority of the representatives when they attended the A.R.M. and they had no opportunity to discuss it with their divisions because the S.R.M. was persuaded that it had to be approved quickly. It is very disturbing that the most important decision the B.M.A. has had to take in over a century should have been made in indecent haste.

The present body which regulates the profession is the General Medical Council, and it was dissatisfaction with its composition and functions which led to the present government inquiry. When the G.M.C. was set up in 1857 the B.M.A. was an embryo organization and did not command the support and resources it now does. It is not surprising, therefore, that the tradition has

Ampicillin for Sore Throat

SIR,—The isolation of *Haemophilus* species from a third of the throat swabs examined from Dr. A. A. Robertson's patients with sore throat (9 June, p. 614) does not, as he suggests, indicate the need for ampicillin treatment. The normal carrier rate of this species in the throat is about 50%, whereas haemolytic streptococci are found in only about 5% of healthy people.¹ The number of patients from whom *Haemophilus* was isolated was therefore less than might have been expected.

It is often desirable to treat acute sore throat before a laboratory diagnosis can be made. Penicillin is the drug of choice for the only common bacterial pathogen, *Streptococcus pyogenes*. Ampicillin unnecessarily disturbs the patient's normal bacterial flora, and side effects are comparatively common. It is, in my view, contraindicated for undiagnosed acute sore throat.—I am, etc.,

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¹ Topley, W. W. C., and Wilson, G. S., *Principles of Bacteriology and Immunity*, 5th edn., ed. G. S. Wilson and A. Miles. London, Arnold, 1964.

Intravenous Cordon Bleu

SIR,—Dr. Bryan Williams, in his *Personal View* (23 June, p. 712), refers to the days when the cordon bleu of intravenous work was attained by the ability to inject black silver salvarsan intravenously. This test of skill would have been unnecessary if a glass adapter had been placed between the needle and syringe to allow blood to be seen when the needle entered the vein.¹ This "window" is a useful but little-known aid when injecting dark solutions into veins.—I am, etc.,

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¹ Birch, C. A., *British Medical Journal*, 1952, 1, 1029.

persisted that the B.M.A. has little place in the regulation of the medical profession and even now there are only 11 elected members out of a total of 46.

The main function of the B.M.A. in the last century has been to unite the profession on the principle that all doctors have more in common than that which divides them. A logical sequel to this principle is that the B.M.A. should become the body regulating the whole medical profession. Some other bodies may question this, but one would have expected the B.M.A. itself to press this argument on the Merrison Committee. The draft report at the outset rejected any suggestion that the B.M.A. should be the responsible body for registration and for professional standards. It sees the functions of the new body as almost exactly like those of the old G.M.C. but with a vast increase in membership which will make the new body even more unwieldy. In doing so, it condemns the B.M.A. for at least another century to a negotiating role for fees and salaries and