	Control Group		Treated Group		P
	No.	Mean $\pm$ S.E.M.	No.	Mean $\pm$ S.E.M.	
Total anuric period (diuresis <100 ml/day) (days) ..	19	10.58 $\pm$ 1.14	21	8.43 $\pm$ 1.66	>0.2*
Total oliguric period (diuresis <500 ml/day) (days) ..	17	15.59 $\pm$ 1.77	17	11.94 $\pm$ 1.26	>0.05*
No. of dialyses	22	6.09 $\pm$ 1.15	20	5.50 $\pm$ 0.72	>0.5†
Time to reach spontaneous decrease of blood urea (days) ..	22	22.05 $\pm$ 2.41	19	19.53 $\pm$ 1.69	>0.2*

*Student's *t* test.

†Mann and Whitney test.

differences between the two protocols utilized. (1) They used higher doses of frusemide—2,000 mg in repeated daily doses until dialysis became unnecessary. (2) It is not stated in their series whether patients treated with frusemide were similar to the controls in all respects—for example, aetiology and severity; were the two patients with obstructive uropathy and acute glomerulonephritis in the treated or control group? (3) What was the mode of randomization? The very disparate numbers of patients treated with frusemide (39) and without (19) during the second period (1969-72) is disturbing, particularly if "treatment was allocated on an alternated patient basis." As suggested a few lines below this statement, the explanation for this difference is perhaps that some patients were excluded from the control group because they had received frusemide before referral to the hospital. If so, the two groups become dissimilar and cannot be submitted to further statistical evaluation.

Since the results of our strictly randomized study do not confirm a beneficial effect of frusemide in established acute renal failure, we do not agree with the conclusions reached by Dr. Cantarovich and his colleagues.—We are, etc.,

D. GANEVAL

D. KLEINKNECHT

L. A. GONZALES-DUQUE

Hôpital Necker,
Paris

Correction of Serum Calcium Measurements

SIR,—It will not be long before the clinicians at this hospital read the two articles on the above subject (15 December, p. 640 and p. 643) and ask when I intend to correct my serum calcium measurements for the patient's serum albumin level. This will put me in somewhat of a dilemma. For a measured serum calcium of 8.0 mg/100 ml and a serum albumin level of 3.8 g/100 ml corrected calcium level, according to Dr. E. M. Berry and his colleagues (p. 640), would be 8.7 mg/100 ml, yet according to Dr. R. B. Payne and his colleagues (p. 643) it would be 8.2 mg/100 ml. Should I therefore give them both corrections and let them exercise their clinical freedom in choosing which correction best suits the clinical situation?—I am, etc.,

C. SANDERSON

Biochemistry Department,
Airedale General Hospital,
Keighley, Yorks

Acute Pancreatitis and Diabetic Ketoacidosis in Hypothermia

SIR,—In their paper reporting the apparent association of acute pancreatitis and diabetic ketoacidosis with hypothermia Dr. D. Maclean and his colleagues (29 December, p. 757) fail to mention whether arterial or

venous samples were used for the measurement of serum amylase and blood glucose levels. Conclusions drawn from venous samples taken during hypothermia are notoriously unreliable because of stagnant blood flow in the cold.¹ The mean serum amylase value for the series $\pm$ 1 S.D.) was 386 $\pm$ 556 Somogyi units/100 ml and that of blood glucose 149 $\pm$ 158 mg/100 ml, indicating a huge scatter of values. Because of this scatter it is likely that these means would not be significantly different from means of values measured in a control group of subjects with normal body temperatures.

Even acid-base calculations made from arterial samples can be misleading unless corrected to what they would be at normal body temperatures.² It is generally accepted that acidosis is not usually severe during hypothermia but may be during rewarming.³ It is likely to be dangerous to the patient if measures are taken because of diagnoses made from biochemical estimations subject to error due to cold.—I am, etc.,

R. E. G. SLOAN

Cheltenham, Glos

- Cooper, R. E., in *Recent Advances in Medicine*, ed. D. N. Baron, N. D. Compston, and A. M. Dawson, 15th edn., p. 343. London, Churchill, 1968.
- Hockaday, T. D. R., and Fell, R. H., *British Journal of Hospital Medicine*, 1969, 2, 1083.
- Keatinge, W. R., *Survival in Cold Water*, p. 70. Oxford, Blackwell, 1969.

Effect of Viruses on Lymphocyte Reactivity

SIR,—Studies of lymphocyte sensitization in health and disease continue to appear in large numbers. The important observations made by Miss Elizabeth Thompson and others (22 December, p. 709) are therefore most timely, since it is not widely appreciated how exposure of lymphocytes (both in humans and experimental animals) to certain common viruses may greatly influence their reactivity to antigens. The phenomenon first obtruded itself in the M.R.C. Demyelinating Diseases Unit in Newcastle in early 1972, when the occurrence of an influenza epidemic was accompanied by wildly random results in the macrophage electrophoretic mobility test.^{1,2} Between 25 January and 2 March all normal guinea-pigs within the colony used to prepare macrophages for the test were found to be sensitized to P.P.D. as well as to encephalitogenic factor (E.F.). A description of what occurred has been published,³ and the episode led to a systematic study of the manner in which guinea-pig lymphocytes reacted to the above antigens after injection with some common viral vaccines. It was found that cellular sensitization occurred not only to the specific inoculum but also to P.P.D. and to E.F., and indeed in lesser degree to other apparently unrelated antigenic stimulants.⁴

Not only do cells from subjects exposed to banal viral infections develop unusual reactivity, but they appear to be "on edge" in the sense that they undergo "spontaneous" transformation in vitro to a greater degree than normally. Such irritability of the cells may persist for some weeks and may indeed occur in the presence of an influenza epidemic even when the subject concerned has not suffered obvious clinical infection.⁵

From the experience gained in the winter of 1971-2 it was possible by introducing the most rigorous discipline in the animal house and laboratories (especially the wearing of masks and removal of workers with "colds") to prevent a recurrence of the troubles in the winter of 1972-3. Inadvertent guinea-pig exposure to infections may be monitored by measuring the response of peritoneal exudate cells to P.P.D. or E.F. in the manner described by Sundaram *et al.*⁶ and Diengdoh and Turk.⁷ A rise above 1.5% in macrophage slowing should be regarded as unacceptable. Recent studies of the sensitivity of lymphocyte-antigen interaction to the presence of linoleic acid⁸⁻¹⁰ will very probably also be highly susceptible to the same errors.

All who hope to achieve consistent and meaningful results while working with lymphocyte reactivity, especially during the winter months, must be prepared to exercise the greatest care in the supervision of animal stocks and should at least inquire about recent exposure of human subjects to influenzal infections.—I am, etc.,

E. J. FIELD

Institute of Pathology,
Newcastle General Hospital,
Newcastle upon Tyne

- Field, E. J., and Caspary, E. A., *Lancet*, 1970, 2, 1337.
- Caspary, E. A., and Field, E. J., *British Medical Journal*, 1971, 2, 613.
- Field, E. J., and Caspary, E. A., *Lancet*, 1972, 1, 963.
- Field, E. J., Caspary, E. A., Shenton, B. K., and Madgwick, H., *Journal of the Neurological Sciences*, 1973, 19, 179.
- Parker, J. W., and Lukes, R. J., in *Proceedings of the 4th Leucocyte Culture Conference*, ed. O. Ross McIntyre. New York, Appleton, 1971.
- Sundaram, K., Phondke, G. P., and Sunderesan, P., *Immunology*, 1967, 13, 433.
- Diengdoh, J. V., and Turk, J. L., *International Archives of Allergy*, 1968, 34, 297.
- Mertin, J., Shenton, B. K., and Field, E. J., *Journal of International Research Communications Systems (Medical Sciences)*, 1973, 1, No. 7, 18.
- Field, E. J., Shenton, B. K., Joyce, G., and Mertin, J., *Journal of International Research Communications Systems (Medical Sciences)*, 1973, 1, No. 7, 17.
- Field, E. J., *Lancet*, 1973, 2, 1080.

Incidence of Postpartum Deep Vein Thrombosis in the Tropics

SIR,—I refer to the articles by Mr. M. A. Hassan and others and by Mr. O. B. Williams and others on postoperative deep vein thrombosis (3 March 1973, pp. 515 and 517) and the letter from Mr. K. R. Orr (9 June, p. 615) which suggests a low incidence of this phenomenon in Vietnam. Since the incidence of postpartum thromboembolic complications in Thailand is also supposed to be rare, 41,056 clinical charts from the Chulalongkorn Hospital for the years 1970-71 were analysed retrospectively to determine the occurrence of postpartum thromboembolism (see table).

The prevalence of postpartum thromboembolic complication was 1.7 per 10,000 deliveries. The results of a similar study at