of exposure to sun and wind, this small group of previously affected children appears to have benefited significantly from the addition of this medication to their skin.—I am, etc.,

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Continuous Insulin Infusion

SIR,—We have read with great interest the papers by Dr. M. McB. Page and others, Dr. Warren Kidson and others, and Dr. P. F. Semple and others on the treatment of hyperglycaemia, ketoacidosis, and diabetic coma by continuous insulin infusion (29 June, pp. 687, 691, and 694). These authors rightly emphasize the importance of continuous insulin infusion in these emergency situations. Though the practice of giving large, frequent doses of insulin in these conditions has been criticized the advantages of continuous insulin infusion have not been generally recognized. Perhaps these reappraisals of infusion therapy together with recent advances in medical technology may change the situation.

Interestingly enough the value of continuous infusion as the treatment of choice for diabetic hyperglycaemia, ketoacidosis, precoma, and coma becomes obvious when one considers the working of the artificial B-cell for the automatic control of blood glucose in diabetics. When such a system of a glucose-regulated insulin infusion (consisting of a Technicon-AutoAnalyzer, a micro-computer, insulin and glucose infusion pumps, and a teleprinter) is used the patient requires from 600 to 1,000 μ U of insulin per minute for about 90 to 120 minutes—that is, so long as blood glucose levels have not returned to normal.¹⁻³ Afterwards only small doses of insulin are needed to maintain normal blood glucose, despite the patient being allowed unlimited food intake as soon as possible. Bicarbonate and potassium are given in usual amounts. In five cases treated so far in this way no untoward side effects have been recorded, despite the rapid lowering of blood glucose produced by our technique.⁴—We are, etc.,

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¹ Albisser, A. M., et al., *Diabetes*, 1973, 22, Suppl. 1, 294.

² Albisser, A. M., et al., 1974. Personal communication.

³ Pfeiffer, E. F., et al., *Naturwissenschaften*, 1974. In press.

⁴ Pfeiffer, E. F., et al., *Hormone and Metabolic Research*, 1974. In press.

Zoster in Three Children in Family

SIR,—The three children of one family, all girls, suffered an attack of varicella in 1966 at a time when the youngest may have been carrying some maternal immunity to the disease. Within a few years each developed zoster (see table).

In the 3,600-3,800 population of our general practice zoster occurs at a rate of 0.7 per 1,000 of persons under 10 years old

Patient	Date of Birth	Age at Varicella	Age at Zoster	Interval between Varicella and Zoster
1	March 1959	7 years	10 years	3 years
2	Oct. 1960	6 years	8 years	3 years
3	Dec. 1965	4 months	22 months	18 months

annually (patient 1 had only just passed her tenth birthday). The chance occurrence of zoster in two siblings is therefore not common, and in fact there have been no other cases of zoster in young siblings among the 350 cases of zoster encountered during 28 years continuous study here. If two cases in the same family are uncommon the occurrence of zoster in three young siblings must be so rare as to fall under the strong suspicion of not being a chance event. Should it not be fortuitous it may offer an opportunity for a better understanding of the mechanisms by which latent zoster-varicella virus is reactivated from latency.

I hope that others with records of siblings, especially children, with zoster will publish the details.—I am, etc.,

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Trasylol for Pancreatitis

SIR,—The leading article (20 July, p. 133) on Trasylol (aprotinin) therapy in the management of acute pancreatitis leaves one with the impression that your advocacy for the use of this expensive substance was based on the findings of a recent double-blind trial.¹ We have previously commented on the high mortality rate of 25% in the control group of patients in that trial.² We have found, in a prospective survey of acute pancreatitis,³ a mortality rate of 11.1% for 90 patients (out of a total 140) who met the criteria of a first attack of idiopathic pancreatitis or gall stone-associated acute pancreatitis. These 90 patients were all managed conservatively without the use of Trasylol (or glucagon). It is therefore our distinct impression that the case for Trasylol remains to be proved against a control group with a much lower mortality rate than 25%.

A further point, on the incidence of the disease, is worthy of comment. Our experience is that acute pancreatitis is a common cause of emergency surgical admission. The catchment population of Glasgow Royal Infirmary is about 500,000 and currently more than 50 patients a year are admitted suffering from acute pancreatitis.—We are, etc.,

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¹ Trapnell, J. E., et al., *British Journal of Surgery*, 1974, 61, 177.

² Imrie, C. W., and Blumgart, L. H., *British Medical Journal*, 1974, 1, 38.

³ Imrie, C. W., and Whyte, A. S. Submitted for publication.

Death after E.C.T.

SIR,—I was interested in Dr. Joan R. Gomez's report of a death after E.C.T. (6 July, p. 45). She did not discuss the question of plasma cholinesterase. Hodges and Harkness¹ pointed out that prolonged apnoea after the administration of suxamethonium was primarily due to a low level

of serum pseudocholinesterase. They associated low levels with patients who were clinically "ill," and they maintained that there was no particular correlation between subnormal pseudocholinesterase levels and any particular disease but rather with the general physical state. Since then much work has been done and numerous papers have associated a low serum cholinesterase with many diseases. There is also the rare person who possesses the silent gene and who has virtually no cholinesterase activity.

Other work, using inhibitors of pseudocholinesterase (dibucaine and sodium fluoride), has identified at least 10 cholinesterase genotypes.² Thus a person in whom the quantitative estimation of serum cholinesterase may be normal may belong to one of these subgroups and his cholinesterase be unable to hydrolyse suxamethonium. Most of these rare groups are identified by their dibucaine and fluoride numbers but some very rare ones can be discovered only by genetic studies of the families. The commonest is the so-called "atypical" group, which occurs in only one out of 3,000 persons. The other groups include the subgroup I₁ of the "intermediate" group and the homozygous fluoride-resistant group. Not all these groups show the same degree of apnoea after suxamethonium but they are all at risk.

Some cases with apnoea do not fall in any of these groups, and possibly if different inhibitors (for example, sodium chloride or tertial butyl alcohol) were used further subgroups at risk might be discovered. Also possibly some of the unexplained cases of apnoea may be associated with a completely different enzyme group. The non-specific aliesterases, for example, detoxicate toxic substances and drugs such as suxamethonium, but in man aliesterases are normally at a low level and it would be difficult to incriminate them.

Dr. Gomez raised the question of the danger of using amitriptyline. This certainly has various cardiovascular effects. It must, however, be remembered that amitriptyline (in common with other tricyclic antidepressants) has a chemical structure similar to the cholinesterase inhibitor dibucaine. In fact amitriptyline is apparently an effective inhibitor of serum cholinesterase.³ The death in her case, therefore, may well have been in a patient in whom the plasma cholinesterase was deficient in quality or quantity and may have been further reduced by the amitriptyline. In this context, therefore, anxiety would have played no part. In fact plasma cholinesterase is very often raised rather than lowered in anxiety states.⁴—I am, etc.,

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¹ Hodges, R. J. H., and Harkness, J., *British Medical Journal*, 1954, 2, 18.

² Harris, H., *Proceedings of the Royal Society of Medicine*, 1964, 57, 503.

³ Kalow, W., Genest, K., and Staron, N., *Canadian Journal of Biochemistry and Physiology*, 1956, 34, 637.

⁴ Rose, L., Davies, D. A., and Lehmann, H., *Lancet*, 1965, 2, 563.