this risk—and at the least avoid prolonged pregnancy.

The second item concerns our double-blind trial of chlorpropamide 100 mg a day in the treatment of gestational chemical diabetes in women of average body weight. We do know that such a dose of chlorpropamide is effective in reversing chemical diabetes in the mother in pregnancy—without apparent harm to the fetus[4]—though we do not recommend this form of treatment for diabetes of a more severe degree, which seems to require more delicate control during pregnancy.[5]—We are, etc.,

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1 Fitzgerald, M. G., and Keen, H., *British Medical Journal*, 1964, **1**, 1568.
2 Sutherland, H. W., Stowers, J. M., and Whethem, J. C. G., *Excerpta Medica International Congress Series*, 1973, **280**, 153.
3 Sutherland, H. W., and Stowers, J. M., in *Carbohydrate Metabolism in Pregnancy and the Newborn*. Edinburgh, Churchill Livingstone. In press.
4 Sutherland, H. W., Stowers, J. M., Cormack, J. D., and Bewsher, P. D., *British Medical Journal*, 1973, **3**, 9.
5 Sutherland, H. W., *et al.*, *Archives of Disease in Childhood*. In press.

Temperature Change and Multiple Sclerosis

SIR,—Within a period of one year, two leading articles in the *B.M.J.* dealing with multiple sclerosis have presented incomplete accounts of our current understanding of the mechanism of temperature effects in this condition.

The omission in the first of these leading articles (2 December 1972, p. 506) was pointed out by Drs. W. I. McDonald and T. A. Sears in a letter (30 December 1972, p. 794) in which they summarized experimental studies in demyelinated nerve indicating a heightened sensitivity to thermal block,[1 2] which by itself gives a sufficient explanation of the clinically observed temperature effects. In the more recent leading article (15 December 1973, p. 626) this clinical temperature phenomenon is discussed in an identically incomplete manner. I do not intend to repeat the contents of the earlier letter from Drs. McDonald and Sears; however, an additional point can be made.

In a recent study[3] the thermal properties of demyelinated nerve have been extensively investigated by the numerical solution of a well-known set of differential equations describing conduction in a model demyelinated axon. In agreement with clinical findings in multiple sclerosis and experimental animal studies this model predicts that the temperature at which conduction block occurs is a steep function of the extent of demyelination alone, so that small temperature increments can block large numbers of conducting fibres. To put the situation in another way, given a population of nerve fibres with varying amounts of myelin loss (such as would be expected to occur in a real lesion in multiple sclerosis) a small increase in temperature would substantially decrease the number of conducting fibres whose blocking temperature exceeds the normal body temperature.—I am, etc.,

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1 Davis, F. A., and Jacobson, S., *Journal of Neurology, Neurosurgery and Psychiatry*, 1971, **34**, 551.
2 Rasminsky, M., *Archives of Neurology*, 1973, **28**, 287.
3 Schauf, C. L., and Davis, F. A., *Journal of Neurology, Neurosurgery and Psychiatry*. In press.

Changing Incidence of Congenital Pyloric Stenosis

SIR,—The letter from Dr. D. E. Murfin reporting an increasing incidence of infantile hypertrophic pyloric stenosis in Barbados (2 February, p. 198) is of some interest because it notes a contrary trend to that which is occurring in most European countries. In Sweden the frequency fell from 4 to 2 per 1,000 live births during a decade.[1] In Belfast County Borough the incidence fell from 3·09 per 1,000 live births in the 7-year period 1957-69 to 2·22 per 1,000 live births in the 6-year period 1964-9 ($\chi^2 = 6{\cdot}2$; D.F. = 1; $0{\cdot}025>P>0{\cdot}01$).[2] A superficial observation of the numbers of affected infants seen at Llandough Hospital, Cardiff during the past two decades shows a similar decrease. In both Belfast and Cardiff there was a rather striking increase in numbers in 1962 which is unexplained. In contrast Ströd r[3] reported an increase in the number of cases seen at a hospital in West Germany, but this was not related to population incidence and may be explicable in other ways.

Despite Dr. Murfin's observation that Negro infants may suffer from this condition more commonly than we have supposed and his suggestion that the rising incidence may reflect environmental changes there is little doubt that racial differences in incidence are at least partly genetically determined. An important study in Hawaii recorded the relative incidence in different racial communities. The preponderance of Caucasians was confirmed and no instances were detected among 11,274 Chinese infants. The authors claimed that cultural differences were of minor importance in Hawaii, that medical facilities were shared, and that their findings reflected variations in susceptibility which were determined by race.[4]—I am, etc.,

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1 Wallgren, A., *Acta Paediatrica*, 1960, **49**, 530.
2 Dodge, J. A., M.D. Thesis, University of Wales, 1970.
3 Stroder, J., *Lancet*, 1962, **1**, 644.
4 Shim, W. K. T., Campbell, A., and Wright, S. W., *Journal of Pediatrics*, 1970, **76**, 89.

Irresponsible Poisoning

SIR,—I was very pleased to see that you have returned to the problem of childhood poisoning in your leading article "Irresponsible Poisoning" (23 February, p. 296). I agree entirely with your conclusion that the dangers of tricyclic antidepressant drugs need to be made clear to all who prescribe them, but was disappointed to see that you decry the use of child-resistant containers. There is very good evidence that the use of these containers would reduce the risk of poisoning by tablets as much as by 90%.[1-3] It is no good relying on pious exhortations to keep medicines out of the reach of children and it is time that positive action was taken. You are also wrong in saying that "most children are at least as resourceful as their parents." At the age of 1-3 years they certainly are not, and this is the age at which poisoning occurs.

What worries me, as a member of the committee of the British Standards Institute concerned in the drafting of the protocol for testing child-resistant containers (not the actual design of the containers as your article states), is that so far as I am aware no further action has occurred since the draft was published. Meanwhile the poisoning goes on.—I am, etc.,

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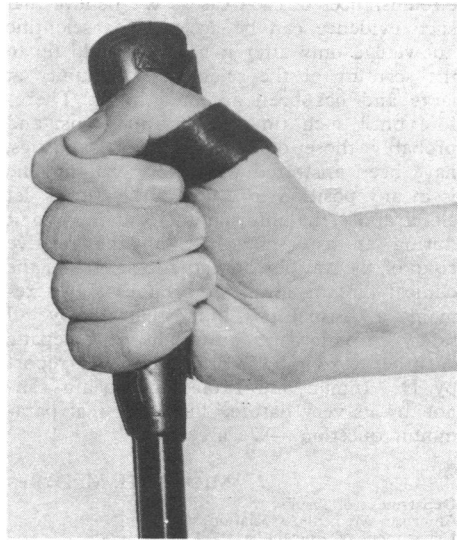
1 Breault, H. J., 125th Annual Meeting, American Association of Poison Control Centers, New York, 16 October 1972.
2 Scherz, R. G., *Pediatric Clinics of North America*, 1970, **17**, 713.
3 Stracener, C. E., and Scherz, R. G., *North West Medicine*, 1970, **69**, 334.

Gamekeeper's Thumb on the Ski Slopes

SIR,—Your leading article (9 February, p. 213) notes yet another type of skiing injury. However, I cannot see how extension of the thumb can damage the ulnar collateral ligament. I believe that, like gamekeeper's thumb, the loose grip is to blame and that this injury can be prevented if bindings have been checked.

The ski stick is normally held through a leather loop on top of the handle that is wrapped around the thumb (see fig.). This loop's main purpose is to prevent loss of



the ski stick in soft snow. If the other fingers do not grip well, twisting and jarring movements when skiing are transmitted through the thumb, which is caught between the stick and the taught loop. The ulnar collateral ligament of the thumb is weakened and later tears on falling (often still trapped in this position).

Skiers can prevent this injury by gripping the ski stick harder—even when they fall. At times the stick can be held without using the loop—for example, on a dry ski slope. Since adopting these simple measures I myself no longer suffer from painful thumbs after skiing.—I am, etc.,

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