suggests that even smallest amounts of isopropyl alcohol carried into the subcutis while injecting insulin can in some individuals lead to another "insulin" allergy besides reactions due to additives, preservatives, or zinc.²

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¹ Diem P, Teuscher A. *Schweiz Med Wochenschr* 1979; **109**:1814-6.

² Feinglos MN, Jegasothy BV. *Lancet* 1979; **i**:122-4.

Anaphylactic reaction to desensitisation

SIR,—Dr David A Rands (27 September, p 854) reported a death due to anaphylaxis following desensitisation for allergic rhinitis. In the past, hyposensitisation was widely used in the treatment of allergic disorders; however, impressions of benefit were often based on anecdote and where controlled trials have been performed results are variable. For example, in studies of desensitisation to the house dust mite, *Dermatophagoides pteronyssinus*, in patients with asthma or allergic rhinitis no benefit was found in one study¹; the British Thoracic Association study² showed marginal benefit (not statistically significant) in patients not on steroids and no benefit in patients on steroids; and two other studies showed improvement.^{3,4}

Today newer drugs (local steroids and sodium cromoglycate) are available which control allergic symptoms in the majority of patients. These drugs, together with measures to reduce exposure to the allergens, are effective and safe and constitute the treatment of choice. Hyposensitisation, which is potentially dangerous and often ineffective, should rarely be required. Although anaphylaxis is a rare complication of hyposensitisation, Dr Rands's article provides a tragic reminder of the consequences of a potentially dangerous form of therapy in a mild disease.

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¹ Gaddie J, Skinner C, Palmer KNV. *Br Med J* 1976; **iii**:561-2.

² Mite Allergy Subcommittee of the Research Committee of the British Thoracic Association. *Br J Dis Chest* 1979; **73**:260-70.

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⁴ Warner JO, Price JF, Soothill JF, Hey EN. *Lancet* 1978; **ii**:912-5.

Unprecedented increase of infantile hypertrophic pyloric stenosis

SIR,—We were interested to read the recent paper by Dr Alison Kerr (13 September, p 714) reporting a remarkable increase in the incidence of infantile pyloric stenosis in Central Scotland during 1978 and 1979. We have for some time been aware of a similar increase in some parts of Wales, and colleagues in other areas of the United Kingdom have also told us of recent local rises.

The Welsh Hospital Activity Analysis, which probably represents incomplete returns, shows that a substantial increase in the numbers of children undergoing Ramstedt's operation occurred from 1976 on. It is of considerable interest that the increased incidence has almost entirely affected male

infants. The numbers of affected females in Wales remained relatively constant, and the sex ratio was not much more than 2:1 from 1970 to 1975 but was as high as 5:1 in 1977 and 1979.

We interpret these findings as indicating that there is a basic incidence in the community which is genetically determined, but that marked increases such as we are observing at the moment may occur as a result of additional environmental factors. These will operate predominantly on the more susceptible sex—that is, on the males—whereas the females, who are relatively protected by their genetic constitution, will not respond to the relatively weak environmental precipitating factor. Like Dr Kerr, we are actively looking into the possible nature of the environmental trigger, and are considering the possibility that the recent marked change in infant feeding patterns, with a resurgence of breast feeding, may be involved.

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SIR,—Dr Alison M Kerr, in reporting the dramatic rise in incidence of infantile hypertrophic pyloric stenosis in the Central Region of Scotland (13 September, p 714), emphasises the importance of knowing whether this change is mirrored elsewhere. A similarly steep localised rise in incidence has been suggested before,¹ but the study was of questionable validity because of inadequate data.

The data in the accompanying table are derived from a survey of all cases of infantile hypertrophic pyloric stenosis born in Buckinghamshire during 1975-9, with diagnoses confirmed at Ramstedt's operation. The overall incidence was 2.3 cases per 1000 live births, with no significant variation ($\chi^2=7.07$; $df=4$; $0.2 > p > 0.1$). In Oxfordshire the overall incidence during 1966-71 was 2.5 per 1000 live births.²

"Epidemics" of pyloric stenosis, localised to particular hospitals, seem to be the experience of many paediatricians. Yet, with few exceptions,² published studies have paid little attention to this aspect of aetiology. Such epidemics, whatever their cause, would offer an alternative explanation for the reported seasonal variation in incidence of pyloric stenosis,³ and reinforce the need for careful application of statistical methods. In particular, the often-used χ^2 test can at best only show lack of variability and cannot effectively be used to measure cyclical changes,⁴ especially when time intervals are arbitrarily chosen to accentuate variability.³

It is surprising, especially as infantile hypertrophic pyloric stenosis is a relatively common condition, how little is known of the aetiology and how few of its aetiological associations at present remain unchallenged. This highlights the need for further information and continuing study.

Incidence of infantile hypertrophic pyloric stenosis in Buckinghamshire (Eton and Beaconsfield Health Districts excluded) in 1975-9

	1975	1976	1977	1978	1979	1975-79
Total live births	5799	6307	6331	6962	7365	32 764
Cases of infantile hypertrophic pyloric stenosis	6	14	21	17	17	75
Incidence (cases/1000 live births)	1.0	2.2	3.3	2.4	2.3	2.3

I thank the consultant paediatricians and surgeons at Stoke Mandeville, Northampton General, Wycombe General, and Wexham Park Hospitals and in Oxford for permission to study their cases.

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¹ Murfin DE. *Br Med J* 1974; **i**:198.

² Adelstein P, Frederick J. *J Med Genet* 1976; **13**:439.

³ Dodge JA. *Arch Dis Child* 1975; **50**:171.

⁴ Edwards JH. *Ann Hum Genet* 1961; **25**:83.

Surveillance of food poisoning and salmonella infections

SIR,—Your article (20 September, p 817) draws attention to the fact that the number of sporadic cases of salmonella infection acquired abroad is not documented, no doubt owing to the present notification system.

It may therefore be of interest that in a survey of 200 patients with salmonellosis admitted to an isolation hospital in Manchester during 1977-9 19 were found to have acquired the infection abroad. Thus approximately 10% of salmonella infections admitted to this isolation hospital were acquired abroad, although it must be noted that this is only a small proportion of salmonellosis in the community.

Analysis of the salmonella infections from abroad shows no consistent pattern either in the country of origin, organism isolated, month of admission, or severity of symptoms—these infections merged into the general picture of salmonellosis.

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Primary biliary cirrhosis: an epidemiological study

SIR,—I was interested in Dr David R Triger's figures on the incidence and prevalence of primary biliary cirrhosis in Sheffield (20 September, p 772), and his comments on the lack of information on this condition in other areas.

In Tayside there is a fairly stable mixed urban and rural population of 397 605 (post-regionalisation revision of 1971 census). A single liver clinic is held in Ninewells Hospital; there is one liver pathologist, also based in Dundee; and practically no referrals are made to or from the area. Since 1975 computerisation has assisted studies of liver disease in particular by rapid retrieval of liver biopsy reports.

In the five-year period 1975-9 inclusive 21 new cases of primary biliary cirrhosis presented in Tayside. In all cases the diagnosis was based on the well-described clinical features,¹ and antimitochondrial antibodies were uniformly present. Histological confirmation of the diagnosis was obtained in all