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.

³ D'Souza MF, Pepys J, Wells ID, et al. *Clin Allergy* 1973; **3**:177-93.

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

cases, although in two female patients this was not obtained until necropsy. There were 19 female and two male patients, the mean age of presentation was 57.9 years, and seven patients (33%) were over the age of 65.

These data reveal that in Tayside the annual incidence of primary biliary cirrhosis is 10.6 per million. On 31 December 1979 16 patients with the diagnosis were still alive, revealing the point prevalence as 40.2 per million. The incidence figure is almost twice that found by Dr Triger in Sheffield (5.8 per million), and by other workers in NE England (6.0 per million—as reported at the September meeting of the British Society of Gastroenterology by Hamlyn *et al.*). The lower value for point prevalence may indicate higher mortality in Tayside, although it is similar to the figure in the Newcastle upon Tyne study (47 per million).

The weakness in the Tayside data is the paucity of information on the number of non-biopsied cases during the study period, and on the number of biopsied cases alive prior to 1975. An attempt is being made to rectify this problem at present, but it is unlikely that these gaps in information have distorted the annual incidence figures greatly or significantly altered the point prevalence as late as the end of 1979.

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¹ Sherlock S, Scheuer PJ. *N Engl J Med* 1973;289: 674-8.

Finger wrinkling after immersion in water

SIR,—Dr G Alvarez and others question the usefulness of the finger wrinkling test, as one-third of the patients investigated by them did not show any skin wrinkling (30 August, p 586).

I recall Mr Wilfred Hynes, at a British Association of Plastic Surgeons meeting in Bristol some years ago, pointing out that when one describes a new operation or test your colleagues do two things—first they modify the procedure and then they come back and tell you that it does not work. The authors kindly refer to my description of the test, but I must point out that they did not carry out the test as described by me.¹ I clearly stated that the hand and forearm should be immersed in water at 40°C for 30 minutes. They did not immerse the forearm, nor did they keep it in water for 30 minutes.

I carry out this test as a routine on patients attending my clinic and find it most useful. A small proportion, perhaps 10-15%, do not show skin wrinkling and in these the test is of no use, but neither is it misleading. Prospective users of this test should also be warned that it does not become positive for a few days after peripheral nerve injury.

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¹ O'Riain S. *Br Med J* 1973;iii:615-6.

SIR,—Dr G Alvarez and others (30 August, p 586) state that it is safe to say that sympathetic function is preserved if finger wrinkling after immersion in water is observed. However, Bull *et al.*¹ showed that a sympathectomised hand deprived of its blood supply will wrinkle normally. The corollary may well be true—that a hand with an intact sympathetic supply but a high blood flow might not wrinkle. This would explain the large percentage of false-positive results that Dr Alvarez and his colleagues found. It is known that an individual may have different flow rates in the two hands. It has been suggested that the dominant hand has a higher flow rate.² They tested only one hand: was it the patient's dominant hand?

I feel that they have usefully pointed out the possibility of a false-positive result occurring with this test. It would have been even more useful to have tested both hands to ascertain whether the variance in their results was symmetrical. If they had done so we would now know whether the opposite hand could be used as a control in cases of unilateral disease such as in peripheral nerve injury.

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¹ Bull C, Henry JA. *Br Med J* 1977;ii:551-2.

² Kumar PP, Kovi J. *JAMA* 1978;70:837-40.

*We sent this letter to the authors, and Dr Alvarez replies below.—ED, *BMJ*.

SIR,—Testing for finger wrinkling was carried out under ordinary bedside conditions. Our habit was to use the right hand as a rule, but this was not invariable and dominance was not ascertained. Mr Williams's theoretical objection therefore stands, although we feel that if in normal individuals wrinkling in only one hand after prolonged wetting were a habitual occurrence attention would have been drawn to it.

A number of our subjects were manual workers. It could thus be argued that mild thickening of the skin over finger pulps and hypothetical trauma to peripheral nerves could account for some of our false positives, but not for as many as we found.

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Porphyria cutanea tarda and beta-thalassaemia minor with iron overload

SIR,—I would like to answer the points raised by Dr R S Day and his colleagues (20 September, p 809) concerning my case report of familial porphyria cutanea tarda and β -thalassaemia minor in a mother and daughter (24 May, p 1255).

(1) They state that the urinary uroporphyrin levels quoted are low for overt porphyria cutanea tarda in their experience. It is clearly stated in the report that porphyria cutanea tarda was not clinically overt in 1968 when the urinary uroporphyrins were measured. I would also refer them to the extensive experience of Harber and Bickers,¹ who state that the levels of urinary uroporphyrin vary widely and cannot be correlated with the degree of clinical involvement in any absolute manner.

(2) Dr Day and his colleagues state that a full porphyrin analysis is necessary to confirm the diagnosis of porphyria cutanea tarda, and raise the possibility of variegate porphyria in the mother. A full biochemical porphyrin analysis is, of course, necessary for accurate diagnosis and it was performed in both mother and daughter, but for

reasons of brevity details of the full biochemical porphyrin analysis were not included in the short report. However, raised urinary uroporphyrins and low (as reported in both patients) or slightly elevated urinary coproporphyrins are characteristic of porphyria cutanea tarda unlike variegate porphyria, where raised urinary coproporphyrins predominate.^{1,2} Urinary porphobilinogen levels (usually raised in variegate porphyria²) were repeatedly normal in both mother and daughter, (characteristic of porphyria cutanea tarda). Elevated faecal coproporphyrins were detected in the mother in 1968 but have not been found since. Faecal protoporphyrin excretion has been repeatedly normal in both mother and daughter (characteristically elevated in variegate porphyria and normal in porphyria cutanea tarda).²

(3) Clinically, despite a long follow-up of 16 years the mother has not shown any acute abdominal crises or neuropsychiatric episodes to suggest a diagnosis of variegate porphyria. Both patients exhibited hepatic siderosis and urinary uroporphyrin excretion decreased on iron removal, a characteristic feature of porphyria cutanea tarda.^{1,2}

I would like to thank Dr Day and his colleagues for drawing attention to the typographical error resulting in the omission of an important line in the published text (see correction in this issue, p 1040). However, I strongly submit that on the evidence of the clinical and biochemical data presented the diagnosis in mother and daughter is porphyria cutanea tarda.

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¹ Harber LC, Bickers DR. In: Malkinson FD, Pearson RW, eds. *Yearbook of dermatology*. Chicago: Yearbook Medical Publications, 1975:9-47.

² Meyer UA. In: Isselbacher KJ, ed. *Principles of internal medicine*. 9th ed. New York: McGraw Hill Inc, 1980:494-500.

Laboratory features of pleural effusions

SIR,—Following your leading article on laboratory features of pleural effusions (20 September, p 763), we would like to present a recent case in which an additional simple laboratory test was of value in the diagnosis of a pleural effusion of uncertain origin.

A 22-year-old Iranian design student was admitted for elective splenectomy. Idiopathic thrombocytopenic purpura had been diagnosed some six years previously, and she had been maintained on corticosteroid therapy. The surgical resection was straightforward with minimal trauma, and the immediate postoperative course was quite satisfactory. Two days postoperatively she developed a tachycardia, fever, leucocytosis, and left pleuritic chest pain, although no haemoptysis and no sputum. Chest radiography showed some collapse and a small effusion at the left base. A ventilation and perfusion lung scan did not show any features of pulmonary embolism, and this was thought to be postpneumonic in origin. There was no clinical improvement with physiotherapy and broad-spectrum antibiotics, and repeated chest x-ray examinations over the next week confirmed the auscultatory findings of a rapidly enlarging left pleural effusion. This was repeatedly aspirated, and 1150 ml of heavily bloodstained fluid was removed. This was sterile on culture, and gas-layer chromatography showed no metabolites of anaerobic infection. Although there had been little clinically to suggest pancreatitis, the amylase content of the pleural fluid was assayed and found to be markedly elevated at 2352 and 2325 U/l. The serum amylase concentration at this time was 310 U/l (normal 70-300 U/l). Following complete aspiration the clinical features returned to normal, and the patient was discharged.

Pleuropulmonary complications are well documented in pancreatitis, with an incidence