was exactly half those I myself had noted. Even more alarming was a letter accompanying the statistics saying: "Increased accuracy is now being achieved and we are rapidly approaching the stage where the figures can be used at local and regional level for managerial and epidemiological purposes."

I believe that those producing these impressive-looking statistics should be responsible for including an estimate of their reliability, based perhaps on a cross-check with more traditional data based on mid-night returns. They should *never* be used for managerial purposes until their reliability is clearly established.—I am, etc.,

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Ampicillin for Sore Throat

SIR,—I apologize to Dr. Joan Stokes (14 July, p. 110) for not making it clear in my letter (9 June, p. 614) that the throat swabs we examined were taken from patients consulting one of us because of sore throat. Approximately one-third of these swabs produced viable *Haemophilus* species in large numbers ($>10^6$). It is accepted that *Haemophilus* species can be pathogenic and it does not seem unduly speculative to conclude that it was so in the majority of these patients. Similar criteria have been used to postulate a causal relationship between microbe and disease in the past.

The results of any survey are valid in the context of the survey, but time and the composition of the sample surveyed can cause large differences between different surveys of the same condition. Thus the carrier rate for *Streptococcus pyogenes* in the 5-15-year age group here in October-December 1967 was 21% and the mean carrier rate over 2½ years in the same age group was 10.2%.¹

I agree that *Str. pyogenes* is the bacterial pathogen most to be feared, notwithstanding the decline in its non-septic complications. It is an organism which is always sensitive to ampicillin and to penicillin. *Haemophilus* species are most frequently sensitive to therapeutic exhibition of ampicillin but relatively insensitive to penicillin. I still suggest that the drug of choice in undiagnosed acute sore throat may be ampicillin.—I am, etc.,

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¹ Ross, P. W., *Health Bulletin*, 1971, 29, 108.

Active Management of Labour

SIR,—The paper by Professor Kiernan O'Driscoll, and others (21 July, p. 135) describing their protocol for the active management of labour paid scant attention to the problem of obstetric pain relief. In their series of 948 primigravidae who proceeded to vaginal delivery, 13 had epidural anaesthesia and the remaining 935 received no analgesia during their

labour except for a maximum of 100 mg of pethidine (57 patients only). If one accepts the figure of 14% for the prevalence of naturally painless labour in primigravidae¹ it appears that over 75% of their patients must have experienced unrelieved pain.

The authors imply that limiting the duration of labour to 12 hours or less in some way counteracts any pain which might occur. This would be a strange attitude if generally adopted in the management of surgical pain and makes one wonder whether these obstetricians believe that labour pain is "real" pain (like that experienced by men) or rather an extension of a female fit of the vapours. At a time when more enlightened attitudes to obstetrics are emphasizing more and more the role of epidural analgesia² it is disturbing to find a fully equipped and staffed obstetrical unit mustering an epidural rate of less than 2% among its primigravid patients.

"Our attitudes to relief of pain in labour remain primitive"² indeed—apparently in the most up-to-date unit.—We are, etc.,

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¹ Potter, N., and Macdonald, R. D., *Lancet*, 1971, 1, 1031.

² *British Medical Journal*, 1973, 2, 323.

SIR,—Professor Kiernan O'Driscoll and his colleagues (21 July, p. 135) report surprisingly large numbers of primigravidae requiring only small doses of pethidine, or no analgesia at all, particularly in view of their statement that labour cannot be conducted without pain. In addition, my experience is that the use of oxytocin to promote labour or the need for a forceps delivery increases the need for analgesia, a point not mentioned by the authors.

As an anaesthetist interested in the relief of pain in labour I would suggest that a more flexible approach is needed than the authors outline to tailor the analgesia to individual patients' requirements. Their remarks on the place of epidural anaesthesia and their pethidine dosage regimen are quite inappropriate to modern obstetrical analgesic management.

I can only conclude that the pain threshold of primigravidae in Dublin is considerably higher than in Sheffield.—I am, etc.,

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Infantile Herpes Zoster

SIR,—Mr. I. K. Lewkonja and Dr. A. A. Jackson (21 July, p. 149) discuss the case of zoster in children and state that "zoster may represent the clinical manifestations of an exogenous reinfection in a partially immune host." There really is no substantial evidence to support this idea.^{1,2}

Because of the rarity of herpes zoster in young children we agree that such well-documented cases are of special interest,

but their case is not unique. We have recently reported other cases of zoster associated with varicella in the mother during pregnancy and reviewed the literature.³ It is perhaps a little misleading of the authors to make no reference whatsoever to previous reports on this subject.—We are, etc.,

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¹ Juel-Jensen, B. E., and MacCallum, F. O., *Herpes Simplex, Varicella and Zoster*, p. 96. London, Heinemann, 1972.

² McKendrick, G. D. W., *Lancet*, 1972, 1, 151.

³ McKendrick, G. D. W., and Raychoudhury, S. C., *Scandinavian Journal of Infectious Diseases*, 1972, 4, 23.

Treatment of Hypothyroidism

SIR,—Dr. David Evered, Professor Reginald Hall, and their colleagues (21 July, p. 131), after careful study of 22 hypothyroid patients, have concluded that the conventional dose of L-thyroxine (T-4) is often excessive, to the possible detriment of the patient. The accuracy of the work of the endocrine department in Professor Hall's charge has led me to send all my sera for thyroid-stimulating hormone estimations from London to Newcastle. Nevertheless, I disagree with the conclusions reached in their paper.

The authors state that after adequate treatment "the serum cholesterol and triglyceride concentrations were very similar to those recorded in a group of control subjects" and, two lines later, "the raised level of serum triglyceride concentration after treatment is unexplained." The two statements are contradictory and the first is incorrect. Their table shows that the serum triglyceride levels for 13 supposedly adequately treated patients averaged 140 mg/100 ml and for a further nine patients averaged 150 mg/100 ml, while the comparable figure for the control group was 103 mg/100 ml.

The fasting triglyceride level is a more accurate reflexion of the abnormal lipid pattern in preclinical hypothyroidism than the serum cholesterol, but we have shown that even the serum cholesterol can be raised years before overt hypothyroidism occurs and is a characteristic finding in preclinical hypothyroidism.^{1,2} Patients with overt myxoedema have probably undergone a process of natural selection in regard to their abnormal lipids. Those with hypercholesterolaemia in the stage of preclinical hypothyroidism are prone to die of coronary artery disease, leaving those with less abnormal lipids to become myxoedematous. It is my experience that patients with myxoedema do not develop coronary disease de novo if given a dose of T-4 sufficient to keep their serum cholesterol at a normal level, but die of coronary disease if their lipids are abnormal. Unfortunately, my follow-up data for patients in the Harrow area are incomplete, since facilities to follow up these patients at Northwick Park Hospital on the closure of Harrow Hospital were denied me. All untraced patients could theoretically have died of coronary disease.