Finally, we, like all those who work with PGI_2, are well aware of the weaknesses of plasma 6-keto-$PGF_{1\alpha}$ measurement as an index of PGI_2 generation. At the moment, however, no other method has been shown to be clearly superior to 6-keto-$PGF_{1\alpha}$ measurement in the study of PGI_2 production in vivo. Many recent papers of Dr Webster and his colleagues on PGI_2, as measured by plasma 6-keto-$PGF_{1\alpha}$ levels, tempt us to believe that he thinks in the same way.

OLAVI YLIKORKALA

University Department of Obstetrics and Gynaecology, Helsinki, Finland

LASSE VIINIKKA

University Department of Clinical Chemistry, Oulu, Finland

1 Preston FE, Whipps S, Jackson CA, French AJ, Wyld PJ, Stoddard CJ. *N Engl J Med* 1981;**304**: 76-9.
2 Shaikh BS, Bott SJ, Demers L. *Prostaglandins in medicine* 1980;**4**:439-47.
3 Hensby CN. In: Lewis PJ, O'Grady J, eds. *Clinical pharmacology of prostacyclin*. New York: Raven Press, 1980:37-43.
4 Friedman LA, Webster J, Hensby CN, Lewis PJ. In: Lewis PJ, O'Grady J, eds. *Clinical pharmacology of prostacyclin*. New York: Raven Press, 1981: 97-101.

Medical aspects of unemployment

SIR,—Professor I M Richardson's letter (16 January, p 193) rightly emphasises that a lot is already known about the medical effects of unemployment.

A study of community health, carried out in Glasgow in 1972, showed that unemployment not due to illness was a major predictor of the prevalence of mental and social symptoms, as well as being significantly associated with the presence of physical symptoms.[1] Quite apart from the personal and social consequences, the hidden costs of unemployment to the health service must be very large indeed. The medical profession may be unable to change the complex economic and social factors involved, but it can continue to draw attention to the health implications of the loss of employment for those who are fit to work.

There is, however, one area of unemployment where doctors do have a direct say and that is on medical unemployment. The evidence is now mounting that we are producing more qualified doctors than this country is able to employ. As the openings abroad are being closed owing to similar difficulties overseas, so the prospects for medical students become dimmer. Sooner or later it seems likely that the medical school intake in the United Kingdom will have to be reduced if we are not to be training doctors for the dole.

D R HANNAY

University Department of General Practice, Woodside Health Centre, Glasgow G20 7LR

1 Hannay DR. *The symptom iceberg: a study of community health*. London: Routledge and Kegan Paul, 1979.

Clinical science and medical art

SIR,—It saddens me that your reviewer Dr Brian Livesley (16 January, p 186) uses my book *Essentials of Clinical Diagnosis in Cardiology* to attack "the teaching of cardiology in general and postgraduate examinations in particular." Sour grapes from a geriatrician will not alter these and neither will facetious remarks about bats, butterflies, and angels. Dr Livesley should have no difficulty in finding compassion in his colleagues, be they teachers or examiners, but quite rightly observes that there is no space for it in a compendium of facts and information.

While one can understand his lack of enthusiasm for minutiae, your reviewer's disdain for eponyms is puzzling in view of his publicised interest in medical history. Surely eponyms provide one of the most effective ways of perpetuating the memory of members of our profession?

R S WINWOOD

Whipps Cross Hospital, London E11 1NR

Little new for audiologists

SIR,—As the series editor of "Studies in Developmental Paediatrics" (MTP Press), I would like to comment on Professor I G Taylor's review (2 January, p 41) entitled "Little new for audiologists" of volume 2 of the series, *The Development of Hearing: its Progress and Problems* by Sybil Yeates.

As is made quite clear both on the dust cover, in the series editor's note, and in Dr Yeates's own preface, these studies are designed for general practitioners, clinical medical officers, paediatricians, health visitors, and others concerned with developmental paediatrics. There has never been any intention of writing a textbook for audiologists. It seems therefore most unfair for Professor Taylor to state that "there is little new for audiologists" when the book is quite obviously not intended for them.

Dr Yeates is quite experienced and skilled enough to write such a book, had she a mind to do so; but that was not her brief. She was asked to write—and I quote from Professor Taylor's review—"to arouse interest in those otherwise unaware of the practice of clinical audiology," and to my mind she has admirably succeeded.

MARGARET POLLAK

Sir Wilfrid Sheldon Children's Centre, Belgrave Hospital for Children, London SW9 0JF

Cold in the jaw

SIR,—The Clinical Curio "Cold in the jaw" should have been called "Pain in the jaw from a cold wind." It can occur in several ways.[1] If the front teeth have lost enamel and dentine because of dental caries or a fracture the cold stimulus is more effective in stimulating the intradental nerves. This can cause pain which is not well localised, especially if several teeth are affected. It starts within a second or so of the application of the stimulus and lasts for a few seconds after the stimulus stops or until the teeth cool. Cold wind can also provoke an attack of idiopathic trigeminal neuralgia and patients occasionally say that they wear a scarf or (in one case) walk backwards into the wind.

A third way is that described in Dr I B Sneddon's note (14 November, p 1314). The cold wind cools the face. When the person returns to a warm room there is vasodilatation, which apparently involves the dental pulps as well, and the increase in blood pressure is sufficient to produce additional neural impulses along the dental nerves. This may feel only like a throbbing in the teeth concerned or may be painful. Such pain is not well localised and seems to spread in that part of the jaw. It passes off after intervals of a few minutes up to about half an hour. Such pain is made worse if disease is present, which may be generating neural impulses below the threshold of perception. This happened to me on a canal boating holiday when I went for a walk in a cold wind. Shortly after returning to the warm cabin pain developed in the right posterior part of my upper jaw. After it had passed off I repeated the walk with a scarf around my face but there was no pain on my returning to the warm cabin. Both tests were repeated with the same results. I also spent time walking in the wind with the scarf on but my mouth open (there was nobody about to see my stupid expression), but this did not provoke pain.

From these tests I decided that I might have a molar with dental caries sufficiently deep to be causing a mild pulpitis which was generating neural impulses below the threshold of perception; the reactive vasodilatation produced additional neural impulses and pain occurred by summation. It also seemed that the dental caries was well hidden from view and well protected from direct cold wind through my open mouth. My dentist found that this was the case and I needed pulp canal therapy. It follows that Dr Sneddon should see his dentist for a check-up—just in case.

J M MUMFORD

Department of Operative Dental Surgery, University of Liverpool School of Dental Surgery, Liverpool L69 3BX

1 Mumford JM. *Toothache and orofacial pain*. Edinburgh: Churchill Livingstone, 1976:118-21, 136-40, 310-4.

Paracetamol-induced acute renal failure in the absence of fulminant liver damage

SIR,—We were interested to see the report by Dr I Cobden and others (2 January, p 21), which confirms our observations that renal failure may complicate paracetamol overdosage in the absence of fulminant hepatic failure.[1 2] Over the period 1969-80 inclusive, 2060 unselected adult patients were admitted to the Edinburgh regional poisoning treatment centre after paracetamol overdosage and 33 (1·6%) developed renal failure, defined as an increase in plasma creatinine concentration to more than 250 μmol/l (2·82 mg/100 ml). Eighteen of these patients developed renal failure in the absence of hepatic failure or encephalopathy. The picture has changed somewhat since specific therapy for paracetamol poisoning was introduced in mid-1973 and renal failure is now virtually confined to severely poisoned patients admitted too late for effective treatment (table). We would take this matter even further than Dr Cobden and his colleagues and point out that renal failure may occasionally complicate paracetamol poisoning

Incidence of renal failure following paracetamol overdosage

Period	Total No of patients	At risk*	No (%) with renal failure
1969-mid-1973	360	57	7 (12·3%)
Mid-1973-1980	1700	267	26 (9·7%)
Mid-1973-1980: treated within 10 h		149	1 (0·7%)
Mid-1973-1980: too late for effective treatment		118	25 (21·2%)

*Plasma paracetamol concentration above a line joining semilogarithmic plots of 200 μg/ml at 4 h and 50 μg/ml at 12 h after ingestion.