In most instances analgesics are prescribed during the healing period. He seems to present the success of his clinical approach as proof that all back pain labelled "slipped disc" is due to spinal compression, presumably of the nerve roots. Unfortunately all too often the diagnosis is applied to cases in which there is no evidence of pain referred from the spinal nerve roots, whether or not there is radiologically demonstrable degeneration of the discs.

Jayson² lists 16 causal headings in the differential diagnosis of back pain, including a large group of patients suffering from non-specific backache. It is particularly from this group that one sees patients wrongly labelled "slipped disc," since many patients with radiologically demonstrable degeneration of discs do not have backache³ and many have backache due to causes other than disc lesions and are labelled "slipped disc" for want of a better term. There is, so far as I am aware, no proof that "compression is the logical cause of most back pain."

The term "spinal compression disorder" may correctly be applied to patients suffering from compression of the nerve roots as a result of disc degeneration with or without disc prolapse.⁴ In persons over the age of 50 the disc pulp has disappeared and so cannot prolapse or "slip." Where there is no evidence of motor and/or sensory disturbance resulting from spinal compression disorder I suggest that there may be pain due to compression of nerve fibrils in a lesion of one of the structures or layers of the back away from the discs and nerve roots. This compression involves trapped or inflamed nerve fibrils in oedematous fibrous, muscular, osseous, cartilaginous, or other connective tissue in the various layers of the back, such as has been found in other parts of the body.⁵⁻⁹ Rather than ask the patient to wait patiently during the long period of natural healing it is my practice to attempt to search out the site of pain by palpation. If this can be done it is a simple matter to treat the inflamed tissue with an injection of corticosteroid mixed with local anaesthetic.¹⁰

It is my experience that spinal compression syndrome is not as common as "non-specific back pain" and I suggest that every effort be made to pinpoint the diagnosis before labelling back pain either "slipped disc" or "spinal compression disorder."¹¹

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¹ Doran, D M L, and Newell, D J, *British Medical Journal*, 1975, **2**, 161.

² Jayson, M V, *Practitioner*, 1970, **205**, 615.

³ Gyntherberg, F, *British Medical Bulletin*, 1965, **21**, 30.

⁴ Ghormley, R K, *Proceedings of the Mayo Clinic*, 1951, **26**, 457.

⁵ Glyn, J H, and Newton, D R L, *Annals of Physical Medicine*, 1957-8, **4**, 176.

⁶ Burgess, G W, and Stapleton, J G, *Applied Therapeutics*, 1967, **9**, 155.

⁷ Roy, S, and Oldham, R, *Lancet*, 1976, **1**, 1322.

⁸ Goodwill, C J, *Modern Medicine*, 1977, **22**, suppl, p 41.

⁹ Yates, D A H, *British Medical Journal*, 1977, **1**, 495.

¹⁰ Silver, M Y, *Applied Therapeutics*, 1963, **5**, 39.

¹¹ Bourne, I H J, *British Medical Journal*, 1977, **2**, 1158.

Possible environmental hazards of gas cooking

SIR,—Melia *et al*¹ reported the results of an epidemiological study which suggested a higher prevalence of "respiratory symptoms and disease" among 6-11-year-old schoolchildren in houses where gas is used for cooking than in those in which electricity is used. The proportion of children in the more severe categories was, as expected, related to

age, sex, and social class, but after allowing for these factors and for urban and rural living differences associated with cooking method still appeared significant, although the authors stated that they had no direct knowledge of parental smoking habits.

Clearly more work is needed to confirm and elucidate these findings, but if there is a relationship between gas cooking and a tendency to respiratory disease in schoolchildren, then it seems important to determine not only the average levels of gaseous emission reached in individual homes but also the peak values during periods of greatest cooker use. In a preliminary study, we have measured the oxides of nitrogen and the carbon monoxide levels in the kitchens of 86 employees of British Gas during the summer and repeated the measurements in 20 of the homes during the winter. Daily average levels of oxides of nitrogen and of carbon monoxide were consistently higher in the winter than in the summer, as were most of the peak values recorded at the time of greatest cooker use. This, together with the range of values found between different households, suggests that kitchen ventilation as well as size may play a major role in determining indoor concentration, as indeed was suggested by Melia *et al*.

Throughout their work we have been in contact with the St Thomas's Hospital team and this is still the case. In addition to their work we are also aware of the epidemiological study undertaken in America during the early 70s by Mitchell *et al* at the Battelle Institute and the Ohio State University, Columbus, which was reported at the EPA/WHO symposium in Paris in June 1974.² Their final report stated that there was "no significant difference in reported respiratory illness between members of households cooking with gas and those cooking with electricity."

Our preliminary studies have allowed us to perfect and validate instruments sensitive enough for use in the field and we are now pursuing wider and more detailed household studies directed towards elucidating the various factors influencing indoor air quality.

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¹ Melia, R J W, *et al*, *British Medical Journal*, 1977, **2**, 149.

² Mitchell, R I, *et al*, in *Recent Advances in the Assessment of the Health Effects of Environmental Pollution Symposium Proceedings*, vol 2, p 47; Luxembourg, Commission of European Communities, 1975.

SIR,—Your leading article (18 March, p 671) "What kind of cot death?" suggests that "a large part of the explanation for the low incidence of cot death in countries such as Sweden may be related to housing, population density, and other special factors." For some years I have been interested in the widespread potential hazard of nitrous fumes, which can cause not only acute pulmonary oedema and death but also chronic lung disease.¹ I have been suspicious that nitrous fumes may be a factor in cot deaths. My suspicions were strengthened by the recent observations of Melia *et al*,² who undertook a four-year longitudinal study of the prevalence of respiratory symptoms and disease in schoolchildren. In 5758 schoolchildren aged 6-11 years they found that boys and girls from

homes in which gas was used for cooking had "more cough, 'colds going to the chest,' and bronchitis than children from homes where electricity was used." They concluded that elevated levels of oxides of nitrogen arising from the combustion of gas might be the cause of increased respiratory illness.

I suggest that the lower postneonatal mortality rates in Sweden compared with the UK³ and Scotland⁴ may relate to the use of electricity for cooking in Sweden and gas in the UK. Furthermore, there has been a major switch from synthetic to natural gas in the UK over the past decade. One would like more information on the emission of oxides of nitrogen from these sources.^{5 6} In your leader you reported that "during the 1960s, in sharp contrast to the previous two decades, the postneonatal mortality rate in England and Wales fell only slowly. In the '70s there has already been a 25% reduction, most of it since 1974. In Scotland the improvement has been even more striking: a 40% reduction between 1969 and 1976." In Stoke-on-Trent the replacement of coal gas with natural gas started in 1969 and was completed in 1973. These dates coincide with the reduction of cot deaths. The high incidence of cot deaths during the winter months also seems to support the possibility that fumes from gas fires may be one of the factors associated with cot deaths. Presumably this hypothesis could be investigated.

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¹ Kennedy, M C S, *Annals of Occupational Hygiene*, 1972, **15**, 285.

² Melia, R J W, *et al*, *British Medical Journal*, 1977, **2**, 149.

³ Office of Population Censuses and Surveys, unpublished information.

⁴ Registrar General for Scotland, *Annual Report Part 1*, Edinburgh, HMSO, 1976.

⁵ Derwent, R G, and Stewart, H N M, *Atmospheric Environment*, 1973, **7**, 385.

⁶ Schwarzbach, E, *VDI Berichte*, 1975, **247**, 16.

Medical course at Nottingham

SIR,—I crave the indulgence of your columns to correct an error in my book recently published, *The Quest for Excellence in Medical Education*. It is there stated that the medical course in Nottingham, including the honours year, lasts six years. In fact it lasts only five. Nottingham was the first university to achieve this. I was aware that the university's proposals were not at first acceptable to the General Medical Council and I had failed to appreciate that agreement had been reached. The fault is nevertheless entirely mine and I apologise to the university and to those who read my book for an error which will be corrected in future copies.

GEORGE PICKERING

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*.*This book is referred to in a leading article at p 519.—ED, *BMJ*.

Radiotherapy for painful heel syndrome

SIR,—Dr B S Mantell (8 July, p 90) found that 11 of 17 patients with stubborn plantar fasciitis improved with low-dose radiotherapy to the painful heel. I wonder how many in fact had ankylosing spondylitis or some other form of seronegative spondylarthritis to which

their plantar fasciitis was secondary? It is well known that the peripheral manifestations of ankylosing spondylitis often respond well to deep x ray and, perusing the table of clinical details of the 17 patients, it appears that at least six of the responders did have an underlying seronegative spondylarthritis of some variety.

It would therefore be wrong to conclude from this report that when "simple" plantar fasciitis fails to improve with conventional treatment it is likely to respond to radiotherapy. A further study of "simple" plantar fasciitis in which seronegative spondylarthritis has been excluded is clearly required.

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Spurious polycythaemia in a neonate?

SIR,—There have recently been published a short report by Dr L E Ramsay (13 May, p 1251) and a letter from Drs B W S Robinson and D Corless (22 July, p 280) referring to Gaisböck's syndrome (spurious or "stress" polycythaemia or pseudopolycythaemia). We have recently seen a neonate who may have been suffering from the same syndrome and I have as yet been unable to find any references to this syndrome in neonates.

A 5-day-old full-term child was transferred to our unit with a diagnosis of Gram-negative neonatal meningitis made on day 3. The initial haemoglobin concentration was reported as 18 g/dl; when repeated on day 5 by heel prick it was 21 g/dl. It was thought that the child might be dehydrated; however her serum electrolyte values were normal and her fluid intake by intravenous infusion was adequate. Alternatively the heel prick might have given a spurious result; the haemoglobin estimation was repeated by venepuncture and the result was 20.9 g/dl. There was no evidence of renal disease or cardiac disease throughout her illness and as her meningitis resolved her haemoglobin level returned to 17-18 g/dl over three weeks.

I wonder whether this syndrome has been described in neonates and whether any other readers have come across the situation.

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Antifibrinolytic agents for ruptured intracranial aneurysms

SIR,—In contrast to the study by Mr R S Maurice-Williams (15 April, p 945), which suggests a beneficial effect of tranexamic acid after rupture of an intracranial aneurysm, we found in a double-blind controlled clinical trial no difference in either mortality or rebleeding rates.¹

Mr Maurice-Williams's study lacked a double-blind procedure and the patients investigated were restricted to those in Botterell's grades I and II. The actual numbers of patients in a state of a clear or clouded consciousness in the two groups are not given, but theoretically this can have biased the results. Our own trial confirmed that the state of consciousness after a subarachnoid bleeding appeared to be the most important factor with regard to mortality.

The author's remark in the discussion that "the same striking improvement" with regard to the use of antifibrinolytic agents has been described in other controlled trials is hardly substantiated by the literature cited. The control group in Sengupta's series²—not double-blind—contained twice as many severely ill patients as the treatment group. The author's reference (No 10) to a personal communication cannot be regarded as additional evidence. Nibbelink's study³ had a follow-up period of only 14 days. Gibbs⁴ compared two groups of patients which were not comparable—namely, an operated group versus a group which apparently could not tolerate operation and subsequently was treated with tranexamic acid.

We do not think that this report adds valuable information to substantiate the assertion that tranexamic acid is of any use in the treatment of ruptured intracranial aneurysms.

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- ¹ van Rossum, J, *et al*, *Annals of Neurology*, 1977, 2, 238.
- ² Sengupta, R P, So, S C, and Villarejo-Ortega, F J, *Journal of Neurosurgery*, 1976, 44, 479.
- ³ Nibbelink, D W, Torner, J C, and Henderson, W G, *Stroke*, 1975, 6, 622.
- ⁴ Gibbs, J R, and Corkill, A G L, *Postgraduate Medical Journal*, 1971, 47, 199.

Autoimmunity in juvenile diabetics and their families

SIR,—Dr G F Bottazzo and his colleagues (15 July, p 165) re-emphasise the importance of both pancreatic islet cell and other antibodies in type I, "juvenile," diabetes. While these may be of importance in the aetiology of the disease, the evidence for their importance in the microangiopathic complications of diabetes is not proved by the evidence provided and their statements in the summary are misleading.

Diabetic microangiopathy, of which retinopathy is a manifestation, occurs in both type I and type II diabetes and there is no evidence to suggest that it is commoner in type I disease. It is a manifestation of the duration and also the degree of control of the metabolic abnormality.¹ After 15 years' duration up to 82% are expected to have retinopathy² and after 20 years up to 100%.³ If autoantibodies were of significance angiopathy would be expected to occur earlier. No patient in this series with disease duration under 10 years had such findings. The seven patients in table II (not 10 as suggested in the summary) with microangiopathy had all had diabetes for 12 years or more. The three who had both retinopathy and nephropathy had had it for over 20 years. In this small study of eight patients with diabetes for 12 years or more seven (88%) had retinopathy. This is not excessive and in view of the small numbers no conclusions can be drawn.

Besides duration of diabetes, diabetic control is of importance in the development of retinopathy, as shown in randomised controlled studies in man⁴ and animals⁵ and in the prospective studies by Pirart.⁶ Members of the same family are likely to have not only similar antibodies but similar eating habits, the same physician, and similar type of management. Control of the disease therefore is likely to be similar. By giving autoimmunity an unwarranted importance in the develop-

ment of the long-term sequelae of diabetes the authors suggest that, since these are "inherited," management of diabetes is of little importance. This is a view I wish to oppose strongly.

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- ¹ Colwell, A R, in *Small Blood Vessel Involvement in Diabetes Mellitus*, ed M D Siperstein, A R Colwell, and K Meyer, p 253. Washington, DC, American Institute of Biological Sciences, 1964.
- ² Larsson, Y, Sterky, G, and Christianson, G, *Acta Paediatrica*, 1962, 51, suppl 130, p 1.
- ³ Dolger, H, *Journal of the American Medical Association*, 1947, 134, 1289.
- ⁴ Job, D, *et al*, *Diabetes*, 1976, 25, 463.
- ⁵ Engerman, R, Bloodworth, J N B, jun, and Nelson, S, *Diabetes*, 1977, 26, 700.
- ⁶ Pirart, J, *Diabète et Métabolisme*, 1978, 3, 97.

Lymphatic fistula: a complication of arterial surgery

SIR,—Mr R J Croft (15 July, p 205) was using the wrong dye. The Patent Blue V which is distributed by May and Baker Ltd for Laboratoire André Guérin is a 2.5% solution of a calcium salt of sulpham blue with a molecular weight of 1159 and a colour index of 42051. It differs from Patent Blue V (Sigma), which is an 11% solution, and Disulphine Blue (Imperial Chemical Industries Ltd), which is a 6.2% solution of the sodium salt of sulpham blue which has a molecular weight of 567 and a colour index of 42045. These dyes, injected into the foot, will colour the groin lymphatics and nodes.

My colleagues and I have used this technique in 38 patients undergoing femoral profundaplasty.¹ Groin lymph structures showed up well and were either avoided, ligated, or sealed with surgical diathermy. No lymph cyst or fistula developed.

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- ¹ Leaper, D J, Evans, M, and Pollock, A V, *British Journal of Surgery*. In press.

Surgery for intracranial aneurysm in the elderly

SIR,—I read with interest the paper by Mr R P Sengupta and others (22 July, p 246) on surgery for intracranial aneurysm in the elderly, but I am puzzled by the authors' conclusions that surgery is justifiable in this age group. Their data would appear to confirm the feelings of others¹ that the results of such surgery are unrewarding.

Eight patients out of 32 (or of 25 if only those over 60 are counted) died or were left seriously disabled after surgery and a further three were left partially disabled. Furthermore, we are told nothing of those patients who died from rebleeding before surgery would have been carried out and who must surely be added to the overall surgical picture. Nor are we told the interval from the haemorrhage until operation, except in the cases of those patients who fared badly. Judging from the figures given for the latter it would seem that in many cases operation was performed late, after the period of maximum rebleeding risk had passed. A person who survives three weeks in good condition without rebleeding has only a 5-10% chance of dying from rebleeding from then on² and it is against this