Size of tumour and operation performed in relation to length of history, 1979

	Short* history	Intermediate history	Long† history
No of inoperable tumours	1	2	7
Average tumour diameter (cm)	1.6	2.6	3.1
No of mastectomies	19	29	8
No of wide local excisions	16	22	1
Total No of patients	36	53	16

*Short history $\leq$ one month.

†Long history $\geq$ one year.

radiotherapy. The selection of patients for conservative surgery is on the basis of the relative volume of the tumour to the breast and on its position. Patients with central tumours still undergo mastectomy but patients with relatively small medial or lateral tumours have wide local excision.

If wide local excision and postoperative radiotherapy can control local disease as effectively as mastectomy, then our message to the public is not that shorter delay necessarily means longer survival but that shorter delay means less radical surgery.

We thank Drs J C Gazet, H T Ford, and T J Powles of the St George's Hospital combined breast clinic at the Royal Marsden Hospital for permission to review their patients.

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¹ Tubiana M, Chauvel P, Renaud A, Malaise EP. *Bull Cancer* 1975;62:341-58.

Screening for the small-for-dates fetus

SIR,—We thank Dr R H Klipstein (21 June, p 1534) for his interest in our two-stage ultrasonic examination schedule to screen for intrauterine growth retardation, which, using the crown-rump length $\times$ trunk area index, detected 94% of small-for-dates fetuses in almost 500 singleton pregnancies (17 May, p 1203) and which we are now subjecting to a prospective controlled trial. However, we cannot agree that the efficacy of a screening procedure can be equated solely with its predictive value (defined as the ratio of true-positive to false-positive diagnoses). Rather we believe that its efficacy must also take into account sensitivity (defined as the ratio of true-negative to false-negative results) and clinical usefulness.

We made the point that false-negative results (that is, undetected growth retardation) are of greater practical significance than false-positives and we therefore viewed the crown-rump length $\times$ trunk area index (false-negative rate 6%, false-positive rate 12%) as more useful than the crown-rump length $\times$ trunk circumference index (false-negative rate 11%, false-positive rate 9%). Many false-positives can be identified as such by further detailed ultrasonic examination following a screening diagnosis of growth retardation. Indeed, further management (including intervention) would depend, as we stated, on further intensive biophysical and biochemical monitoring and also, of course, on continued clinical surveillance. It is also

relevant that half our false-positives related to babies with birth weights below the 10th percentile (sometimes taken as the cut-off point defining growth retardation) but above the 5th percentile (our cut-off); it seems to us no great harm that these fetuses in particular should be subjected to investigation of their subsequent growth and wellbeing.

Thus we can reassure Dr Klipstein that there need be no undue concern that our chosen screening parameter will necessarily lead to more unnecessary intervention than would the use of the crown-rump length $\times$ trunk circumference index.

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Family trends in psychotropic and antibiotic prescribing in general practice

SIR,—The paper by Drs J G R Howie and A R Bigg (22 March, p 836) is a further useful addition to the relatively small amount of information about prescribing in general practice. It is of interest, however, that the authors did not consider the possibility that the children of anxious mothers actually do have more respiratory diseases requiring antibiotics than the children of mothers not so anxious.

There is also little comment on the finding that anxious mothers most often need psychotropic drugs while the children are young or while they are approaching puberty. Such a finding is perhaps not unexpected and might also tie in with the finding that mothers with more children are less likely to be high users of psychotropic drugs. These findings would reinforce the author's suggestions that family structure and dynamics may well be an important influence in prescribing.

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Deaths from non-accidental injury in children

SIR,—I would concur with Dr J A Baldwin's estimate (21 June, p 1533) that the number of children who die annually from non-accidental injury is considerably higher than the Registrar General's figure for "deaths due to homicide and injury purposely inflicted by other persons," as was suggested by Dr K T Farn (3 May, p 1145).

There are three aspects to deaths from this cause which merit consideration. Firstly, non-accidental injury occurs almost exclusively in the home in the absence of independent witnesses. Secondly, the injuries sustained may not be the immediate cause of death. Thirdly, some deaths due to non-accidental injury get misdiagnosed.

When a child dies in suspicious circumstances, the parents deny deliberately injuring it, and the medical evidence cannot determine conclusively that the injuries had been purposely inflicted the coroner has no option but to record an "open verdict." With nine of the 21 dead children in the sample of battered children reported by Smith and Hanson, the coroner had reached this verdict.¹ These "open

verdict" deaths are recorded by the Registrar General under the category "injury undetermined whether accidentally or purposely inflicted." In 1977 the number of child deaths recorded in this category² was 30—a figure which when added to the 78 homicides in that year exceeds Dr H B Valman's estimate (15 March, p 775).

There are also the cases where the inflicted injuries do not cause death in themselves but so weaken the child's resistance to infections as to lead to death from "natural causes." In 1975 nine dead children were referred to the Registers of Suspected Non-Accidental Injury maintained by NSPCC Special Units³ in five different parts of the country. Two of these children were recorded as having died of bronchopneumonia, one with 106 bruises of different ages on her body; both mothers were prosecuted for assault. Two of the other dead children (siblings) had originally been diagnosed as cases of cot death. It was not until their healthy 4½-year-old brother died in the same manner that their deaths were re-examined. Half of Johnson's 17 cases of battered babies, originally thought to have died of natural causes, had been diagnosed as cases of cot death.⁴ It would seem that cases where the attending doctor misdiagnoses the cause of death are not as uncommon as one might expect.

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¹ Smith SM, Hanson R. *Br Med J* 1974;iii:666-70.

² Office of Population Censuses and Surveys. *Mortality statistics: childhood and maternity 1977*. London: HMSO, 1979.

³ Creighton SJ, Owtram PJ. *Child victims of physical abuse*. London: National Society for Prevention of Cruelty to Children, 1977.

⁴ Johnson HRM. *Med Sci Law* 1969; April: 102-6.

Dorsal column stimulation in multiple sclerosis

SIR,—Your leading article (31 May, p 1287) presents a very reasonable assessment of the current state of affairs with regard to dorsal column stimulation in multiple sclerosis. The statement, however, that there is "total lack of any data showing how the treatment works" is too sweeping. Data exist but hard facts do not. Any proposition needs to explain why dorsal column stimulation will help "central" pain and bladder function and why the effect on micturition is insidious, usually maximal at six to seven days, wearing off equally slowly.¹ This favours a more hormonal than an electrical effect, although both may be responsible.

The fragmentary data relating to mechanism are: (1) Dorsal column stimulation elevates adrenaline, noradrenaline, and dopamine in plasma and noradrenaline in cerebrospinal fluid.² (2) There is no change in CSF homovanillic acid or 5-hydroxyindole-acetic acid.³ (3) Dorsal column stimulation reduces urethral sphincter tone,⁴ which suggests α -sympatholytic or parasympathomimetic action. (4) Dorsal column stimulation has no effect on or possibly depresses CSF met-enkephalin levels. It is possibly naloxone reversible.¹ (5) It has evoked potentials⁵ and H responses.⁶ (6) It has improved leg blood flow.⁷ Observations 1-6 are based on very small numbers. The possibility that a neuropeptide or prostaglandin is involved needs close scrutiny.

The overwhelming problem in multiple sclerosis is how to disentangle possible benefits

of dorsal column stimulation from the random variability of the disease, placebo effects, and suggestibility. This can in part be overcome by making multiple observations so that the variance before treatment is defined, which should take care of the disease fluctuations. Placebo and suggestibility effects tend to wear off after a few weeks' therapy and are less of a problem when studying autonomic function. Automated urinary flow studies (for example, by Disa Mictograph) are probably the single most useful non-invasive test by which the effects of dorsal column stimulation can be measured. Finally, it is suggested that anyone embarking on trials of dorsal column stimulation should discuss it fully with a qualified medical statistician so that the results can be properly analysed.

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¹ Hawkes CH, Fawcett D, Cook ED, Emson PC, Paul EA, Bowcock SA. *Appl Neurophysiol* (in press).

² Levin BE, Hubschmann OR. *Neurology* 1980;30:65-71.

³ Cook A, Nidzgorski F, Hurwic M, Winsberg B, Klutch A. *Lancet* 1978;i:46.

⁴ Hawkes CH, Wyke M, Desmond A, Bultitude MI, Kanegaonkar GS. *Br Med J* 1980;280:889-91.

⁵ Sedgwick EM, Illis LS, Tallis RC, et al. *J Neurol Neurosurg Psychiatry* 1980;43:15-24.

⁶ Illis LS, Sedgwick EM, Oygar AE, Sabbahi Awadalla MA. *Lancet* 1976;i:1383-6.

⁷ Tallis R. *Appl Neurophysiol* (in press).

Surgical treatment for prolapsed lumbar disc

SIR,—I read with nostalgic interest the letter by Dr Laurence Wood (14 June, p 1454), who took issue with your leading article (22 March, p 814) "Surgical treatment of prolapsed lumbar discs." In latter years of active orthopaedic practice I became convinced, like Dr Wood, that a lumbar flexion rotational strain with minor subluxation of a low lumbar joint and not just some derangement of the intervertebral disc was the common cause of the ordinary "acute lumbar pain, with or without sciatica." Treatment by manipulative means would usually produce relief of the patient's symptoms, without some prolapse of an intervertebral disc always being imagined as the cause.

When I commenced my orthopaedic career 54 years ago, a common diagnosis of the acute back was subluxation of a sacroiliac joint. Treatment, either with or without an anaesthetic, was to lie the patient on his side on the operating table, when the surgeon forcibly struck the iliac crest with the point of his elbow, thus not only jarring the sacroiliac joint but in fact producing a rotational strain of the lower lumbar joints. This usually resulted no doubt in reduction of the slight lumbar joint subluxation. Subsequently we went through the phase of general manipulation of the lumbar spine under general anaesthesia, followed by physiotherapy. Then came the vogue of putting the lumbar spine in a plaster jacket for a month or so, allowing the patient to walk about in it, which no doubt jarred the spine sufficiently to relieve the minor lower lumbar joint subluxation. Then followed the phase of the operative exploration of the lower lumbar joints—laminectomy. Lucky the surgeon if, on exposing the lumbar intervertebral joint, he found a loose piece of disc lying alongside the dural sac: removal of this cured the patient. Unlucky the patient whose surgeon, feeling a small lump protruding under the posterior spinal ligament, opened it and scraped out with a spoon that disc, degenerated or not: that patient might have his sciatica relieved, but he was left with intractable backache. Spinal traction did not produce

any dramatic long-lasting cures of itself, unless combined with spinal rotation exercises to reduce the subluxation.

To illustrate my views, backing up Dr Wood's surprise at his results of manipulation with subluxation in mind, may I quote the following incident? Before retirement from hospital I had a young woman patient being treated unsuccessfully for sciatica by bed rest and traction. I advised and she agreed to laminectomy, and at that time when in doubt I sent my sciatica patients to a neurological hospital for myelography. On the day that I was due to operate I visited this patient, and after looking at a negative lumbar myelogram I asked her how she felt and if I was to proceed that afternoon with my planned laminectomy. To my surprise she said that when being forcibly flexed on the x-ray table she had experienced a short and sudden pain in her lower lumbar spine, and now all sciatica had gone. Subsequent outpatient follow-up confirmed that surely a lumbar joint subluxation had been reduced.

Latterly I found that if I could persuade a patient who had taken to his bed with acute backache and sciatica to get on to the floor on his hands and knees and crawl round the room, first in one direction and then in the other, and on a third round of the room to arch and re-arch his back when crawling and then return to bed, repeating these manoeuvres the next day if necessary, in a day or so all symptoms would be relieved.

I myself proved this, after retirement and in my seventies, when I had to lift up from the floor my late wife who had fainted. I felt a click and sudden pain in the back. I took a couple of Equagesic tablets and performed the crawling therapy. Next day I felt as if I had been kicked in the lower back, but full recovery followed within a few days. I suggest Dr Wood tries this therapy on his low back pain patients.

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Infected hip prostheses

SIR,—Your leading article (24 May, p 1241) traces the possible treatment of an infected hip prosthesis from a few days after the original operation, through excision of infected soft tissues and possible replacement of a loose prosthesis using antibiotic-loaded acrylic cement to, finally, a Girdlestone-type procedure. You mention that even this operation may not overcome infection. At all these possible stages of the infective process it is important that the correct antibiotic is given and works. We have found in this hospital that the antibiotic action is improved by hyperbaric oxygen therapy given at the same time. It is known that either aerobic bacteria are killed or their division is slowed down by hyperbaric oxygen.¹ We have now treated 10 such cases in 11 years, as follows:

(1) Four cases—seven days, two years, three years, and six years following hip replacement. These have all healed following antibiotics and hyperbaric oxygen, the last two needing a superficial operation for the removal of soft tissue. (2) Two cases of hip replacement with sinus discharging pus for some years. Sepsis cleared up in these cases but the prostheses were loose and needed replacement. (3) One case of hip replacement, infected and with discharging sinus for six months. Antibiotics and hyperbaric oxygen were given until the infection was cleared, a Girdlestone type operation was performed, and antibiotics and hyperbaric oxygen were continued until healing was complete. (4) Three cases of Girdlestone type operation for an infected prosthesis with obvious infection 14 and 16

days after operation and one with an infected sinus for two years. These have all healed and remained so.

It is not suggested that hyperbaric oxygen takes the place of standard treatment. It does, however, make success more probable.

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¹ Watt J. *Proc R Soc Med* 1971;64:880.

Idiopathic carpal tunnel syndrome

SIR,—I should like to comment on the interesting study by Dr S Dekel and others on the relationship between the idiopathic carpal tunnel syndrome and carpal stenosis measured by computed tomography (31 May, p 1297).

The authors have not related the area of the carpal tunnels measured by computed tomography to a somatic parameter such as height, weight, or possibly the area of the carpal bones on computed tomography. Consequently, one is left not knowing whether their female patients with the idiopathic carpal tunnel syndrome were simply a group of small women or were women with small carpal tunnels. This failure to relate the carpal tunnel area to another parameter has led the authors to suggest that females may develop the carpal tunnel more often than men because they have smaller carpal tunnels. However, women in general are smaller than men. Women do not have spinal cord compression more often than men because they have narrower spinal canals.

It would be interesting to know whether standard window widths and levels were used in all the computed tomography examinations, and whether the carpal tunnel areas were estimated without knowledge of the patient's symptoms in order to eliminate observer bias.

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*.*We sent this letter to Dr Dekel, whose reply is printed below.—Ed, *BMJ*.

SIR,—I would like to make the following points in reply to Dr Witcombe.

Firstly, I do not think that somatic measurements such as height and weight of the patients versus the controls are relevant as we measured the areas of the carpal canal and found them statistically smaller than those of the female controls. However, we did measure the cross-sectional area of the proximal carpal bones in all three groups examined and there was no statistically significant difference between female patients and female controls (mean for female patients $721.27 \pm 24.85 \text{ mm}^2$, 42 hands; for female controls $757.04 \pm 26.84 \text{ mm}^2$, 39 hands, $p > 0.1$). Thus the ratio between the carpal canal versus the carpal bone in female patients was greater than that in female controls (3.94 v 3.62).

Secondly, female patients were found to have significantly smaller carpal canals than male controls. The difference was found to be around 25%. Thus minimal oedema of the content of the carpal canal or a space-occupying lesion will increase the pressure more markedly in a small carpal canal than a large one. This may explain the female propensity for this condition. A positive correlation was found between the cross-sectional area of the