who had it postnatally; we could find no references where this has been investigated previously.

The authors' analysis of risk factors highlights some common misconceptions: a quarter of all the women with eclampsia were parous, 18% of all the women with eclampsia were parous with no history of pre-eclampsia or eclampsia in previous pregnancies, and teenagers were at three times greater risk than older women. Overall, this paper is a sobering reminder of the vigilance required if we are to reduce further the morbidity and mortality from this relatively common condition.

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1 Douglas KA, Redman CWG. Eclampsia in the United Kingdom. *BMJ* 1994;**309**:1395-400. (26 November.)
2 Department of Health, Welsh Office, Scottish Home and Health Department, Department of Health and Social Services Northern Ireland. *Report on confidential enquiries into maternal deaths in the United Kingdom 1988-1990.* London: HMSO, 1994.

Authors' reply

EDITOR,—D G Daniel and R H Golding ask for more information on the women with eclampsia. Altogether 198 women were reported but excluded (60 after talking to consultants and 138 after the case note review) because they did not meet the criteria for the study. Seventy three of these had pre-eclampsia but no seizure; 56 had uncomplicated epilepsy; 40 had simple faints, hypoglycaemic fits, or pseudoseizures; and 29 had seizures without an apparent cause but did not have signs of pre-eclampsia.

Eighty nine women had a fit while under community based care, and for 64 of these we had access to a complete record of the care. Of the 64 women, six had hypertension alone, 11 had proteinuria alone, and five had hypertension and proteinuria documented before the onset of seizures. Twenty six of the 64 had been seen in the week preceding the fit.

The 27 women with multiple pregnancy were significantly less likely to have antepartum seizures than those with singleton pregnancies (relative risk (95% confidence interval) 0·18 (0·05 to 0·69)), but there were no significant differences between the two groups in terms of the presence of prodromal signs or symptoms, the gestation at onset of seizures, the number of seizures, or the ensuing maternal morbidity or mortality.

National data on number of previous stillbirths and live births do not exist, so that accurate calculation of rate of eclampsia in primiparous compared with multiparous women cannot be calculated accurately. Seventy one (99%) of the teenagers were primiparous compared with 215 (69%) of the women aged 20 or more. Part of the increased risk associated with teenage pregnancy may therefore be due to primiparity.

Whether or not the multigravidas had new partners was not determined, but 26 (27%) had a history of pre-eclampsia and one (1%) had a history of eclampsia. Multiparous women were significantly more likely to report prodromal symptoms than primiparous women (relative risk 1·31 (1·11 to 1·53)), but there were no significant differences in outcome measures.

Daniel and Golding's analysis of antenatal care assumes that eclampsia was uniformly spread through the last 112 days of pregnancy. Seizures were more common in the last four weeks of pregnancy, when it is routine to see women weekly; this helps to explain the high proportion of women seen within seven days of their seizure. Other women may have been brought back early or sought medical advice about symptoms. Our data are not detailed enough to supply this information.

There were no significant differences in the presence of prodromal symptoms (relative risk 1·00 (0·75 to 1·33)) or signs (1·08 (1·0 to 1·17)) between women who had postpartum eclampsia after caesarean section or vaginal delivery. We did not record whether ergometrine was used in the third stage of labour.

In answer to the questions raised by Edward O'Donnell and David Somerset, women with antepartum eclampsia had significantly higher systolic and diastolic blood pressures within one hour of the onset of seizures than those with postpartum seizures (table). This is consistent with the overall picture of antepartum eclampsia being a more severe condition.

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Comparison of blood pressures in women with antepartum onset of seizures and women with intrapartum and postpartum onsets. Figures are means (SD)

	Antepartum onset (n=147)	Intrapartum and postpartum onsets (n=236)	Significance*
Last systolic pressure (taken within one hour of onset of seizures) (mm Hg)	166 (26·2)	156 (21·2)	5·67, P=0·017
Last diastolic pressure (taken within one hour of onset of seizures) (mm Hg)	105 (14·2)	94 (14·0)	18·05, P<0·001
Maximum recorded systolic pressure (mmHg)	185 (26·0)	179 (20·7)	4·51, P=0·034
Maximum recorded diastolic pressure (mm Hg)	120 (13·0)	113 (11·6)	26·49, P<0·001

*Kruskal-Wallis *H* test (1 df).

Treating leg cramp

Naftidrofuryl is a safe and effective alternative

EDITOR,—As part of their meta-analysis of the efficacy of quinine in treating leg cramp Malcolm Man-Son-Hing and George Wells review the pharmacological alternatives to quinine for this painful condition.[1] They fail to mention naftidrofuryl. In a double blind placebo controlled crossover study of cramp at rest we showed naftidrofuryl to produce both a significant reduction in the frequency of cramp and an increase in the number of days free of cramp.[2] Man-Son-Hing and Wells point out the possible serious side effects of quinine, and we suggest that naftidrofuryl should be regarded as an alternative, potentially less hazardous treatment for cramp at rest.

We agree with Man-Son-Hing and Wells that the condition is extremely common; indeed, we have shown a prevalence of 37% in the elderly population overall.[3] Such a high prevalence further emphasises the need to use treatment with a low profile of side effects.

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1 Man-Son-Hing M, Wells G. Meta-analysis of efficacy of quinine for treatment of nocturnal leg cramps in elderly people. *BMJ* 1995;**310**:13-7. (7 January.)
2 Young JB, Connolly MJ. Naftidrofuryl treatment for rest cramp. *Postgrad Med J* 1993;**69**:624-6.
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The trial of naftidrofuryl[2] received support from Lipha Pharmaceuticals, which provided the active drug and placebos, randomisation envelopes, and trial booklets to monitor patients recruited to the trial.

Study quoted had flawed design

EDITOR,—In their meta-analysis of the efficacy of quinine for night cramps Malcolm Man-Son-Hing and George Wells analysed six trials that met strict criteria for design.[1] Included in these six trials is one carried out by one of us (NRD) in 1991 on patients from his practice and a practice in Southampton.[2] It is surprising that the authors included the data from this trial, since it was stated clearly that the results showed that the design of the trial was invalid. As the table shows, there was a clear carryover effect: patients who received quinine first, followed by placebo, experienced more nights with cramp in the placebo period than did those who received the placebo first. The test for carry over is significant (Mann-Whitney W=247·5, P<0·05).

Proportion of nights with cramp

Treatment	1st Period (n=750)	2nd Period (n=750)	Both periods (n=1500)
Quinine	203 (27)	104 (14)	300 (20)
Placebo	278 (37)	488 (65)	765 (51)

Because of the carryover effect the only valid comparison was between the two treatments in the first period. The difference was not significant (Mann-Whitney W=183, P=0·37). The use of the combined figures for both periods by the authors of the meta-analysis is therefore not legitimate. The results from this trial suggest that the withdrawal of quinine after four weeks' treatment tends to induce leg cramps. This effect is difficult to explain and has not been reported elsewhere.

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Protection afforded by cycle helmets

EDITOR,—According to Richard Keatinge and Ruth Parry, we "defy engineering evidence [unspecified] in stating that cycle helmets reduce the risk of serious head injury in accidents involving motor vehicles."[1] They take us to task for not having mentioned the paper of Spaite *et al.*[2] We referred to this paper in our report in 1993,[3] which we referenced in the letter to which Keatinge and Parry are responding.[4] Spaite *et al* found that injuries to the head and body were less severe in helmeted riders. Sex and age were separately associated with use of a helmet, severity of injury, and mortality. In these circumstances a more sophisticated analysis was warranted, and Spaite *et al*'s conclusion that helmet wearing was simply a

marker for more cautious cyclists cannot be accepted uncritically.

Keatinge and Parry quote a secondary source to the effect that cycle use in Victoria decreased by 40% after wearing a helmet became compulsory. Cycling by children and teenagers decreased by an average of 36% in the two years after the law was introduced, but cycling overall increased.[5]

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Pain in the neck, shoulder, and arm

Terminology used is unhelpful

EDITOR,—The series title "ABC of Rheumatology" implies that the fundamentals of the subject are clearly set out. The article on pain in the neck, shoulder, and arm, however, is likely to result in more confusion than enlightenment.[1] If "mild or moderate degenerative changes [in the neck] are often seen in asymptomatic individuals" then on what evidence do the authors state that "common causes [of pain referred to the arm include]... degenerative changes"? What are the distinguishing features that allow one to conclude that "degenerative changes, including apophysial joint or ligamentous hypertrophy and osteophytes," are among these common causes? And what is meant by mechanical disorders? Does this term refer to a prolapsed cervical disc or include degenerative changes as well?

I agree that "early mobilisation or manipulative techniques... are usually helpful," but where does the idea that "manipulation involves moving the joint beyond normal range" come from? If one attempted to do this it could result in dislocation or fracture.

What is meant by "periarticular disorders [of the shoulder]"? Is this the same thing as disorders of the rotator cuff? What is the evidence for stating that "impingement or tendinitis of the rotator cuff is the commonest problem [causing shoulder pain"]? And what is meant by impingement?

I find it difficult to understand why, if an injection is required to treat a disorder of the rotator cuff, it is given into the subacromial bursa. Surely infiltrating the part of the rotator cuff that contains the lesion would be more effective? Furthermore, the rotator cuff consists of the fibrous capsule of the shoulder joint blended with the tendons of the subscapularis, infraspinatus, and teres minor muscles; it is therefore inaccurate to refer to the "musculotendinous rotator cuff."

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1 Barry M, Jenner JR. Pain in neck, shoulder, and arm. *BMJ* 1995;310:183-6. (21 January.)

Long acting steroid injections are safe and effective if given correctly

EDITOR,—The article on pain in the neck, shoulder, and arm advises on the choice of steroid preparations for intra-articular and soft tissue use in the conditions mentioned.[1] I disagree with the authors that the use of long acting depot preparations should be avoided. Several studies have shown that hydrocortisone acetate is the weakest and triamcinolone hexacetonide and triamcinolone acetonide are the most potent of the steroids currently available in terms of both efficacy and duration of action (M deSilva *et al*, 15th international congress of rheumatology, Paris, 1981).[2,3] Furthermore, relatively large volumes of hydrocortisone acetate are needed for a reasonable dose of steroid, and this is particularly relevant in soft tissue injections for medial and lateral humeral epicondylitis, in which injections have to be made into tight restricted spaces. With the more potent preparations, smaller volumes can be used.

There has been some concern about the use of depot methylprednisolone acetate in soft tissue injections for, for example, the carpal tunnel syndrome. This relates mainly to the fact that this preparation, like hydrocortisone acetate, is a microcrystalline suspension so that crystals may be retained in soft tissues long after the injection. This also explains the postinjection flare seen more commonly with these preparations[4] and is extremely rare with triamcinolone hexacetonide. Few of my patients have complained of postinjection pain after the use of this preparation for intra-articular and soft tissue injections, including for golfer's and tennis elbow. The important factor is that these preparations must be used in the proper dosage and not repeated more than once in superficial soft tissue sites. The need to repeat injections is usually due either to poor technique or to wrong diagnosis.

I also dispute the rationale of injecting steroids and local anaesthetic into the subacromial bursa for disorders of the rotator cuff when direct injection into the shoulder joint is the standard practice. Injection into the subacromial bursa would be more appropriate in acromioclavicular arthritis as direct access to the joint is not particularly easy.

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1 Barry M, Jenner JR. Pain in neck, shoulder, and arm. *BMJ* 1995;310:183-6. (21 January.)
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4 McCarty DJ, Hogan JM. Inflammatory reaction after intrasynovial injection of microcrystalline adrenocorticosteroid esters. *Arthritis Rheum* 1964;7:359-67.

Authors' reply

EDITOR,—Malcolm DeSilva is inaccurate in stating that we recommended that long acting steroid preparations should be avoided for intra-articular injections. In fact, we did not recommend a particular preparation for intra-articular use. Conventional practice is to use long acting preparations when injecting into joints. For soft tissue injections, long acting preparations provide maximum benefit, but this has to be weighed against the greater tendency of these compounds to cause local tissue necrosis if they are not injected into a cavity or if they are accidentally infiltrated into the skin.[1,2] Injection into a tendon may cause it to rupture.[3] For general use we recommend hydrocortisone, although experienced practitioners may prefer to use a long acting preparation in certain situations.

We disagree that intra-articular injection of the shoulder joint is the standard practice for disorders of the rotator cuff. There is a close anatomical relation between the rotator cuff and the subacromial bursa, and reactive inflammation in this bursa is often present in tendinitis of the rotator cuff. The subacromial space or bursa is the recommended site of injection for treating the commoner causes of shoulder pain—namely, impingement, tendinitis of the rotator cuff, and subacromial bursitis.[4]

With regard to degenerative changes in the cervical spine, it is accepted that there is a high prevalence of asymptomatic radiological osteoarthritis in the population. When these changes are seen in a patient presenting with neck pain it therefore does not automatically follow that the neck pain is due to the osteoarthritic changes, and other reasons should be sought. In patients with neck and radicular symptoms, however, advanced osteoarthritic changes causing entrapment of a nerve root may be seen on magnetic resonance imaging. Gabriel Symonds agrees that mobilisation or manipulative techniques aimed at restoring the full range of movement may be helpful in treating neck disorders. In such cases, when a joint is restricted in movement mobilisation entails moving the joint within its range while manipulation entails moving the restricted joint beyond its "normal" range and attempting to improve the range or restore the full range.

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1 Fitzgerald RH. Intrasynovial injection of steroids: uses and abuses. *Mayo Clin Proc* 1976;51:655-9.
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Detection of prostate cancer

Recent evidence suggests screening may be justified in high risk younger men

EDITOR,—Screening for prostate cancer has been the subject of much debate, and Fritz H Schröder considers the published data.[1] His conclusion that population based screening is not yet justified is fair. There is much anxiety that screening for prostate specific antigen will detect a large number of indolent cancers, whose detection will not decrease mortality, and because of false positive results morbidity and mortality may be increased.[1,2] Recent data from a nested case-control study, however, are an important addition to knowledge.[3]

Serum samples were taken from 68% of 22 071 doctors randomised in a continuing study of β carotene in 1982. Three hundred and thirty six men who provided serum samples developed prostate cancer during 10 years of follow up. Three aged matched controls who also supplied serum samples were selected. When a cut off concentration of prostate specific antigen of 4·0 ng/ml was used, at four years of follow up the sensitivity of detection was 87% for aggressive tumours but 53% for non-aggressive cancers. Specificity was more or less unchanged over time at 91%. Nearly 80% of all aggressive prostate cancers occurring within five years would have been detected by a single measurement of prostate specific antigen. Importantly, only 32 of 80 cancers arising more than five years after the sampling time were not aggressive.