between the two heads of the flexor carpi ulnaris muscle, and bulging of the medial ligament: in some otherwise normal elbows diminution of capacity in the flexed-elbow position is such as to cause transitory "sleep palsy."¹⁻³ The cubital tunnel syndrome may usefully be classified as follows³: *physiological* ("sleep palsy"), *acute* and *subacute* (the cubital tunnel external compression syndrome), and *chronic*, which may in turn be divided into space-occupying lesions and tardy ulnar palsy associated with cubitus valgus following injury to the capitular epiphysis (the sequence of events being damage to the trochlear-capitular ossific link, lateral shift of the ulna, and approximation of the arcuate ligament to the floor of the tunnel).

From my experience, I cannot agree that one-third of cases of the cubital tunnel syndrome are of unknown aetiology: I respectfully suggest that many of these cases may be due to external compression. Cases of the subacute form of the cubital tunnel external compression syndrome are not uncommonly seen in hospital practice¹⁻³; all those concerned with caring for patients in the operating theatre and in the ward need to be aware of the syndrome, and external pressure over the cubital tunnel should be avoided.

In those cases of the cubital tunnel syndrome requiring surgery, the usual choice is between simple division of the arcuate ligament, which is then sewn together deep to the ulnar nerve, and anterior transposition. However, the latter procedure is mandatory for the dislocating, hypermobile nerve that is the subject of neuropathy.

The *elbow flexion test*, which I have described,³ is a useful clinical sign for the cubital tunnel syndrome, particularly where the differential diagnosis is confusing: the elbow is kept fully flexed for five minutes and the test is positive when there is initiation or aggravation of numbness or paraesthesiae, or both, in the ulnar nerve distribution. As explained, there is tautening of the arcuate ligament, bulging of the medial ligament, and consequent intraneural hypertension.

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¹ Wadsworth, T G, and Williams, J R, *British Medical Journal*, 1973, **1**, 662.

² Wadsworth, T G, *Anesthesia and Analgesia*, 1974, **53**, 303.

³ Wadsworth, T G, *Clinical Orthopaedics and Related Research*, 1977, **124**, 189.

Dangerous antihypertensive treatment

SIR,—I refer to your leading article (28 July, p 228) concerning rapid blood pressure reduction in severe hypertension. The disquieting report by Dr D H Cove and others (p 245) focuses attention on a distressing complication of malignant hypertension—namely, blindness. Although they had found no previous reports we described this complication in two of 197 patients,¹ one following drug-induced hypotension and one unrelated to treatment. Our patients were more fortunate in that the blindness was temporary, lasting for 30 minutes in one patient and two hours in the second.

For many years it has been our practice to use intravenous diazoxide or other parenteral medication only for hypertensive emergencies such as hypertensive encephalopathy, acute left ventricular failure due to hypertension, aortic dissection, intracranial bleeding, and severe hypertension in late pregnancy. We do

not use parenteral agents routinely in malignant hypertension.

Because intravenous diazoxide may cause hypotension we recently conducted a random allocation study to assess the relative value of intravenous diazoxide, labetalol, and clonidine in 30 episodes of severe hypertension in 27 patients.² Labetalol, which you mention as the alternative to diazoxide, was disappointing in our study. Most patients were already receiving some medication when the episode of severe hypertension occurred and we found labetalol in a dose of 100 mg intravenously ineffective in these patients, as did Rosei *et al.*³ Clonidine in a dose of 300 µg in 10 ml of saline injected over 10 minutes produced a more gradual fall in the blood pressure than diazoxide; hence at five minutes the fall was not as great as that seen with diazoxide, but similar levels were seen between 10 and 180 minutes after the injection. Severe hypotension occurred on only one occasion and this followed intravenous diazoxide. Side effects were encountered with all three medications, but were clinically insignificant in the context of the complications which may warrant such medication. Drowsiness and dry mouth were the major side effects following clonidine.

Both clonidine and diazoxide were equally effective but there may be some advantage in using clonidine because of the more gradual fall in blood pressure seen with this drug.

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¹ Kincaid-Smith, P, McMichael, J, and Murphy, E A, *Quarterly Journal of Medicine*, 1958, **17** (new series), 117.

² Yeung, C K, *et al*, *Medical Journal of Australia*, in press.

³ Rosei, E A, *et al*, *Australia and New Zealand Journal of Medicine*, 1976, **6**, 83.

SIR,—The recent accounts by Dr D H Cove and others (28 July, p 245) and Dr J H Wetherill (1 September, p 550) of young women suddenly losing sight within the first few days of treatment for malignant hypertension should raise alarm in those who treat this condition and make one reassess the teaching that the blood pressure must be lowered to normal as soon as possible.

The pathology of papilloedema in malignant hypertension differs from that of raised intracranial pressure in that there is a patchy ischaemia with fibrinoid necrosis of some fine arterioles and a flooding of the remaining capillary bed from breakdown of autoregulative arteriolar vasoconstriction. There may or may not also be an element of raised intracranial pressure and of raised intraocular pressure. The raised intraocular pressure was recognised in 1964¹ but not its significance, which probably did not matter with the drugs then available. Now that powerful drugs are in use we have the dire results reported. A sudden drop in arteriolar pressure not balanced by as rapid a drop of intraocular pressure would certainly reduce the perfusion pressure. Even with a normal intraocular pressure there may be some risk when the arterial pressure drops suddenly, as in exsanguination blindness. The interstitial pressure—that is, the intraocular tissue pressure—is the intraocular pressure. It would be raised not from the disc oedema but from raised capillary pressure throughout the eye.

In the patient, not blind, reported in 1964¹ there was a week of sedatives and assessment before more drastic treatment, and even at two weeks when the intraocular pressure was back to normal the blood pressure was still 260/130 mm Hg. This man was 70 and did very well. The recent cases unfortunately responded much more dramatically, their youth probably making them so responsive rather than any hormonal factor.

Finally, two more points for the teachers on malignant hypertension. I have been struck by the lack of headache in these ill patients, though one is led to expect it; and there is often unnecessary panic if papilloedema is unilateral and malignant hypertension is suspected—one cannot have the hypertension unilaterally.

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¹ Primrose, J, *British Journal of Ophthalmology*, 1964, **48**, 19.

Medicine and the media

SIR,—I have argued elsewhere¹ that a constructive and co-operative attitude by NHS managers towards the media is likely to do good rather than harm, and that we should be ready to use the vast potential of the media to disseminate understanding of what we are doing and why. May I through your columns congratulate Dr J M Bird on his objective report of his study of the effect on patients' attitudes of publicity about electric convulsive therapy (1 September, p 526), and encourage other clinical colleagues to undertake similar research?

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¹ Elwood, W J, *Community Medicine*, 1979, **1**, 97.

General practice in hospital accident and emergency departments

SIR,—By comparing the rates of use of hospital accident and emergency departments and the general practitioner service, Dr Joyce M Watson and others conclude that no significant shift in work load from general practice to hospital has occurred in their district (11 August, p 365).

It has been suggested that attendance at accident and emergency departments for reasons other than trauma is likely to be inappropriate.¹ If increasing numbers of patients were proceeding directly to hospital, without first consulting their general practitioner, one might expect an increase in inappropriate attendance, which would not necessarily be evident from examining the total attendance figures. The incidence of self-referral in a locality may reflect the local availability of hospital care and the accessibility of the general practitioner²; there may thus be considerable variation in the attendance rates between districts.

To investigate these aspects of the work load of the accident and emergency department, I examined the results of a small survey conducted at the Cambridge Military Hospital, Aldershot, which serves both the military and civilian populations of the area. Seventy-three consecutive patients attending outside normal

working hours were classified according to their presenting complaint, route of referral, and previous attendance at the department. The information collected was tested against two null hypotheses suggested by Dr Watson's article, using the chi-squared technique.

In this small purposive sample no significant difference could be demonstrated with respect to inappropriate attendance either between old and new patients or between those who were self-referred and those arriving through other agencies. This contradicts the subjective impression of some casualty officers that patients may be using the accident and emergency department in lieu of their general practitioner. It also adds support, from a different locality and a different viewpoint, to the conclusion that even though total casualty department work load may be increasing, this does not reflect a shift away from the general practitioner to the hospital department.

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- ¹ Durbin, F C, *British Medical Journal*, 1974, **1**, 125.
² Morgan, W, *et al*, *Hospital and Health Services Review*, 1974, **70**, 189.

Fenclofenac-induced nephrotic syndrome?

SIR,—We feel that the recent report of fenclofenac-induced nephrotic syndrome by Dr D V Hamilton and others (*BMJ*, 11 August, p 391) should be interpreted with caution. We have recently seen a similar case of nephrotic syndrome in rheumatoid arthritis in which there was complete spontaneous recovery and no demonstrable cause.

The patient is a 60-year-old woman with a three-year history of severe seropositive rheumatoid arthritis who has marked rheumatoid deformities and nodules. In March 1979 she was noted to have proteinuria of 0.9 g/l. At this time she had been on penicillamine for six weeks and was having 250 mg twice daily, with naprosyn 750 mg daily and indomethacin suppositories at night. Drugs used in the past included a 15-month course of gold (stopped in August 1978), a three-month trial of fenclofenac in 1977, and intermittent ibuprofen. Penicillamine was stopped, but proteinuria increased and nine weeks later sacral and ankle oedema developed. She was admitted for investigations, which revealed a serum albumin of 12 g/l and urinary protein of 7.5 g/l. Other abnormalities included: urea 8.3 mmol/l, creatinine 138 μ mol/l, haemoglobin 10.6 g/dl, viscosity 2.22 mPa s (2.22 cP), rheumatoid arthritis haemagglutinating antibody titre 1/160, and plasma globulin 51 g/l (increased α_2 -globulins on electrophoresis). C3, C4, C_{H50}, and C-reactive protein were normal. There was no evidence of the hepatitis B surface antigen (HBsAg). A rectal biopsy did not show any evidence of amyloidosis or vasculitis. Intravenous urography was normal. Renal biopsy was performed and showed a mild, diffuse increase in mesangial cells and matrix with moderate diffuse focal interstitial fibrosis. The immunofluorescence test gave essentially negative results and there was no evidence of basement membrane deposits. During a period of three weeks, without any change in treatment, urinary protein decreased to 0.06 g/l and albumin increased to 33 g/l.

This patient clearly had an acute episode of severe nephrosis in rheumatoid arthritis which was entirely self limiting. Although the patient had been on penicillamine, neither the time course of the illness nor the renal histology were consistent with penicillamine nephropathy, the features of which have been recently documented.¹ No cause was demon-

strated in this case but the mesangial nephropathy would be consistent with an acute episode unrelated to her rheumatoid arthritis, such as a viral infection. Clearly there is the possibility of falsely implicating any drugs being taken, with the ubiquitous non-steroidal anti-inflammatory drugs most liable to guilt by association. A variety of renal abnormalities have been described in rheumatoid arthritis, and the role of anti-inflammatory drugs remains unclear.²

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- ¹ Bacon, P A, *et al*, *Quarterly Journal of Medicine*, 1976, **180**, 661.
² Symposium on Renal Disease in Connective Tissue Disorders, *European Journal of Rheumatology and Inflammation*, in press.

Psychosocial stress in pregnancy

SIR,—I read with interest the article by Dr Richard W Newton and others (18 August, p 411). Their conclusion that stressful events may precipitate premature labour is hardly justified by the data presented.

It is well known that women who have delivered premature babies may experience feelings of guilt and recrimination. It is likely that these mothers, when compared with mothers of normal babies, will have better recall of the major life events, especially the more subjective ones listed such as the husband being sarcastic, undue worry over having a handicapped child, and increase in family arguments.

There is a further problem in the method of interview described. Published work¹ has shown that different interviewers may obtain different responses to the same questionnaire. It is therefore unfortunate that the two groups of mothers were questioned by different interviewers.

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- ¹ Choi, I C, and Comstock, G W, *American Journal of Epidemiology*, 1975, **101**, 84.

Spread of steroid-containing foam after intrarectal administration

SIR,—There has recently been an extensive advertising campaign advancing new evidence on the spread of steroid foam for intrarectal use in the management of ulcerative colitis. The advertisers have taken the unpublished results of a study presented at the March 1979 meeting of the British Society of Gastroenterology and used them to demonstrate the efficacy of their preparation. This study has now been published in abstract form but not in detail.¹ [A paper will shortly be published in the journal.—Ed, *BMJ*.]

This would presumably be quite justified were it not for the fact that a similar study using a slightly different technique reported to the Surgical Research Society in January this year by my colleagues and myself showed markedly different results. This latter study has now been published both in abstract form² and in detail.³ The results of both studies are known to the advertisers yet they have publicised only the favourable one.

I completely accept that our study, presented to the Surgical Research Society, may

have used incorrect methods and thus produced fallacious results. It is equally the case, however, that the results presented to the British Society of Gastroenterology arise from incorrect technique. We consider that the method used for labelling the foam in this latter study fundamentally altered its characteristics and thus gave erroneous information. The answer will only come from further study of the characteristics of the labelled foam used in each investigation.

The fact remains that a company promoting a treatment for a serious condition has apparently ignored one of two sets of contradictory data. Advertising by pharmaceutical companies should present all the evidence to the medical practitioner in a scientific fashion in order that he can decide for himself the merits of the preparation which he is being encouraged to use.

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- ¹ Farthing, M J G, *et al*, *Gut*, 1979, **20**, 453.
² Hay, D J, *et al*, *British Journal of Surgery*, 1979, **66**, 359.
³ Hay, D J, *et al*, *British Medical Journal*, 1979, **1**, 1751.

* * * We sent a copy of this letter to the manufacturers, whose reply is printed below.—Ed, *BMJ*.

SIR,—Professor M Irving refers to a series of advertisements used by us in the promotion of Colifoam. These quoted extracts from a clinical paper presented at the British Society of Gastroenterology in March of this year and subsequently published in abstract form in *Gut*.

Professor Irving's main point is that comparable work undertaken by his own unit was not simultaneously included in our promotional material. However, the investigation by Professor Irving's unit was not published until May of this year, when it appeared in abstract form in the *British Journal of Surgery*; subsequently it was published in full in the *BMJ*.

Thus when our promotional material was prepared we had no knowledge that Professor Irving's work had been completed and therefore were not able to make any reference to it. Future promotional material for Colifoam will, of course, include references to both trials, in line with our normal practice. Since publication of the full work in the *BMJ* in June our representatives have been featuring this in their detail activity side by side with the other work. I hope this fully clarifies the sequence of events in a promotional exercise that might otherwise have implied an omission on our part.

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Design of forms for clinical trials

SIR,—While Drs Patricia Wright and John Haybittle (1 September, p 529) are to be congratulated on their attempts to design simple forms for clinical trials, we are a little uneasy about their suggestion that in order to improve accuracy clinicians might be asked to record the physical size of tumours.

We have already shown that data recording may well be extremely unreliable when tumour size and lymph node status in breast cancer are considered.¹ More recently Irving