lot of extra work on the police; (*c*) they did not have enough statistical knowledge; and (*d*) compulsion represented an infringement of individual rights.

Through the London Ambassadors and High Commissioners of the sixteen countries where seat-belt wearing is mandatory an imposing amount of data has been assembled; and at Mr William Rodgers's request has been sent to the appropriate quarter in the Department of Transport.

With regard to (*a*) I could quote, for example, the Soviet reply: "It has been confirmed that safety belts are effective in reducing the gravity of injuries in accidents at speeds below 90 km/h provided they were used properly—that is, when there was the necessary minimum gap between the belt and the body. If the gap is increased the effectiveness of safety belts is sharply reduced. The belts also prove ineffective in the accidents occurring at speeds higher than 90 km/h. Practically all fatal accidents involving persons using safety belts occurred at speeds of 90-140 km/h. There is no record of cases when safety belts were causing injuries." The other countries agree that seat belt injury is too uncommon to be of statistical significance.

In regard to (*b*), none of the police forces in these countries find that their work has become more onorous. To quote the Swiss Embassy, "An inquiry among competent cantonal police organisations reveals that the compulsory wearing of seat belts causes no appreciable extra work"; and Sweden says, "As to the police co-operation, the central police authorities have been much in favour of the compulsory legislation." Objection (*c*) can be countered by the mass of reports now at the Ministry of Transport. France's extensive official survey of its results had to be translated into English—as had the Israeli one.

It was understandable that the Lords should have viewed objection (*d*)—infringement of individual rights—with some misgiving; for like their legal counterparts they desire to uphold the principle of freedom wherever possible. And none can deny that this is something very valuable. But Dr James Cameron in his letter to Parliament brings to our attention that more than half of all male deaths in the age group 15 to 19 are due to road traffic accidents. This sombre fact must surely make the Lords think again.

We, as GPs and surgeons, are more acutely aware than any others in our community of the deep and lasting distress caused by the tragic deaths of these young people; and it is to be earnestly hoped that Parliament will take our advice.

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Use of car headlamps

SIR,—Your special correspondent (9 December, p 1619) does not, I feel, stress sufficiently the need for the use of headlamps in conditions of poor visibility. We are told that the use of the "fog code" described in the latest Highway Code could "much reduce accidents," but the point needs to be made that drivers are required by law to use headlamps or fog lamps with rear lamps when driving under conditions of seriously impaired visibility, whether due to fog, snow, rain, or smoke. This has been of particular relevance recently when drivers, in extremely poor conditions, can be fleetingly seen speeding along motorways in daytime with only sidelights lit. Similarly, drivers in urban areas seem to care little whether or not they can be clearly seen by other road users, including pedestrians.

Although headlamp dazzle worries some drivers, the possible reduction in accidents, particularly those occurring at hazardous road junctions, would surely be considerable if cars were more easily visible. This argument, of course, not only applies to conditions of poor visibility due to fog or snow, but also extends to the routine use of dipped headlamps in urban areas at night. Motorcyclists obviously appreciate the advantages of being clearly seen. Many now regularly use headlamps in all driving conditions and well-known motorcyclists have endorsed this practice.

The police are obviously at a disadvantage in enforcing any law dealing with the compulsory use of headlamps, simply because of the work involved. Publicity explaining the advantages, both to drivers and to pedestrians, of properly aligned headlamps under specific conditions in the daytime and more generally at night could be life saving.

A similar argument could be applied to publicity in favour of wearing seat belts, but if a driver chooses not to wear his seat belt then it is he who is thrown through his windscreen in the event of an accident. If he does not use his headlamps in conditions of poor visibility then other road users, possibly children, may be injured as a consequence of their lack of awareness of his approach.

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Shingles: a belt of roses from Hell

SIR,—The last paragraph of the leading article on shingles (6 January, p 5) has moved me to write this letter because I have never seen real improvement in patients referred to me by neurologists or other physicians for postherpetic pain. I am quite certain, however, that patients starting treatment on the first or second day of the formation of blisters given three to five doses of 50 rads on successive days at the root ganglion or ganglia concerned do not develop postherpetic pain. Moreover, the skin condition may heal more rapidly than usual, although of this I am not certain. The field size to be used and the estimate of the position and depth for the dose to be delivered depend on the anatomy. The doses given are so small, and to such a small volume, that unwanted early or late clinical radiation effects are absent. No radiation is given to the affected skin. The important point is to give the treatment early. In many cases even without treatment of any kind no pain would develop, but to wait to see if there is postherpetic pain is to leave any treatment too late.

The reason for the effect is not established. My working hypothesis (right or wrong) was that there was an acute small round cell inflammatory reaction of the root ganglion and that the radiation caused disintegration of the lymphocytes, setting free gammaglobulins in large concentration at the site of the virus activity. If the inflammatory process is allowed to follow its natural course, scarring in the ganglion provides a permanent pathology which produces the pain.

In some way a failure of immune response seems to be the precipitating cause of shingles, as shown by its development in patients with grossly disturbed immune mechanisms mentioned in the sixth paragraph of the leading article. I have seen a patient die with purpura fulininans complicating extensive shingles due to cytotoxic drug treatment of Hodgkin's disease. Radiation in large doses to large volumes can be followed after a latent period by shingles not necessarily in the irradiated region.[1] I have always been puzzled by the apparently capricious affection of one rather than another nerve, although the development of more extensive disease when the immune damage is greater suggests that the virus might be widespread even when the shingles is not.

F Ellis

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[1] Ellis, F, and Stoll, B A, *British Medical Journal*, 1949, **2**, 1323.

SIR,—I enjoyed reading your leading article on herpes zoster (6 January, p 5), and I would like to express the following points of view as an otolaryngologist.

Herpes laryngis may be associated with facial herpes, as was first noticed by McKenzie,[1] and examination of the larynx is recommended in cases of head and neck zoster.[2] Also, in cases of idiopathic cord palsy, especially if of recent onset, serological tests for herpes zoster may give a clue to the cause. As your article indicated, pain during the attack may be very severe and in this case carbamazepine (Tegretol) may be of help if the pains are along the trigeminal nerve.[2]

Although your article indicated that zoster affecting the facial nerve appears to have a good prognosis, some authorities believe that the prognosis in cases of herpes zoster is worse than in cases of Bell's palsy[3]; however, this point is questioned by others.

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[1] McKenzie, D, *Journal of Laryngology and Otology*, 1915, **30**, 339.
[2] Pahor, A L, *Journal of Laryngology and Otology*, 1979, **93**, 93.
[3] Dalton, G A, *British Medical Journal*, 1960, **1**, 1765.

Effect of once-daily atenolol on ambulatory blood pressure

SIR,—Dr M W Millar Craig and his colleagues (27 January, p 237) provide interesting information on circadian variations of blood pressure in six patients on once-daily dosing of atenolol, obtained using intra-arterial recording. After a paper about untreated hypertensive patients[1] several letters[2–4] drew attention to earlier observations obtained using non-invasive methods of recording blood pressure. These results were notable for their similarity to those reported with the intra-arterial ambulatory method,[1] and suggest that information obtained by non-invasive methods may contribute to understanding circadian variations.

Dr A M J Woolfson and I used non-invasive automatic blood pressure recording equipment and reported measurements of blood pressure over 24-hour periods in patients before and during hypotensive therapy with atenolol.[5] An effect was seen on nocturnal and early morning blood pressure, suggesting that once-daily atenolol can control nocturnal and early morning blood pressure. Only one of five

patients showed a temporary morning rise to pretreatment levels when on regular administration. Further studies are needed before conclusions are accepted based on our paper on five patients and on Millar Craig's paper based on six, especially as these conflict.

In a reply to Dr Millar Craig's earlier paper,[1] we discussed the reasons for some of our uncertainties about whether the early morning rise is important in causing morbidity and mortality.[4] Earlier studies carried out by myself and other colleagues of the late Dr D H Davies[6] were initiated in an attempt to explain why vascular changes in accelerated hypertension could be reversed by treatments which caused only daytime hypotension (the early ganglion-blocking drugs were effective only in the upright position). Patients on such treatment showed abrupt changes in blood pressure with changes in posture. We demonstrated that, after treatment with the hypotensives in common use at the time, post-treatment circadian variation in blood pressure usually reflected the variations recorded before treatment. Those patients with reduced day-night variation, as observed in accelerated hypertension before treatment,[6] showed less day-night variation than others—even after treatment.

The intra-arterial technique is elegant and very suitable for studies on circadian rhythmicity, but may not invalidate all previous observations. With the improvement in the quality of data collected more sophisticated analysis should be possible. There should be some reluctance to describe a circadian rhythm until this has been characterised by methods developed by those interested in biological rhythmicity.[7] The term "circadian variation" may be better than "circadian rhythm" if a statistically significant similarity to a sine wave has not been established.

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[1] Millar Craig, M W, *et al*, *Lancet*, 1978, **1**, 797.
[2] Stewart, I M, *Lancet*, 1978, **1**, 1210.
[3] Pickering, G, *Lancet*, 1978, **1**, 995.
[4] Knapp, M S, *Lancet*, 1978, **1**, 1211.
[5] Woolfson, A M J, and Knapp, M S, Proceedings of the Royal Society of Medicine, 1977, **70**, 36.
[6] Shaw, D B, Knapp, M S, and Davies, D H, *Lancet*, 1963, **1**, 797.
[7] Halberg, F, *et al*, *Chronobiologia*, 1977, **4**, suppl, p 1.

SIR,—Dr M W Millar Craig and others (27 January, p 237) present data on six subjects with essential hypertension, studied with intra-arterial blood pressure recordings before and after treatment with once daily atenolol, and conclude that it is ineffective at night and in the early morning. Their data differ markedly from our own experience, where 12 subjects were studied before and after chronic treatment with atenolol. A significant antihypertensive effect was noted throughout the 24-hour period.[1] Discrepancies between these two studies may be due to methodological and technical difficulties with intra-arterial records and to differences in the interpretation of the data.

The authors state that control of blood pressure was achieved in the clinic before a second study was considered. Control was defined as 140/90 mm Hg. However, it would appear from their published illustration that the averaged pressures in this "treated" group began the day at 180/100 mm Hg, and indeed average systolic pressures did not fall below 145 mm Hg, even during sleep. We would conclude that some of this group were inadequately treated before restudy.

There are several additional explanations for their findings of a significant fall in daytime pressure without a change in pressures at night. We have previously pointed out that this method of pooling patients' blood pressures in terms of hourly means can give spurious results, particularly during the early hours of the morning.[2] The differing hours of waking in different patients are clearly responsible for the apparent early morning "pre-waking" rise in blood pressure reported by Millar Craig[3] and currently claimed to be unresponsive to beta-blockade; when the same data are analysed around the accurately determined point of waking, we found that the rise over the previous hours was only about 5 mm Hg. The actual rise in pressure on waking is large and sudden.[4] In the atenolol study reported, the small numbers (6 and 4) plus the scatter in waking times at this period of the day could easily account for this apparently insignificant control of blood pressure in the early morning. As the number of subjects studied is rather small, one must be cautious in the blind interpretation of statistics. Damping of the pulse wave at night is a common problem; this as well can distort results. Furthermore, circadian rhythms in blood pressure are primarily responses to exogenous rather than endogenous processes; consequently blood pressure data cannot be readily interpreted without knowledge of the subjects' corresponding activity. Some assessment of the depth and quality of sleep should be made whenever one wishes to examine blood pressure in subjects who are free to sleep in their own home. We are told that changing the time of medication from 8.30 am to 11.00 pm had no effect on night-time pressures. This confirms our thesis that the duration of action of atenolol might not be responsible for the reported lack of night-time effect.

The authors suggest also that beta-blockers may not affect α-adrenergic discharge. We showed earlier that the fall in blood pressure during sleep was largely due to a fall in peripheral resistance, which we would agree is probably due to a fall in sympathetic tone.[5] The subject is complex; studies in animals and man have shown that sympathetic traffic may be decreased by the central action of beta-blockers,[6-8] and also that noradrenaline released at nerve terminals may be reduced by beta-blockade.[9] Although it is often said that some beta-blockers do not enter the central nervous system, clinical observations (dreams, sedative effect) would argue to the contrary. Moreover, important areas of the brain concerned with cardiovascular regulation are devoid of a blood-brain barrier.

We would suggest that the factors listed above are more likely to be responsible for the observations reported, and that it is difficult to draw firm conclusions from the observations of Millar Craig and his co-workers. The inference that once-daily atenolol fails to control blood pressure over 24 hours cannot be made from this evidence. Our experience, with a greater number of subjects, shows that the drug is effective in a single daily dose—a finding that is in agreement with numerous other studies using conventional non-invasive measures of arterial pressure.[10]

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[1] Floras, J S, *et al*, in preparation.
[2] Floras, J S, *et al*, *Clinical Science and Molecular Medicine*, 1978, **55**, 395S.
[3] Millar Craig, M W, Bishop, C N, and Raftery, E B, *Lancet*, 1978, **1**, 795.
[4] Littler, W A, and Watson, R D S, *Lancet*, 1978, **1**, 995.
[5] Bristow, J D, *et al*, *Cardiovascular Research*, 1969, **3**, 476.
[6] Day, M D, and Roach, A G, *Clinical and Experimental Pharmacology and Physiology*, 1974, **1**, 333.
[7] Lewis, P J, *Clinical Science and Molecular Medicine*, 1976, **51**, 549S.
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[9] Ljung, B, *et al*, *Blood Vessels*, ed A J Bevan, *et al*, p 311. Basel, Karger, 1975.
[10] Marshall, A J, Barritt, D W, and Harry, J D, *Postgraduate Medical Journal*, 1977, **53**, suppl 3, p 168.

SIR,—Dr M W Millar Craig and others (27 January, p 237) observed no effect of once daily atenolol administration on nocturnal and early morning blood pressure. It has previously been pointed out[1 2] that their method of analysis using average hourly pressures, without reference to the time of waking, may obscure the important influence of waking and physical activity on blood pressure. The importance of these events was not discussed in their paper. This method of recording blood pressure in free-ranging individuals cannot be used to discern alterations in blood pressure which are independent of these factors.

We have measured intra-arterial pressure with the same method in hypertensive patients (mean age 40 years) before and after treatment with β-adrenoceptor antagonists administered for 11 weeks (range 8-17 weeks). Our patients were studied in hospital under carefully defined conditions, particularly with regard to waking and physical activity. They were treated with metoprolol 200 mg, propranolol 240 mg, or acebutolol 400 mg. The last dose of drug was administered between 7.00 and 8.00 am on the day of admission, after which they received no treatment.

The accompanying table shows average systolic and diastolic pressures, analysed beat by beat on a computer, with reference to physical activity rather than time. The results indicate that treatment substantially reduced blood pressure during sleep, after awakening and remaining in bed between 6.00 and 9.00 am, and during ambulation around the ward between 9.00 and 10.30 am—that is, at least 24 hours after the last dose of drug. Differences between our observations and those of Millar Craig *et al* are unlikely to be due to differences in the duration of action of different β-adrenoceptor antagonists, since others have demonstrated a significant reduction of arterial pressure 24 hours after atenolol[3-5]; we consider that recognition of the influence of physical activity and waking is essential for the proper interpretation of 24-hour blood pressure recordings and that failure to do so may confound rather than clarify. Furthermore, when pressures before

Mean blood pressures (mm Hg) before and after treatment with beta-blockers during different activities

Activity	Systolic blood pressure		Diastolic blood pressure	
	Pre-treatment	After treatment	Pre-treatment	After treatment
Sleep n = 11	118±5	108±6**	74±3	66±3**
Bed rest after waking n = 8	130±5	113±6**	83±4	72±4*
Ambulant in ward n = 9	140±8	118±7**	93±5	73±4***

*P<0·05; **P<0·02; ***P<0·005; Student's *t* test for paired observations, 2-tailed.