## Discussion

This study showed that an unrestrained rear seat passenger was not a significant hazard to a restrained front seat occupant. Tests using dummies in controlled crashes suggested a large increase in risk of injury when there was a rear seat passenger[2]; these tests, however, were all in severe collisions (change in speed on impact 48 km/h). In this study only 2·5% of the crashes were due to such great changes in speed, and we would therefore not necessarily expect similar conclusions to apply. Examination of individual accidents showed that in some cases injuries to front seat occupants were worsened by the presence of rear seat passengers.[1 6] This study suggests that such instances are not common, and even in this moderately large sample there were only a few cases of very severe injury. Any causal factor for such injuries would therefore need to have a large effect to be detectable.

Table II shows an increase with crash severity of the effect of rear seat passengers on injury to unrestrained front seat occupants. It is reasonable to suggest that a study that concentrated on restrained front seat occupants in high severity crashes would also show an effect. Such a study would not, however, include a representative sample of crashes, or it would need to select a subset of cases from a much larger study. The low proportion of accidents with a large change in speed on impact, together with the low prevalence of use of seat belts in this study, makes the appropriate subset too small to give useful results.

This study showed that the use of a restraint by the person in front seemed to make rear seat passengers safer. This may be because the combination of the front seat and its restrained occupant has an increased capacity for absorbing energy that outweighs the increased risk of person to person injury.

The exclusion of minor accidents for which ambulances were not called out may have affected the results. The results at higher crash severity (table II), however, suggest that inclusion of minor accidents would not greatly have affected our conclusions for front seat occupants. In the case of rear seat passengers (table IV) it seems likely that any bias in the excluded cases would be towards cars with restrained front seat occupants, so any corrections would reinforce the conclusions.

The greatly increased prevalence of use of seat belts after the introduction of legislation is unlikely to have put rear seat passengers more at risk, and belted front seat occupants do not appear to be greatly endangered by their rear seat passengers, with or without restraint.

There were few restrained rear seat passengers in this study (two adults and 19 children). We therefore have no significant evidence about the effectiveness of rear seat belts. Analysis of the mechanisms of injury, however, suggests that rear seat passengers were injured by being thrown against the source of the injury rather than by it intruding on them. As this type of contact and the resultant injury can be effectively prevented by the use of seat belts it is likely that their widespread use in rear seats would considerably decrease the incidence of injury to car occupants.

We thank Dr R J Tunbridge, Transport and Road Research Laboratory, for helpful discussions in the preparation of this paper. The work described in this paper was carried out under contract to the Transport and Road Research Laboratory, but the views expressed are ours and not necessarily those of the research laboratory or any other part of the Department of the Environment, the Department of Transport, or any other government department.

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# SHORT REPORTS

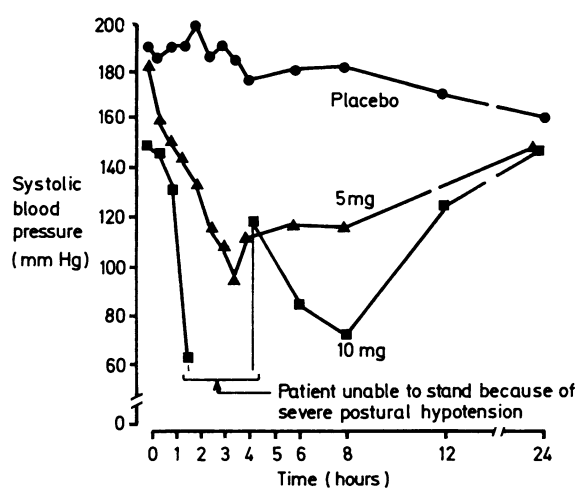
## Initial dose of enalapril in hypertension

Enalapril, a new angiotensin converting enzyme inhibitor, has recently been released in the United Kingdom for the treatment of hypertension and congestive heart failure. The datasheet recommends that "when enalapril is added to previous diuretic therapy in hypertension the initial dose is 10 mg." We believe that this recommendation requires further evaluation because our recent experience suggests that this starting dose may be hazardous in some patients.

### Case report

A 47 year old hypertensive woman participated in a randomised double blind clinical study in August 1984. After four weeks' treatment with bendrofluazide 5 mg daily as the sole drug she was allocated to receive a placebo and 10 mg and 20 mg enalapril as single oral doses separated by at least seven days. On each study day supine and standing blood pressures were measured with a Hawksley random zero sphygmomanometer before and at intervals after administration of the test drug.

The first study day, when she received the placebo, was uneventful (figure). On the second study day, when she received 10 mg enalapril, both supine and standing blood pressures fell steeply within one and a half hours of ingestion of the drug. She felt weak, lightheaded, and nauseated and was unable to stand up during the next four hours because of postural hypotension. Her blood pressure gradually returned to the usual value but she experienced nausea and vertigo for the next 72 hours. No abnormal neurological signs were elicited. Her treatment allocation was decoded, and she did not receive the 20 mg dose of enalapril. Two weeks later 5 mg enalapril was given under identical conditions, and a large drop in blood pressure was again observed. On this occasion symptoms of hypotension did not develop. On the second study visit, when she received 10 mg enalapril, the 24 hour urine volume was 2408 ml, 24 hour urinary sodium excretion 137 mmol (mEq), supine plasma aldosterone concentration 480 pmol/l (173 pg/ml), and supine plasma renin activity 11 nmol/l/h (14·3 ng/ml/h).




Standing systolic blood pressure at intervals after placebo and single doses of enalapril 5 and 10 mg.

We subsequently studied according to the same protocol 13 hypertensive patients taking bendrofluazide 5 mg daily. Symptomatic hypotension occurred after enalapril in three of them. We were unable to differentiate the hypotensive effects of single oral doses of 10 and 20 mg enalapril.

### Comment

It is well recognised that angiotensin converting enzyme inhibitors may cause a profound drop in blood pressure when given to hypertensive patients, especially if the patients are receiving diuretic treatment or are salt depleted.[1] Profound hypotension may also occur in some patients with congestive heart failure.[2]

We used only a modest dose of background thiazide diuretic in our study; many hypertensive patients receive larger doses and more potent diuretics and may be at greater risk of hypotension. Whether a lower initial dose of enalapril such as 2·5 or 5 mg would cause hypotension is uncertain, and further studies are planned to investigate this. In the meantime we emphasise the need for caution when enalapril is added to existing diuretic treatment.

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# Accidental digitalis poisoning due to drinking herbal tea

I report on a patient who developed digitalis toxicity after drinking a herbal tea made with foxglove leaves, which he had mistaken for comfrey leaves.

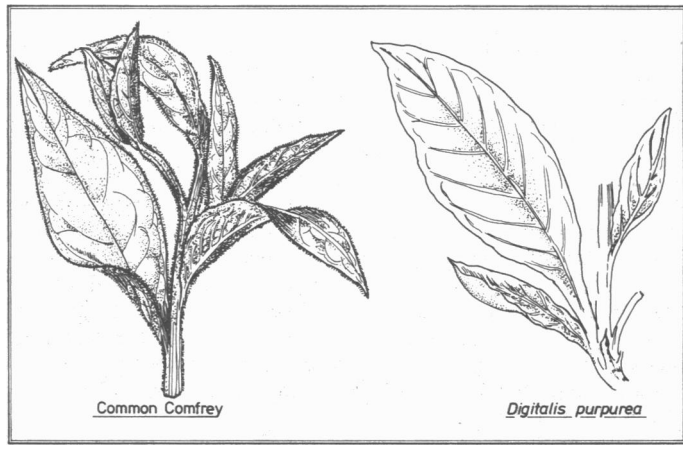
### Case report

A 70 year old, previously healthy, man was admitted with a two day history of frequent bile stained vomiting and a one day history of watery diarrhoea. On admission he was confused, listless, lethargic, and dehydrated and had hiccoughs. He complained of seeing flashing lights and was hypotonic and unsteady on his feet. In the casualty department he was noted to have an irregular pulse of around 45 beats/min and a blood pressure of 150/90 mm Hg; he did not have fever or any other abnormal cardiovascular or respiratory signs. Abdominal examination showed minimal epigastric tenderness and normal bowel sounds. Neurological examination showed him to be mildly confused, with no focal neurological signs, normal reflexes, and downgoing plantars. An electrocardiogram showed a bradycardia with a varying P-R interval (0·14-0·21 s), occasional dropped beats, and "reversed ticking" of the ST segment in leads I, AVL, $V_{4-6}$, II, III, and AVF. Serum biochemistry gave the following results: amylase activity 184 IU/l, urea concentration 9·0 mmol/l (54 mg/100 ml), sodium 138 mmol (mEq)/l, potassium 6·8 mmol(mEq)/l, chloride 105 mmol(mEq)/l, and bicarbonate 28 mmol(mEq)/l.

Digoxin toxicity was provisionally diagnosed, although he denied having taken any medication and close questioning of his family did not indicate any possible source of digoxin. Electrocardiographic monitoring showed complete heart block with a ventricular rate of 40 beats/min. Serum was sent for digoxin assay, and he was given 0·6 mg atropine intravenously slowly, and activated charcoal by mouth. The hyperkalaemia was corrected with 20 units soluble insulin in 500 ml 10% dextrose given intravenously, the serum potassium concentration falling to 4·9 mmol/l.

After the administration of atropine the electrocardiogram showed complete heart block with accelerated idionodal rhythm at 100 beats/min. He remained well perfused and normotensive. Two hours later, however, he again had complete heart block with a slow ventricular rate. A temporary pacemaker was inserted pervenously into the right ventricle and he was paced at a ventricular rate of 70 beats/min.

His wife then said that he had for many years brewed "knitbone," a herbal tea concocted from comfrey leaves (*Symphytum* spp) that he harvested locally, and it was to this potion that he attributed his previous good health. He had drunk one litre of the brew over the preceding few days. His wife provided a similar bottle of knitbone of a more recent brew, and this was retained for analysis.




He remained confused with visual disturbance for four days, after which his electrocardiogram indicated first degree heart block (P-R interval 0·21 s). Ten days after admission he had completely recovered and was in sinus rhythm, and he was discharged from hospital. An electrocardiogram did not show ischaemic heart disease. Subsequently he brought in a sample of the plant that he had harvested thinking it was comfrey. It was a specimen of foxglove.

### Comment

Herbal tea is a common drink throughout the world and is becoming more popular in England. It is surprising that only one similar incident of accidental digitalis poisoning, which occurred in Ohio, America, has been reported.[1] Those wishing to make comfrey tea must distinguish the broad leaves of comfrey from those of the foxglove (figure), particularly when the plant is not in flower and so is less immediately identifiable.

In acute digitalis poisoning the treatment is essentially supportive. Mortality is high and depends on the dose ingested and the presence of any underlying ischaemic heart disease.[2] Even comfrey tea correctly made may not be without its dangers: the leaf is carcinogenic in rats,[3] and pyrrolidine alkaloids in the leaves and roots can produce severe liver dysfunction.[4]

I thank Dr M MacIver for permission to report this case and Dr S R Willetts and Dr J Wood for undertaking the digitoxin assays.

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# Treatment of a phaeochromocytoma of the urinary bladder with nifedipine

Calcium channel blockers could be useful in the treatment of a phaeochromocytoma.[1] In this report we describe the effects of nifedipine in a patient with a phaeochromocytoma of the urinary bladder which caused severe rises in blood pressure as a result of mechanical stimulation during voiding.

### Case report

A 25 year old woman was referred because of hypertension. For six years she had noticed that severe headache, blurred vision, nausea, and pallor always occurred immediately after voiding. Extensive investigation confirmed the presence of a phaeochromocytoma of the wall of the urinary