results in vasodilatation and relaxation of the mammalian penis leading to erection.[1-3] However, there is no evidence to suggest that the transmitter responsible for this inhibitory effect is cholinergic in nature. Indeed the identity of the transmitter has so far not been established. We have previously suggested the possibility that the inhibitory transmitter leading to erection may be histamine.[4] The smooth muscle of the erectile corpus cavernosa of the human penis in vitro is either contracted or relaxed by histamine. The former effect is abolished by mepyramine (a histamine H_1-receptor antagonist), which also potentiates the relaxant effect of histamine on this tissue. The relaxant effect of histamine on the human penis is abolished by burimamide,[4] which is chemically related to cimetidine and is known to antagonise actions of histamine mediated through H_2 receptors.

Thus cimetidine, by blocking H_2 receptors on the body of the penis, may prevent erection. However, definite proof that histamine is the neurotransmitter at the sacral parasympathetic nerve is lacking.

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[1] Langley, J N, and Anderson, H K, *Journal of Physiology*, 1895, **19**, 85.
[2] Henderson, V E, and Roepke, M H, *American Journal of Physiology*, 1933, **106**, 441.
[3] Bacq, Z M, *Archives of International Physiology*, 1935, **40**, 311.
[4] Adaikan, P G, and Karim, S M M, *European Journal of Pharmacology*, 1977, **45**, 261.

A case of compulsory admission

SIR,—Your correspondent JSP (7 April, p 949) may have found the *Man Alive* programme of 27 March entertaining, but if he considers that it was a "responsible piece of journalism" he was not paying attention. He describes it as an excellent programme; it was a slanted, misleading distortion.

Four doctors at the time (two psychiatrists, a consultant physician, and the GP who had been more concerned with the patient recently than any other) agreed that admission was appropriate. Only one, Dr Whitehead, the NHS psychiatrist involved, disagreed; he had not seen the patient for several days, and my attempt to make contact with him at the time was unsuccessful. The social worker saw the patient for a few minutes only and could not be expected to consider the differential diagnosis; the patient's "housekeeper" had been with him for just about three weeks, and "his" solicitor first became involved a fortnight before (the solicitor who had looked after the patient's affairs for 30 years and who, I am informed, is now doing so again was temporarily displaced——perhaps by reason of the patient's disordered mental state at the time).

On admission he was seen by a consultant physician, who noted, "On arrival here he was thin, unfit, and moderately dehydrated. . . . With adequate feeding, nursing care, etc, his condition improved steadily without specific therapy other than for his anaemia, and his weight rose steadily from 8 stones to 8 stones 8 pounds."

He remained on as an informal patient for five months after expiry of the Section 25 order, so presumably even the patient considered his admission was not misplaced. He then left to live with one of the daughters pilloried in the programme. He has been with her for a year. All strange behaviour indeed for one who has been "Put Away," as the programme was emotively entitled.

Dr Whitehead's opinions are not shared by the Royal College of Psychiatrists: "The College feels that, as far as possible, relatives themselves should be encouraged to take responsibility and continue to be involved in admission and discharge procedures rather than to relegate this entirely to a professional group (social workers)."[1]

Why was this propaganda exercise allowed to be presented as a serious documentary programme? One can but speculate on this; surely, however, it is time that measures were taken by the BBC to improve their standards of reporting.

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[1] *Bulletin of the Royal College of Psychiatrists*, April 1979.

New approach to treatment of recent stroke?

SIR,—Dr A K Admani's report (16 December, p 1678) of a double-blind trial of naftidrofuryl (Praxilene) in acute stroke has prompted the manufacturers, Lipha Pharmaceuticals Limited, to produce a brochure extolling the virtues of the drug in stroke patients.

The brochure includes descriptions of two patients admitted to the stroke unit of the Northern General Hospital, Sheffield, presumably under Dr Admani's care, in September 1978. The first was admitted following the sudden onset of loss of consciousness and a left-sided weakness. A distinction between cerebral haemorrhage and infarction was not attempted. The paper itself indicates that strokes due to recent ischaemic cerebral infarction were studied, whereas the manufacturers' literature gives as the definition of stroke used for the purposes of the trial a deficit produced by an ischaemic lesion in the cerebral hemisphere (thromboembolic or haemorrhagic insult). The second case description refers to a patient with a left hemiplegia and a carcinoma of the bronchus. Treatment included dexamethasone. The paper on the trial indicated that only strokes due to vascular causes were included.

Both patients made good recoveries, with the implication that naftidrofuryl had been at least partly responsible. The assumption is unsatisfactory in the first case, where the distinction of haemorrhage from infarction was never attempted, and in the second case, where adequate measures to exclude a cerebral metastasis were not taken.

A criticism of the trial paper has already been published (10 February, p 412). The unwarranted assumption by the manufacturers about the efficacy of the drug in two patients who would not have qualified for the trial in the first place and their description in their literature of "a major development in acute stroke therapy" seem to indicate that they have not understood or considered the merits of that criticism.

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Shortening hospital stay for psychiatric care

SIR,—In his letter (17 March, p 751), Dr Peter F Kennedy has misinterpreted the study we reported earlier (17 February, p 442), in which we carried out a randomised experiment to examine the effect of an across-the-board administrative policy to shorten hospital stay for acute psychiatric patients.

The success of the experimental variable, random assignment to brief care, in reducing length of stay is reflected by the fall in the mean length of stay of 33 days for all admissions during the year before the study (1974) to a length of stay of 22 days for patients randomly assigned to brief care during the year of the study. This was an overall saving of 33%; but from the point of view of number of patients affected the saving is even greater in that the median length of stay fell from 24 days to 9, a reduction of 63%. The difference in length of stay between the brief and standard care patients fell during the experimental year owing to a halo effect. However, the potential administrative saving which can result by arbitrarily deciding that inpatient care should be as short as possible must be judged by comparing the length of stay of the experimental group to that of all patients during the year before the study.

We have data to show that the standard care patients received, if anything, higher doses of medication, and that there was no difference in the amount of social work or outside support given to the two groups. This was possible because brief and standard care patients were treated in the same facility spread between five consultants, so that heterogeneity of approach was more likely.

Unfortunately, the approach which Dr Kennedy mentions, in which all brief-care patients are sent to a separate experimental ward, does not throw light on the effects of brief care as such, for their therapeutic approach was different. This, Dr Kennedy states, "involved staff having to learn new skills and deploy their time quite differently." I will be interested to see if Dr Kennedy is able to identify whether his results are due to one experimental variable or the other.

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Drug-induced neurological disease

SIR,—Dr E M R Critchley in his article on drug-induced neurological disease (31 March, p 862) listed a wide range of drugs capable of causing convulsions. Recently, with the increasing number of inquiries we have received regarding solvent inhalation, it has come to our attention that convulsions may result from the acute intoxicant effects of glue sniffing.

Case 1—While sniffing Evostik glue, a 14-year-old boy became suddenly unconscious and was observed to have a generalised convulsion involving all four limbs. He remained unrousable over the following ten minutes, during which time he had a further convulsion. On arrival at hospital he was drowsy and complained of being unable to see clearly. Apart from widely dilated pupils, examination revealed no abnormality and after 24 hours of observation he was discharged home. The patient had a two-year history of glue sniffing, of which his family were unaware.

Case 2—A 15-year-old boy collapsed after sniffing Evostik glue continuously for four hours. While he was unconscious his companions reported several episodes of violent shaking of his arms and legs. By the time he was admitted to hospital he was fully conscious and very aggressive. Physical examination was normal and he was discharged home the following day. The patient had a two-year history of intermittent glue sniffing and had previously experienced five similar episodes of collapse while inhaling solvents.

Case 3—The patient, a boy of 12 years, was

brought to hospital having been found unconscious at home after sniffing Evostik glue. Almost immediately on arrival at hospital he had a typical grand mal convulsion, which was controlled by 5 mg of diazepam given intravenously. He regained consciousness after 20 minutes and was detained in hospital for four days. Investigations revealed no evidence of hepatic or renal dysfunction and he was subsequently discharged home. It is not known whether this patient had indulged in glue sniffing in the past.

Toluene, the chief constituent of Evostik glue, is the agent most likely to be responsible for these seizures, although its mode of action is not clearly known. A rise in cerebrospinal fluid pressure and even cerebral oedema may occur following glue sniffing[1 2] and toluene in particular has been implicated in causing cardiac arrhythmias.[3] Whatever the cause, glue sniffing is a popular practice among the young and is worth bearing in mind in previously non-epileptic adolescents who present at hospital as a result of an epileptiform seizure.

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[1] Barman, M L, *et al*, *California Medicine*, 1964, **100**, 19.
[2] Winek, C L, Collom, W D, and Wetch, C H, *Lancet*, 1967, **1**, 683.
[3] Taylor, G N, and Harris, W S, *Science*, 1970, **170**, 866.

Accidental hypothermia and low-reading thermometers

SIR,—Professor G L Mills suggests (21 April, p 1082) one reason for diagnosing hypothermia is failure to use a low-reading thermometer. In the accident and emergency department of Edinburgh Royal Infirmary if temperatures fail to register on a normal clinical thermometer the nurses routinely use an electronic thermometer (range 15-45°C) with a rectal probe. This avoids any errors due to failure to shake down the mercury and the thermometer can accompany the patient to the ward with the probe left in situ for repeated measurements, thus avoiding the hazard of unnecessary movement of the patient. Glass and mercury thermometers have the additional danger that they can break during use.

In the treatment of hypothermia "space blankets" have been replaced by polyethylene sheeting, which is equally effective,[1] is cheaper, and is less liable to tear. It also has the advantage that there is none of the continual crackling noise produced by the metallised "space blanket," which is very distressing to the confused patient.

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[1] Marcus, P, Robertson, D, and Langford, R, *Aviation, Space and Environmental Medicine*, 1977, **48**, 50.

SIR,—While agreeing with Professor Gordon L Mills (21 April, p 1082) that a low-reading thermometer is useful in detecting and indeed essential for diagnosis of hypothermia I would suggest that those most sensitive of diagnostic tools the hands are just as useful in detecting variations from normal body temperature. While a patient with a normal core temperature may have cool extremities owing to poor peripheral perfusion, palpation of the trunk will in nearly all cases alert the clinician to the presence of abnormal body temperature. If the hand is inserted between the patient and the bed on which he lies the impression is more accurate than that gained by feeling exposed parts. The suspicion of either fever or hypothermia should then be checked with a suitable thermometer.

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The use and abuse of Distalgesic

SIR,—Following the comments by Dr J M Gumpel (24 February, p 551) and the recent controversy regarding the use and abuse of Distalgesic which has featured in your columns, we have undertaken a preliminary survey at the rheumatology clinic at St Stephen's Hospital in order to determine the prescribing pattern for Distalgesic and explore the possibility of its abuse among the rheumatic patients.

One hundred and four consecutive patients attending the rheumatology outpatients department were interviewed by one of us (JF) and a detailed record was made of current drug therapy, use of dextropropoxyphene-containing medications, side effects, and source of prescription. Each patient was closely questioned regarding the possibility of habituation and of exceeding the prescribed dosage. A record was also made of the patients' understanding of why they were taking the drug.

Thirty-seven patients (35·6%) were currently taking Distalgesic—no other preparations containing dextropropoxyphene were being prescribed. The dose range varied from two to eight tablets per day but 22 patients were taking it only on an occasional basis. There were no instances of habituation or of excessive consumption and only one patient complained of a side effect (constipation) which could be attributed to Distalgesic. Three patients did not know why they were taking Distalgesic, but all the others used it for pain relief.

The Washington-based Health Research Group has asserted that dextropropoxyphene's relative ineffectiveness as an analgesic has caused patients to increase dosages beyond the prescribed regimens.[1] This is certainly not our experience; and although we recognise the hazards of overdosage of dextropropoxyphene, particularly in combination with alcohol, we would endorse the views of Dr Gumpel that Distalgesic is a relatively safe and effective analgesic in the management of chronic painful conditions.

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[1] Dickinson, J, *Pharmaceutical Journal*, 1979, **222**, 221.

Nebulised salbutamol in life-threatening asthma

SIR,—The study by Dr P Bloomfield and others (31 March, p 848) sets out to compare the efficacy of salbutamol given by an intermittent positive breathing (IPPB) device and intravenously in acute asthma. We are very conscious of the problems in setting up such a comparison; nevertheless, we feel that there are several controversial points in the study and its conclusions that merit discussion.

They used a change in pulsus paradoxus as their most "sensitive index of improvement" in severe acute asthma. In our experience pulsus paradoxus has often proved to be an unreliable sign in the assessment of asthma. Shim and Williams[1] showed that the respiratory pattern can alter this sign. Increasing the inspiratory flow rate while maintaining a constant tidal volume immediately before the reading of the systolic fluctuation can increase the degree of pulsus paradoxus; while slowing the inspiratory flow rate can cause the sign of pulsus paradoxus to disappear in many patients. Therefore our most serious criticism of the study by Bloomfield *et al* is that pulsus paradoxus may be absent in the presence of severe airways obstruction; hence reliance on the sign of pulsus paradoxus as an index of severity or response to therapy may be misleading.

Secondly, we are surprised that the authors merely refer in passing to the use of aminophylline. The evidence that salbutamol given by IPPB or intravenously is better than intravenous aminophylline in acute asthma is tenuous, and modern treatment regimens, including if possible measurement of plasma levels, have reduced the incidence of side effects from aminophylline.[2] We feel that we are not alone in still regarding aminophylline as the bronchodilator of first choice in severe acute asthma.

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[1] Shim, S, and Williams, M H, jun, *Lancet*, 1978, **1**, 530.
[2] Mitenko, P A, and Ogilvie, R I, *New England Journal of Medicine*, 1973, **289**, 600.

SIR,—I was interested to read the article on the comparison of salbutamol given intravenously and by intravenous positive-pressure breathing (IPPB) by Dr P Bloomfield and others (31 March, p 848). However, the dose of salbutamol used was not stated except in terms of 0·5% solution being given for three minutes. There was no statement of the volume of solution nebulised during the three-minute period, and therefore no way of assessing the dose given to the patient (only about 25%[1] of the dose nebulised in fact being retained by the patient). This makes it difficult to assess their findings and impossible to compare their paper with other work in the field, and is most unhelpful for those of us who may wish to use this technique clinically.

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[1] Shenfield, G M, *et al*, *American Review of Respiratory Disease*, 1973, **108**, 501.

**We sent a copy of these letters to the authors, whose reply is printed below.—ED, *BMJ*.

SIR,—Drs Costello and Honeybourne misquote our paper, which actually said, "Our results suggest that relief of pulsus paradoxus may be a more sensitive index of improvement than simple measurements of ventilatory function." Their suggestion that pulsus paradoxus may be absent in patients with severe airways obstruction is not questioned, but it must be an uncommon occurrence since our clinical experience supports that of Knowles and Clark,[1] who found it in 80% of their patients and showed it to correlate well with severity of disease. Reliance on pulsus paradoxus as an index of severity of disease or