ticularly important. The *Sunday Times* recently[1] publicized the case of a hospital nurse, Mrs. O'Neill, and the excrementally soiled lavatory walls of which she complained. It is remarkable that the hospital secretary should be reported to have stated, as it were in defence, that the walls remained soiled for eight days rather than the 18 days claimed by Mrs. O'Neill. It is even more extraordinary that the case should be said to draw "attention to the lack of any agreed procedure for disciplining nurses." What it should draw immediate attention to is the lack of any agreed procedure for cleaning hospitals.

Part of the trouble may lie in the Crown exclusion of N.H.S. hospitals from the province of the (public) Health Inspectorate. Can we not now open the hospital services to inspection by the health inspectors of the new local authorities?—I am, etc.,

MAIR THOMAS

Elizabeth Garrett Anderson Hospital,
London N.W.1

1 Gillie, O., *Sunday Times*, 17 March, 1974, p. 6.

Coping with Nose-bleeds

SIR,—There is a surprising omission from your leading article (9 March, p. 405). The treatment of anterior epistaxis is adequately summarized, and even the use of an inflatable rubber bag in the nasal cavity is mentioned. However, posterior epistaxis, which is generally more profuse and constitutes a more serious problem in management, receives less attention. The only advice given to the general practitioner or casualty surgeon is to transfer the patient, presumably still bleeding profusely, to hospital for administration of a general anaesthetic. The use of a Foley catheter obviates the necessity for anaesthesia with the concomitant dangers of regurgitation of blood from the stomach and possible aspiration. The catheter can be easily inserted through the nasal cavity, even local anaesthesia being unnecessary, and inflated when it reaches the epipharynx. Gentle traction into the choanal opening produces an effective seal and prevents the swallowing or aspiration of blood. Transport and further management of the patient are thereby simplified and removal of his form of postnasal packing is easily performed and causes minimal patient discomfort.—I am, etc.,

IAIN W. S. MAIR

Department of Otolaryngology,
Sentralsykehuset,
Tromsø, Norway

SIR,—Epistaxis is a common problem, as your leading article states (9 March, p. 405), and in the middle-aged to elderly it is often difficult to control. In this group it is often impossible to detect a bleeding point in the accessible area of the nose. Nasal packing is not an easy operation and often badly done; it is certainly unpleasant and often distressing. For this reason I have used epsilon-aminocaproic acid or tranexamic acid by intravenous injection when the simple primary procedures of recumbency, sedation, and external compression have failed.

In my own experience of some dozens of such cases I can recall only one failure and so was surprised at the brusque dismissal of this method in your leading article. I am sorry to be unable to give learned comment on the fibrinolytic activity of the nasal mucosa, but Rasmussen has done so.[1] Nor do I feel competent to organize a satisfactory double-blind controlled trial in a busy accident and emergency department. It does, however, seem logical to employ an antifibrinolytic agent when bleeding is persistent, even in the absence of evidence of primary inhibition of fibrin formation, as it may have an effect on clot formation, which in the circumstances is clearly inadequate for whatever reason. To argue that clotting cannot be enhanced by the use of an antifibrinolytic agent seems as illogical as to contend that epistaxis is persistent because of the absence of a nasal pack.

In fact I am in the excellent company of many prostatic surgeons in finding epsilon-aminocaproic acid an excellent haemostat in awkward circumstances.—I am, etc.,

R. H. HARDY

Accident and Emergency Department,
General Hospital,
Hereford

1 Rasmussen, A. B., *Acta Otolaryngologica*, 1966, **61**, 221.

SIR,—Concerning your leading article (9 March, p. 405) on the above subject, I feel that in view of the rather involved measures described I should write to you of my experience of nose-bleeding.

About 35 years ago as a houseman I was called to a medical ward to a patient who had nose-bleeding, and on my way I contemplated my previous rather ineffective experiences of packing the nose in such cases. When I arrived I was confronted by the very experienced, very senior ward sister who announced, "Whatever the cause there is only one treatment and it never fails." A cork about an inch long is placed between the upper and lower teeth to hold the mouth open and a wide bandage is wound around the head from the chin to the top of the head, where it is knotted. This maintains gentle pressure on the cork and as the mouth is thus held open the patient is obliged to breathe through the mouth and the clotting process is left undisturbed. The nose must not be blown or sniffed. It usually takes about 15 minutes. I have a high degree of confidence in this procedure which I have used and seen used many times during these many years and I hope its use might be the means of avoiding some of the more involved procedures described.—I am, etc.,

J. H. L. NEWNHAM

Tunworth, Hants

Death during Dental Anaesthesia

SIR,—I read with interest the comment made by Dr. J. G. Bourne (16 March, p. 516) on the case of a recent death during dental anaesthesia which was reported in your medicolegal column (2 February, p. 207). I should like to report a recent fatal case of severe unaccountable collapse following administration of anaesthesia in a dental chair.

A healthy child aged 10 needed dental extractions as he was suffering from an alveolar abscess and was in pain. He had developed chickenpox two days earlier. The child was instructed to take two 10 mg trimeprazine tablets an hour before attending.

The child walked in to the dental surgery and was seated in the chair semi-upright (45°). He looked rather pale but at the time this was presumed to be due to the fact that he was in pain and had had a sleepless night. He was anaesthetized with 75% nitrous oxide, 25% oxygen, and 1·5% halothane. The concentration of halothane was later reduced to 1·0%. The respiration and colour of the child were monitored continuously and the pulse was felt intermittently. The dentist then removed the first four molar teeth. Both induction and extraction were quick and easy, together taking five minutes at the most. At the end of the procedure the child looked extremely pale and his pulse was slow and feeble. However, it was noted that he was not sweating. The chair was immediately tilted right back so that the child was in about 10° head-down tilt and his legs were held up by a nurse. External cardiac compression was commenced and the lungs inflated rhythmically with oxygen. The child was taken to hospital, where he was treated for ventricular fibrillation but improvement was transient and he died 2½ hours after the collapse. Necropsy findings were negative.

It appears that the child had a vasovagal attack. In this case the hypotensive episode or cardiovascular collapse occurred without apparent cause or warning. Once peripheral vasodilation has begun and blood pressure is falling the sitting position will increase cerebral oligaemia by gravitational pooling of blood in the dependent portions of the body and will further lead to reduced filling of the right atrium and cardiac output. There is no doubt that whatever the cause of hypotension its outcome in terms of brain damage or death of the patient is greatly influenced by the upright position traditionally used in dentistry. Though death during dental anaesthesia is rare, the difficulty in recognizing a vasovagal attack accounts for the fact that it occurs even with the most careful and experienced anaesthetists. Perhaps either by abandoning the sitting position or by adopting intensive monitoring while anaesthetizing patients in the dental chair one could avoid these occasional fatalities.—I am, etc.,

S. MEHTA

Anaesthetic Department,
Burnley General Hospital,
Burnley, Lancs

Central Nervous System Effects of Pentazocine

SIR,—We were interested to note the findings of Dr. A. J. J. Wood and others (23 February, p. 305), who investigated the effects of pentazocine on the central nervous system. We also have noted hallucinations in patients who received pentazocine in the postoperative period. However, we have found that hallucinations in association with other analgesic drugs are not uncommon. In a study of patients in the postoperative period we have compared the incidence of hallucinations following the administration of pentazocine with that following morphine.

Patients admitted for elective general surgery were allocated randomly to two groups. For postoperative pain relief those in one group were given pentazocine 40-50 mg intramuscularly and those in the other group were given morphine 10 mg intramuscularly. The frequency of administration of these drugs was at the discretion of the nursing staff. Twenty-four hours after operation the patients were questioned regarding unusual experiences since the operation, particularly whether they had seen, heard, or felt anything that seemed

strange. Patients who had received central nervous system stimulants or depressants other than the analgesic under study either before or after the operation were excluded.

There were 114 patients of whom 31 had auditory or visual hallucinations, distributed as shown in the table.

	Morphine	Pentazocine	Total
No. of patients	49	65	114
Patients experiencing hallucinations	7	24	31
Hallucinations pleasant	2	0	2
Hallucinations unpleasant	2	12	14
Neither pleasant nor unpleasant	3	12	15

These figures suggest that hallucinations after surgery occur more often in association with pentazocine than with morphine ($\chi^2 = 6{\cdot}133$; $P < 0{\cdot}05$) but we think it is important to appreciate that the problem is not exclusive to pentazocine.—We are, etc.,

J. I. ALEXANDER

United Bristol Hospials, Bristol

ALASTAIR A. SPENCE

Western Infirmary, Glasgow

Haemophilus Influenzae

SIR,—In a recent American case-report and review of the literature on *Haemophilus influenzae* cellulitis, published in a British journal, Rasmussen[1] stated that this syndrome had not been reported in the European literature. In fact, one case had been mentioned in a paper from Oxford about haemophilus epiglottitis.[2] In the light of Rasmussen's comments we wish to report two further cases of *H. influenzae* type b infection recognized in Oxford in the past few weeks.

The first patient, an 11-month-old boy, was admitted to the Nuffield Orthopaedic Centre on 4 February with an 18-hour history of irritability and a tender swelling of the right calf. Findings on admission included: temperature 38·5°C; pulse rate 130/min; W.B.C. 38,000/mm³ with marked neutrophil leucocytosis; and E.S.R. (Westergren) 90 mm in 1 hr. His right upper calf was swollen (1·25 cm greater in diameter than the left) and evident cellulitis extended upwards behind the right knee, though without any detectable joint effusion or limitation of movement. There was shotty enlargement of his right inguinal lymph nodes. He was treated with oral cloxacillin and ampicillin and was discharged from hospital after five days. Meanwhile two blood samples taken at the time of his admission had given pure cultures of a type b strain of *H. influenzae*. No bacteriological investigation of the leg lesion had been attempted.

The second patient, a 9-month-old boy, was admitted to the Oxford Eye Hospital on 28 February with a 12-hour history of swelling of his left eyelids and surrounding tissues. On admission he was febrile and generally unwell. He had marked swelling of his lids and periorbital tissues on the left side and a pseudomembranous conjunctivitis of the left eye, which was discharging pus and blood-stained watery fluid. He was thought possibly to have some orbital cellulitis. He was treated with gentamicin eye-drops for the first 24 hours and improved markedly, but after that his treatment was changed to chloramphenicol eye-drops and oral ampicillin. This was because culture of the purulent discharge had produced a light growth of *H. influenzae* type b and because his fever suggested the need for systemic antibiotic treatment. He made a rapid recovery and was sent home after four days. We cannot say with certainty that this was a case of cellulitis as his marked periorbital swelling may have been entirely due to oedema secondary to a severe conjunctivitis.

The lack of references to haemophilus cellulitis in the European literature is probably due to infrequent recognition rather than to infrequent occurence. Significantly Rasmussen (private communication) has seen several more cases since reporting his first. A similar connexion between awareness and reported incidence is clearly apparent for haemophilus epiglottitis.[2] The aetiology of the first case described here would never have been known if blood cultures had not been set up. As for our second case, the mere finding of a light growth of *H. influenzae* might have been equivocal, as it could have been due to contamination with one of the non-capsulated strains commonly present in the upper respiratory tract. Capsulated strains are far less common, and strains of capsular type b can be far more virulent. Clear recognition of the aetiology therefore depended on typing of the isolated haemophili. In this connexion we would stress the value of first demonstrating iridescence of colonies on Levinthal's or other suitable transparent agar, since non-iridescent (that is, non-capsulated) strains may give confusing reactions in agglutination tests with capsular-typing sera. It is also important to use reliable sera. We used those made by Hyland Laboratories, which we have always found reliable in agglutination and capsule-swelling tests. Batches of commercially available *H. influenzae* typing sera from another source, however, have given unsatisfactory results in our hands, and in particular have failed to detect known type b strains.

We are grateful to Mr. J. Spivey and Mr. A. Bron for permission to describe patients admitted under their care.

—We are, etc.,

K. CARTWRIGHT
D. C. TURK

Bacteriology Laboratories, Churchill Hospital and Radcliffe Infirmary, Oxford

1 Rasmussen, J., *British Journal of Dermatology*, 1973, **88**, 547.
2 Addy, M. G., Ellis, P. D. M., and Turk, D. C., *British Medical Journal*, 1972, **1**, 40.

Platform Shoe Syndrome

SIR,—I have seen a considerable number of young women with painful knees recently, some with effusions and all with tenderness over the patellar ligament. This development has paralleled the wearing of thick-soled, high-heeled, so-called "platform" shoes.

It is easy to see why, if one watches these girls walking. The knee is always flexed and the weight is thrown on to the anterior ligaments of the knee joint. Treatment is removal of the cause by insistence on the wearing of more sensible shoes.—I am, etc.,

MICHAEL WHITEHOUSE

Camberley, Surrey

Cannulation of Internal Jugular Vein

SIR,—It would be unfortunate if the report of extensive neurological damage after cannulation of the internal jugular vein by Dr. C. E. Briscoe and others (23 February, p. 314) caused serious concern about the safety of this technique. This case report implies that strong solutions of alkali (8·4%) and calcium chloride (20%) were injected more or less directly into the tissues of the neck. It is hardly surprising that severe tissue damage was found. In our large experience[1] of internal jugular vein cannulation no such incident has occurred in about 2,000 cases.

We think it worth while to stress the following points: (1) It is not merely advisable but imperative that a cannula is used which is at least 15-20 mm long. This will make dislodgement unlikely and ensure that a true central venous pressure is recorded. (2) It is advisable to use the right internal jugular vein when at all possible as it provides a straight "run" to the superior vena cava and avoids the possibility of damage to the thoracic duct. Insertion into the right side of the neck is also more easily accomplished by right-handed operators. (3) In any case where difficulty is either anticipated (hypovolaemia) or encountered the use of a guide wire (Seldinger) technique should be strongly encouraged.—We are, etc.,

B. S. JENKINS
ANTHONY CLEMENT
J. A. BELL
R. D. BRADLEY

Intensive Therapy Unit, St. Thomas's Hospital, London S.E.1

1 Bell, J. A., Bradley, R. D., and Jenkins, B. S., *Lancet*, 1973, **1**, 105.

Turner's and Noonan's Syndromes

SIR,—As one who is studying these syndromes at this institute I welcome your timely leading article (16 March, p. 470). However, I was disappointed to see that there was no reference to the otological component which is obviously an integral part of these syndromes. There is no doubt that many of these patients have an otological abnormality and our findings are in accordance with other published series. This commonly takes the form of a sensorineural hearing loss, though other types of impairment are occasionally found.[1]

My colleagues and I are preparing for publication the temporal bone findings in a 47-year-old woman with Turner's syndrome who died after craniotomy for an inoperable cerebral tumour. She had been deaf since early childhood and hearing tests revealed a long-standing sensorineural loss. These findings are to be published in full elsewhere.

If clinicians concerned with the care of patients with Turner's and Noonan's syndrome would like to refer them to us we would be pleased to carry out a complete otological and vestibular examination. In addition we would be interested in examining further temporal bones from patients with these and other chromosomal abnormalities.—I am, etc.,

GEORGE BUCHANAN

Institute of Laryngology and Otology, Gray's Inn Road, London W.C.1

1 Anderson, H., *et al.*, *Acta Oto-Laryngologica*, 1969, Suppl. no. 247.

Primary Defect in Hepatolenticular Degeneration

SIR,—I find remarkable the passage in your leading article on abiotrophies (2 March, p. 337) which refers to hepatolenticular de-