years and these had been worrying him more recently. E.C.G. showed ventricular extrasystoles, right bundle-branch block, and left axis deviation. He was started on 50 mg practolol twice a day. In January 1974 the palpitations were still persisting and his practolol dosage was increased to 100 mg twice a day.

In April 1974 he was admitted to hospital with a four-week history of pericardial pain, malaise, fever, and some dyspnoea. On examination there was a loud pericardial rub and mild cardiomegaly. E.C.G. showed atrial flutter with 2:1 block. E.S.R. was 23 mm in 1 hour. The practolol was continued and he was started on digoxin. The atrial flutter reverted, his heart size returned to normal, and he was discharged from hospital in one week on 200 mg practolol and 0·25 mg digoxin a day.

In August 1974 he was readmitted with a four-day history of colicky abdominal pain and vomiting. There was no hernia or previous abdominal surgery. He was thought to have an upper small-bowel obstruction and a laparotomy was performed. Two generations of adhesions were found. One firm, well-organized adhesion was causing obstruction distal to the duodenojejunal flexure. There were more recent diffuse, filmy adhesions over the lower 4 ft (1·2 m) of ileum and a small amount of free peritoneal fluid was present. The omentum was swollen, firm, and oedematous. These findings were unexpected and unexplained in view of the fact that there had been no previous laparotomies and no source of peritonitis was found. An omental biopsy showed rather prominent vessels and fairly numerous clumps of lymphocytes both on the surface and deeper within the omentum suggestive of chronic inflammation. An E.S.R. four weeks after surgery was 57 mm in 1 hour. He was started on prednisolone and two weeks later the E.S.R. had fallen to 6 mm in 1 hour. Practolol has been discontinued and he is now feeling well.—We are, etc.,

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1 Proctor, C. R., *Interim Report on Adverse Reactions Associated with Practolol*. I.C.I. Australia Ltd., 1974.

Uticillin

SIR,—In their letter Drs. H. R. Ingham and J. B. Selkon (9 November, p. 344) comment on the unjustifiability of testing the sensitivity of organisms isolated from the urinary tract to carfecillin (Uticillin), since this is an oral compound absorbed and hydrolysed to the active carbenicillin.

We began testing such isolates with both carbenicillin and carfecillin discs each of 100 μg and during the first two weeks encountered two strains of *Escherichia coli* which were apparently sensitive to carfecillin but resistant to carbenicillin. Further testing by tube dilution methods showed that both these strains had minimum inhibitory concentrations to carbenicillin of greater than 800 mg/l (μg/ml) and one would have expected from this that though high-dosage carbenicillin may have been effective in therapy carfecillin, with a manufacturers' stated achievable concentration in the urine of the order of 800 mg/ml, would not.

These preliminary findings suggest that testing isolates from urinary tract infections for carfecillin sensitivity is not only unjustifiable but also unreliable and misleading, and that carfecillin may have antibacterial properties which are not shared by carbenicillin.

Your correspondents have expressed the arguments for retaining carbenicillin for *Pseudomonas pyocyanea* infections and few would disagree with these views. These arguments, our findings of *E. coli* strains showing a high degree of resistance to carbenicillin, and the failure of carfecillin disc testing to detect these suggest that it would be reasonable for carfecillin to be reserved for the treatment of pseudomonas infections of the urinary tract in patients for whom oral therapy is suitable and from whom organisms have been isolated which have been shown by adequate laboratory testing for carbenicillin sensitivity that such therapy is likely to succeed.—We are, etc.,

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Visual Evoked Potentials and Transient Ischaemic Attacks

SIR,—Transient ischaemic attacks are not usually associated with permanent brain damage. Brain scan, E.E.G., and clinical examination are generally normal after an attack. We have correlated the findings of visual evoked potential (V.E.P.) recordings with the results of conventional E.E.G. to find out if the former are more sensitive than the latter in giving information about brain lesions in these cases.

Twelve men and eight women aged from 39 to 62 years (mean 50 years) who suffered from transient cerebral ischaemic attacks were studied. In 11 of the patients the attacks were internal carotid in origin and in nine vertebro-basilar. A diagnosis of transient ischaemic attack was made only when the symptoms lasted for less than 24 hours, and carotid and vertebrobasilar cases were differentiated according to the criteria adopted by the National Institute of Neurological Diseases and Blindness.[1] The E.E.G.s and V.E.P.s were recorded on average 20 days after the last attack, 10 of them within 20 days of it. The V.E.P.s were recorded at the beginning of the routine E.E.G., the E.E.G. activity being monitored simultaneously. During the recording the patients were lying in a dimly-lit room with closed eyes. They received 100 successive flash stimuli with a frequency of 2 Hz. The duration of a flash was 1 ms and the intensity 0·42 J. The analysis time was 500 ms. The stroboscope was placed in the midline 30 cm above the eyes. The derivations used were frontal, central, parietal, and occipital referred to the ipsilateral ear. On the basis of our own control material and the findings of others[2 3] V.E.P.s were judged abnormal by the following criteria: (1) no single deflection before 80 ms after the stimulus (complex I); (2) no biphasic deflection between 80-300 ms after the stimulus (complex II); (3) unilateral lack of a V.E.P. deflection recorded in the homologous brain area; (4) amplitude difference between homologous deflections, the lower potential complex being less than 50% of the higher complex amplitude; and (5) unilateral total lack of the rhythmic after-activity.

As is evident from the results (see table) the V.E.P.s were more informative than the E.E.G., especially during the first three weeks after the attack. In the cases of hemispheral attacks in which the laterization of the ischaemia could be determined the abnormal V.E.P.s were on the ischaemic side. It seems that transient ischaemic attacks may be followed, at least in some cases, by permanent brain tissue damage but that the lesion, either because of its small size or its location in a "silent" region, may escape detection in a clinical examination. The V.E.P. recording seems to be more sensitive than the E.E.G. in detecting possible brain damage in these cases.—We are, etc.,

Findings of E.E.F. and V.E.P. Examinations in 20 Patients Related to Interval between Examination and Attack

Interval (days)	No. with Focal or Lateralizing E.E.G.	No. with Abnormal V.E.P.
<21 days (n = 10)	4 (40%)	10 (100%)
⩾21 days (n = 10)	3 (30%)	5 (50%)

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1 National Institute of Neurological Diseases and Blindness, *Neurology*, 1958, **8**, 405.
2 Ciganek, L., *Electroencephalography and Clinical Neurophysiology*, 1961, **13**, 165.
3 Kooi, K. A., and Bagchi, B. K., *Annals of the New York Academy of Sciences*, 1964, **112**, 254.

Deaths during Dentistry

SIR,—In answer to Dr. P. J. Tomlin (14 December, p. 655), let me first make clear that when using the terms "faint" or "fainting" I refer to nothing other than the common fainting attack.[1] Dr. Tomlin rightly values facts above opinions, but in the case under dispute he has been somewhat selective about the facts he discloses, restricting himself to quotations from the pathologist's necropsy report. He makes no mention of the clinical facts. It is the clinical facts attending the administration leading up to the cardiac arrest that are usually the more informative ones when a death has occurred with an anaesthetic and necropsy fails to reveal a specific cause. It was these facts in this case that led the anaesthetist, the pathologist, and me to attribute the death to fainting, the pathologist believing with me that the faint probably came on at the start of induction, immediately after the application of the nasal mask. Briefly, the clinical facts were as follows.

The boy was anaesthetized sitting up by one of the most experienced dental anaesthetists in the country. Induction seemed uneventful. He went under smoothly, there was no difficulty with the breathing, no asphyxia, no shortage of oxygen, and no mistake in the administration of the anaesthetic. Two deciduous teeth were quickly and easily extracted, the mask was removed, and the head was held forward for recovery. There was noticeably little bleeding and there was concern over delay in recovery; cold water was splashed on his face, but there was no response. Then suddenly, unaccountably, there was cardiac arrest.

The case exactly matches other reported cases[2 3] in which young, healthy patients collapsed and died in the dental chair under anaesthetics—given in several of the cases by consultant anaesthetists. It also matches a case that occurred at my own hands[4 5] in a man who fainted during induction while his blood pressure was being continuously recorded. He was being closely watched by five of us, four doctors and a dentist. None