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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J. L. FREW

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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 hemispherical attacks in which the lateralization of the ischaemia could be determined the abnormal V.E.P.s were on the ischaemic

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%)

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.,

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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

of us spotted that he had fainted; we all mistook the faint for the onset of smooth anaesthesia, and but for a warning cried out by the technician who was following the blood pressure on the recording apparatus the patient might well have died.

In the boy's case outlined above, Dr. Tomlin (2 November, p. 288) somewhat surprisingly attributes the death to pulmonary oedema. I have pointed out⁵ that in cases of sudden collapse and death in the dental chair when fainting was the only rational explanation pulmonary oedema seems to be a constant necropsy finding. Indeed, Dr. Tomlin himself has reported this finding.⁶ The case was that of a woman aged 22 sitting up in the dental chair who lost consciousness and collapsed during the injection within the mouth of 1.5 ml of a standard local analgesic solution. She died and "the post-mortem revealed acute pulmonary oedema." Discussing the cause of this death, Dr. Tomlin makes no mention of the pulmonary oedema. He attributes the death either to acute sensitivity to the analgesic agent or to "a severe dysrhythmia or a 'faint'."—I am, etc.,

J. G. BOURNE

Salisbury, Wilts

- ¹ Bourne, J. G., *Studies in Anaesthesia*, p. 131. London, Lloyd-Luke, 1967.
- ² Bourne, J. G., *Anaesthesia*, 1970, 25, 473.
- ³ Bourne, J. G., *Lancet*, 1973, 1, 35.
- ⁴ Bourne, J. G., *Lancet*, 1957, 2, 499.
- ⁵ Bourne, J. G., *Lancet*, 1966, 1, 879.
- ⁶ Tomlin, P. J., *Anaesthesia*, 1974, 29, 551.

John Locke

SIR,—I was interested to read Mrs. Hilda M. Stowell's letter (30 November, p. 530) about my article on John Locke (5 October, p. 34). I am sorry if one sentence in my article gave the impression that Locke was in exile for the whole period 1660-89. In fact there is nothing in the article to suggest that. Locke followed Shaftesbury into exile in 1683 (that is, during Charles II's reign), lay low to avoid association with those involved in Monmouth's rebellion, and returned with William III in 1688. My article refers to Locke's five years in the Netherlands, which makes it clear that he went there in 1683.

If Mrs. Stowell cares to read my book on Locke¹ she will, I think, find little to quarrel with.—I am, etc.,

M. V. C. JEFFREYS

Lyndhurst, Hants

- ¹ Jeffreys, M. V. C., *John Locke; Prophet of Common Sense*. London, Methuen, 1967.

Imported Diseases

SIR,—The recent articles on imported diseases are useful and point to some of the possible causes. But articles of this kind, designed for the general reader, must take especial care to inform and not to misinform. I must therefore take issue with Dr. A. M. Geddes (23 November, p. 454) on several points.

A paragraph is devoted to Lassa fever. This is far from common, even in Africa, and few in Britain have seen even a single case of this highly infectious, distressing, and often fatal disease. The same amount of space is given to dengue fever, but no mention at all is made of the multitude of other viruses transmitted by mosquitoes,

sandflies, or ticks and which are known to be responsible for disease in man (usually fever but sometimes more severe manifestations such as encephalitis) not only in tropical and subtropical regions but also in areas as close to Britain as the south of France, Italy, and Cyprus. Viruses of this type are also known to be active in Scandinavia, Austria, and Portugal. Details of these viruses are probably a matter for the specialist, but their existence should be recognized by all practitioners.

Diagnosis in the cases of Lassa, dengue, and yellow fevers is dismissed rather cavalierly, as "confirmed by serum antibody studies." This is so, but the pertinent question is—where can these be done? To my knowledge there is no virus laboratory in the United Kingdom where a service is available for the routine diagnosis of arbovirus infections. This is a small but important lacuna and one which it would be relatively inexpensive to fill.—I am, etc.,

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Medical Nemesis

SIR,—The gist of your leading article on Ivan Illich's *Medical Nemesis*¹ (7 December, p. 548) is that, while clearly much is wrong with medicine, there is nothing that doctors and other citizens cannot set to rights, that Illich is a somewhat wild man, if interesting, and that one cannot put the clock back. Of the three reviewers of his book (7 December, p. 573) one, Dr. A. Paton, gracefully accepts almost the whole Illichian thesis and two reject much of it.

Professor G. Discombe makes four chief points. First, that Illich is often obscure; agreed. Second, that he is talking mostly about American medicine, to which the right answer is that increasingly American medicine is the kind that dominates the West and its outposts in underdeveloped countries. (Professor Discombe is, I am sure, aware that the U.S.A. is importing some 4000 doctors a year, many from underdeveloped countries, and at a time when the American male's expectation of life at birth is falling). Third, he seems to think that Illich would disapprove of the removal by means of the products of Western medical technology of "the shackles of ignorance, of disease, and of starvation from which the third world is trying to escape." In fact, I think, Illich would disapprove only if the price of such removal were to be a take-over of the indigenous culture by Western technology. After all, it is Professor Discombe, not Illich, who says, "In an African town or village most people seem to be fairly happy and contented. But appearance is no guide to the load of sickness"—to which Illich would add that if he *had* to choose between destroying the load of sickness and perpetuating that of happiness he would choose the latter. He has no fear of the barefoot-doctor approach (or of alternative technology generally), only of its practitioners learning from doctors to professionalize themselves by means of a College of Barefoot Doctoring. Fourth, Professor Discombe thinks Illich an enthusiastic romantic—that is, that Illich is not a realist.

Professor P. Rhodes, the third reviewer,

adds various points—for instance, that "many would reject the thesis that pain, sickness, and death are to be welcomed." If what is meant is all pain, sickness, and death, then Illich would be one of the many. He says (*Medical Nemesis*, p. 121), "De-professionalization does not mean the abolition of modern medicine . . . [or] disregard for the special needs which people manifest at special moments in their lives: when they are born, break a leg, become crippled or face death." Professor Rhodes thinks "no man is an island," but believes that Illich wants man to be just that: Illich thinks industrial man is an island and that no man ever should be. Finally, Professor Rhodes too thinks Illich offers as a solution a retracing of our steps: "his solution is now not possible if it ever was."

One common thread is clearly that Illich is not a realist (unlike doctors). As your reviewers and your leading article indicate, Illich regards medical nemesis as a part of a more generalized industrial nemesis, and it cannot be understood except in that larger context. As an unashamed romantic—in the Illichian mode—I think Illich is not a prophet of industrial (or medical) nemesis: like the rest of us, he is now a witness of its occurrence. The clock is visibly going back. In what manner we should start going "forward" again—when that becomes possible—is perhaps the major question of our time. Illich supplies an answer to it.—I am, etc.,

JOHN S. BRADSHAW

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- ¹ Illich, I., *Medical Nemesis*. London, Calder and Boyars, 1974.

Vitamin D Deficiency in Rheumatoid Arthritis

SIR,—Drs. P. J. Maddison and P. A. Bacon (23 November, p. 433) omitted to give adequate details of the drug history in their rheumatoid arthritic patients who had clinical and biochemical evidence of osteomalacia. If they had been receiving long-term mild analgesics and anti-inflammatory drug therapy, I wonder if the authors had considered the role of a drug-induced disturbance of vitamin D metabolism in the aetiology of their patients' bone disease?

There is now considerable evidence that long-term treatment with anticonvulsants can disturb the hepatic metabolism of this vitamin, probably by virtue of their powerful microsomal enzyme inducing properties.¹⁻³ It is known that many mild analgesic drugs have a similar effect on liver enzymes,⁴ and their chronic administration could therefore create a state of increased turnover of the vitamin in patients whose dietary intake and exposure to sunlight are already below average.

Measurements of urinary D-glucaric acid excretion⁵ or plasma antipyrine or quinine half lives,^{6,7} which are indices of hepatic enzyme induction, would be interesting in these patients.—I am, etc.,

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- ¹ Richens, A., and Rowe, D. F. J., *British Medical Journal*, 1970, 4, 73.
- ² *Lancet*, 1972, 2, 805.