Sex	Age (Years)	Peak Acid Output (mEq/hr)		$\frac{\text{P.A.O. I}}{\text{P.A.O. Hg}} \times 100$
		Histalog	Insulin	
F	34	18.5	0.88	5
M	66	1.24	0	0
M	63	17.58	9.54	54

results of gastric secretion tests⁸ are shown in the table.

These results could be interpreted as showing absence of impairment of vagal innervation of the stomach in these three patients with peripheral and autonomic neuropathy. Kassander⁹ had originally suggested that "a diabetic patient at some point in the progression of his diabetes may 'vagotomize' himself." Myelin degeneration has been seen in the vagal nerves of diabetics as well as in their peripheral nerves.¹⁰ Taken together these histological abnormalities¹⁰ and the gastric secretory studies^{5, 7, 8} support this concept of the diabetic vagotomizing himself,⁹ analogous to achalasia of the oesophagus. In 32 patients with achalasia vagal nerve function was considered absent in eight because of failure of acid response to insulin but adequate response to maximum Histalog stimulation.¹¹ Autovagotomy should be considered as part of a spectrum of diabetic autonomic neuropathy, so that it is not surprising that the incidence of duodenal ulcer in diabetics is only one-third of that of the normal population.¹¹—I am, etc.,

J. H. BARON

Department of Surgery,
Royal Postgraduate Medical School,
Hammersmith Hospital,
London W.12

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Death after E.C.T.

SIR,—In her report of the death of a 55-year-old woman following electric convulsion therapy (E.C.T.) Dr. Joan R. Gomez (6 July, p. 45) raises three points deserving consideration.

(1) The dangers of "cardiotoxic drugs" such as the tricyclic antidepressants used in conjunction with E.C.T. must be weighed against their advantages in preventing the fairly high incidence of early relapse (with risk of suicide) in patients treated with E.C.T. alone,^{1, 2} particularly as giving additional E.C.T. after symptomatic relief has not been shown to prevent relapse.² A number of studies do, however, suggest that concomitant tricyclic administration reduces the incidence of relapse.^{3, 4}

(2) Many investigators have commented unfavourably on the dosage and mode of atropine administration prior to E.C.T.⁵

Though Rich and Pitts,⁶ in a series of over 1,500 E.C.G.-monitored E.C.T.s, failed to demonstrate "anything approaching a life-threatening vagal-induced arrhythmia" using 0.65-2.5 mg atropine by the subcutaneous route, pharmacological evidence suggests that the commonly used dosage of atropine—that is, 0.65 mg—is of placebo value only in preventing vagal inhibition.

(3) Acute anxiety resulting in sympathetic/parasympathetic stimulation can undoubtedly be responsible for cardiac arrhythmias; some anaesthetists in general surgical and dental practice are using such compounds as lorazepam intramuscularly as sole premedication with satisfactory results. It should be remembered, however, that most currently available effective anxiolytics also have anticonvulsant properties which might be antagonistic to the convulsant stimulus.—I am etc.

JOHN L. BARTON

Department of Psychiatry,
University of Leeds

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SIR—I was most interested by Dr. Joan R. Gomez's letter (6 July, p. 45) reporting the death of a woman apparently from vagal inhibition due to the combination of electric convulsion therapy (E.C.T.) and amitriptyline and by her suggestion that the standard premedicating dose of atropine (0.6 mg) is inadequate. After I had had a couple of nasty experiences (a cardiac arrest in a grossly obese man who was fortunately easily resuscitated and a profound bradycardia in a young, apparently healthy man) I made it my standard practice to give atropine 1.2 mg intramuscularly at least an hour before E.C.T. and with this regimen I found no further problems of this type, though I was averaging 50-60 E.C.T.s a week in a predominantly middle-aged and elderly population. Since peripheral circulation is often relatively impaired in patients under stress, it would be interesting to know by which route atropine was given to Dr. Gomez's patient and how long before the fatal E.C.T. I think one reason why the 0.6 mg dose remains standard is because nearly all of us, doctors and nurses alike, tend to think of "drying up secretions" as being the main reason for giving atropine. Actually, if one stops to think logically about it, it is obvious that if one passes an electric current through the brain all the cranial nerves will be overstimulated and therefore one should use a dose of atropine sufficient to paralyse the vagus in order to avoid cardiac arrest.

Regarding Dr. Gomez's last question, surely the usual effect of anxiety on the

heart is to produce a tachycardia, but it would be reasonable to suppose that this in itself may make the heart more vulnerable to further stimuli and also it is possible that the tachycardia might stimulate the vagus in an attempt at homeostasis. In any case, as the patient was 55 at the time of her re-admission it is probable that the E.C.T. she received for her puerperal illness would have been given without anaesthesia and so she may well have been far more apprehensive than are most patients prior to their first E.C.T.—I am, etc.,

PAULA H. GOSLING

Craig Phadrig Hospital,
Inverness

Tests of Hearing in School

SIR,—In your leading article (6 July, p. 3) doubt is expressed about school screen audiometric tests and it is suggested that perhaps impedance measurements for middle ear function could replace them. The article also gives incorrect information about the standard procedures.

School screen audiometry is carried out at 20-dB levels, which is now the generally accepted standard, and all frequencies from 250 to 8,000 c/sec should be tested. Children should be tested as soon as possible after entry into the infants' school and again at the junior stage, but it is desirable to test all children, if at all possible, three times during their stay in the primary school. The value of this test is amply confirmed by direct experience from areas where school screen audiometry is practised properly. Extensive literature is available which clearly shows the importance of school screen audiometry, which is one of the most valuable methods of detecting children with previously unknown hearing losses, both conductive and sensorineural. Without this test many children with hearing losses would remain undetected.

From time to time, in order to check the results of school screen audiometry, groups of children who have failed the screen test and subsequent threshold audiometry test are examined by me at audiology clinics. Recently, in one group of 36 children I found 21 with a conductive loss and two with a sensorineural one; four had wax in their ears which needed syringing and nine had normal hearing. Of those with conductive loss, only three had already had E.N.T. treatment; six were referred by us for treatment for serous otitis media; seven needed further investigation and almost all of them needed some help and were visited at the school by a peripatetic teacher of the deaf. Of the two with a sensorineural hearing loss, one had a moderate high frequency one and one almost total unilateral deafness which had so far been undetected. Of the nine children with normal hearing, several had histories of variable conductive losses and needed further follow-up. Other checks have given similar results.

There is no doubt whatsoever that any school health service without a properly organized audiometry programme neglects a very important aspect of school child health. The suggestion that impedance measurements should replace school screen audiometry reveals a lack of understanding of the functions of these tests. Many children are detected by school screen audiometry who would not be detected by the impedance

test. On the other hand, many who have symptomless mild or transient serous otitis media would be detected. What should be done with them? Sent to E.N.T. departments for myringotomy and insertion of grommets? Of course, most otologists would consider this as preposterous. We have difficulty at present in getting children with significant hearing losses examined and treated within a reasonably short time when we think treatment is urgent.

Impedance testing and school screen audiometry are complementary. There is some overlap between the two, but their functions are different. First of all we must consider the time taken for a screen test. It takes one minute per ear to test by screen audiometry. For the impedance test one needs five minutes per ear. A skilled audiometrician can screen out 40-60 pupils in about two hours. For the same number of children impedance testing would take 10 hours.

To criticize the screen audiometric test on the grounds that it is badly performed in some areas or that it is performed by untrained and unskilled people is not an argument against the test itself. It is to be hoped that your readers, particularly those involved with school health services, will not be influenced by this unhelpful and misleading article.—I am, etc.,

L. FISCH

Institute of Laryngology and Otology,
London W.C.1

Contaminated β -Lactamase and Blood Cultures

SIR,—The letter from Mr. R. Lynn and Dr. Susannah Eykyn (6 July, p. 46) is of considerable interest since their finding that blood cultures have become contaminated by diagnostic additives is of obvious concern. However, we were disturbed by the final sentence of their letter, which states that the enzyme preparation is now filtered through a Millipore filter. In the light of our previous experiences with β -lactamases, especially that of *Bacillus cereus* 569/H, it occurred to us that such a procedure could lead to removal of enzymatic activity. This enzyme is notorious for the avidity with which it adsorbs to surfaces such as glass powder¹ and Celite (diatomaceous silica).² It seemed to us possible that the enzyme would also adsorb to the material of the Millipore filter (a mixture of cellulose acetate and nitrate).

The Whatman β -lactamase preparation is not in routine use in this laboratory, but we had been fortunate enough to receive a sample vial (batch 3) from the manufacturers. The contents were dissolved in 10 ml of M/30 Sørensen's phosphate buffer, pH 7, and half of the solution was passed through a filter of pore size 0.22 μ m (Millex disposable filter unit, Millipore Ltd.), using minimum pressure. The unfiltered and filtered portions were then assayed for β -lactamase activity against benzylpenicillin (for total activity) and oxacillin (for β -lactamase II activity³), each substrate separately at 2 mg/ml, using hydroxylamine assay.⁴ We found that passage through the filter removed all but 0.7% of the total activity. β -Lactamase II activity was less affected, 3.6% remaining after filtration; however, β -lactamase II makes up only 2.5%

of the original total activity of this particular batch of Whatman β -lactamase.

Thus passage of the enzyme through the filter may have the effect of removing almost totally the active principle of this preparation. Several other commercially available β -lactamase preparations⁵ are also derived from *B. cereus*, and filtration of these might also be expected to result in loss of activity. Therefore the investigator has two possibilities. Firstly, to accept that the enzyme preparation is sterile, or secondly, to question the practice of adding β -lactamases to blood cultures media routinely. In view of the well-known lability of the β -lactam moiety to sulphhydryl groups and in reducing conditions⁶ such as obtain in media used for the culture of anaerobes, β -lactamase may be unnecessary. Timing of the blood culture in relation to the last dose may enable the use of penicillinase to be avoided where the concentration of penicillin has fallen to a low level. On dilution this would become insignificant. Studies to settle this important question would be worth while.—We are, etc.,

J. M. T. HAMILTON-MILLER
W. BRUMFITT

Department of Medical Microbiology,
Royal Free Hospital,
London W.C.1

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Sun, Wind, and the Skin

SIR,—Further to your timely leading article (13 July, p. 72), it would seem clear that the aggravation of sunburn by wind is largely, if not entirely, due to the desiccating action of the wind on the horny layer of the epidermis on all exposed surfaces. This takes place irrespective of the environmental temperature so that skiers as well as soldiers wearing the professional military garb of shirt and shorts (which is entirely unsuitable for desert wear,^{1,2} unlike the excellent protection provided by the Bedouin burnous and Arab headdress against sun, wind, and sand) can be affected. These are almost perfectly adapted to hot desert conditions.^{1,2} The holiday-maker similarly clad and, even more so, the girl in her picot-bikini find themselves in a climate little different from that of an arid desert when a fresh sea breeze is blowing.

Ichthyotic infants and atopic children with lichenification of the hands, elbows, knees, ankles, and/or face are particularly vulnerable in this respect and should therefore wear light jeans and long-sleeved cotton shirts or blouses to reduce the risk of windburn on the sea shore. Sea bathing itself is of course to be encouraged in these patients, who will benefit from the high salt content of sea water. This helps to combine water more effectively to the keratin of the horny layer, thereby reducing loss of water through evaporation.

There is another cause of sunburn of which many yachtsmen may not be aware. The burn occurs when a cotton shirt or other garment becomes saturated with sea

spray through which the shorter wavelengths of the sunlight, excluding the ultra-violet, can penetrate and damage skin. It should be remembered that water tends to absorb the longer wavelengths of the invisible spectrum as well as the infrared and to allow the shorter waves to penetrate further. Therefore in these conditions yachtsmen should wear plastic garments impenetrable by the sun's rays.—I am, etc.,

E. J. MOYNAHAN

Hospital for Sick Children,
Great Ormond Street,
London W.C.1

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Who is the Dental Anaesthetist of the Future?

SIR,—The remarks attributed to an operator/anaesthetist recently publicized in the national press following another patient fatality (see medicolegal report, p. 419) make one wonder when the powers that be will say, "Enough!" It is a fact that the British Dental Association¹ has cautioned the dental profession as to possible legal repercussions of using operator/anaesthetist technique—but no more. It is another fact that the Joint Sub-Committee on Dental Anaesthesia of 1967² deprecated the practice of dentists giving general anaesthetics alone. It is also a regrettable fact that the then Secretary of State decided not to implement the recommendations of this subcommittee because he "did not wish to interfere with the clinical freedom of dentists."³

Two professional people must be the bare minimum for any dental work done under general anaesthesia, a fully-trained anaesthetist ideally being one of these. "Dental anaesthetics is one of the most difficult branches of the medical specialty of anaesthesia" (Flt. Lt. D. Hogg, 18 May, p. 386). If the professional bodies will not stop this dubious and uniquely exercised prerogative of the dental profession in accepting the operator/anaesthetist, then perhaps the medical defence organizations should cease to provide cover for the operator/anaesthetist. Must we wait for litigation?—I am, etc.,

T. E. MCEWAN

Eastman Dental Hospital,
London W.C.1

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Reporting Deaths to the Coroner

SIR,—Dr. Harold Price (20 July, p. 171) writes that "the doctor is obliged by common law to report all sudden and unexpected deaths . . ." etc. I feel that this statement requires an important proviso before it can be regarded as accurate. The Brodrick Report¹ states (para. 12.02 (iv)) that "there is an obligation at common law on 'any person about the deceased' to give immediate notice to the coroner of circumstances requiring the holding of an inquest" and (para. 12.02 (v)) that "there is no duty on doctors to report any death to the police