treatment. Naturally, because replacements are not allowed in a match below representative grade, the team doctor is expected to apply a hasty second-aid Elastoplast strapping and even a local anaesthetic to mask the agony. Admittedly, the player reappears of his own free will, but he knows well that if he does not, his team mates must battle on, with one position unfilled. His courage gets the better of his common sense, and the situation impedes the giving or accepting of sound medical judgement. Many doctors associated with clubs and teams throughout the whole rugby world must be familiar with cases of players who aggravate an injury by resuming exercise too soon and thus, ensure for themselves a long painful lay-off. It is well recognized that all this is further bedevilled by the all-round increase in fixtures and player engagement, which rarely leaves more than a couple of days to shake off the effects of even trivial trauma. Replacements will be more widely accepted in the future. Doctors and consultants, who are close to the problem, could hasten the day by using their influence in the corridors of rugby power.—I am, etc.,

FREDERICK C. DWYER,
President,
Devon Rugby Football Union

Plymouth

Bacteroides Infections

SIR,—In your epidemiological notes (17 March, p. 686) reference is made to reports gathered by the Public Health Laboratory Service of the isolation by blood culture of species of *Bacteroides* from 76 patients during the last 9½ months of 1972. This is the greatest concentration of such infections yet reported in the United Kingdom and the Irish Republic and it follows other recent accounts, such as that of six patients treated in Glasgow in 1972.¹

Between 1933 and 1944 I isolated organisms of the genus *Bacteroides* from 12 patients in hospitals in North London, and records of these were published in 1955,² with an analysis of 268 other cases in the literature from eight countries. From 1944 to 1965 no new example of the infection was found in the same hospitals, although looked for. The organisms are known to be common saprophytes of the human gastrointestinal tract and are relatively easily cultivated. From my own experience I think that the variation in the reported incidence of infection is more probably due to altered opportunities for the invasion of the tissues than to varying attention of bacteriologists to the possibility. The bacteraemia in 1972 "was most commonly reported in elderly patients, most of whom were known to have an underlying illness." In the records of 20 years ago² the patients were of all ages from childhood onwards and the infection was more often the primary and sole disease in the form of extensive and often multiple suppuration.

The bacteroides are the cause of many lesions in wild and domestic animals and reach human beings by contact and probably by consumption of infected animal tissues. Among the 280 cases which I analysed there were four main groups. One was of infection of the skin or subcutaneous tissues by way of civilian injuries or war wounds, or directly from live animals or their

tissues in the case of farm workers and butchers. A second group, the largest, was of infections which started in the throat, after tonsillectomy or with sore throat or otitis media as the first symptom. Thirdly, there were many infections by way of the female genital tract, mostly after abortion or in the puerperium, and fewer, in either sex, by the alimentary tract, sometimes after surgical operation, or by the urinary tract. The fourth group showed empyema, with or without lung abscess, as the first symptom. When lung abscess preceded empyema it was thought that inhalation from the mouth or throat might be the origin.—I am, etc.,

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- 2 Alston, J. M., *British Medical Journal*, 1955, 2, 1524.

Zollinger-Ellison Syndrome Reclassified

SIR,—A number of recent papers in journals without correspondence columns have complicated the picture of the Zollinger-Ellison considerably and, despite your excellent leading article (6 January, p. 2), I find certain of my colleagues and most medical students thoroughly confused.

The syndrome to which Zollinger and Ellison gave their name¹ (though they were not the first to describe cases) had a diagnostic triad of (1) fulminating peptic ulceration (often multiple, recurrent, and atypically situated), (2) gigantic gastric acid secretion, and (3) non-insulin-secreting islet cell tumours of the pancreas. It was subsequently recognized that the tumours could occur in ectopic pancreatic tissue,² the substance they secreted was identified as gastrin,^{3,4} and eventually the secretory cells were shown to be D cells.⁵ The picture thus far is clear.

Unfortunately it was soon shown that the clinical features of the syndrome could be caused by mere hypertrophy of the pancreatic islets,² and more recently Polak and her colleagues showed that profound antral G cell (that is, gastrin-secreting cell⁶) hyperplasia could produce a similar picture.⁷ The latter divided patients into a group with hyperplasia or frank tumour in the pancreatic islets and a group with G cell hyperplasia and a normal pancreas. Last year, resection of a gastric tumour containing gastrin and appearing to arise from antral G cells from a case of "clinical" Zollinger-Ellison syndrome was reported.⁸

Diarrhoea was described early in certain cases of the Zollinger-Ellison syndrome¹ and has been variously attributed to hypersecretion by the small intestine, due to the direct action of excess acid;⁹ increased secretion of pancreatic enzymes;² and steatorrhoea, which undoubtedly occurs in many cases and is almost certainly due to irreversible inactivation of pancreatic lipase by the low jejunal pH, though dilution of bile acids and precipitation of glycine dihydroxyconjugated bile acids may play a part.¹⁰ A few cases have obvious causative anatomical lesions.² Two patients with intractable diarrhoea associated with non-β cell islet cell tumours of the pancreas were described in 1958 by Verner and Morrison¹¹ and further examples were discussed by

Telling and Smiddy,¹² who suggested that they were dealing with a situation different from classical Zollinger-Ellison syndrome. Howe² described more cases and even documented the lack of associated gastric hypersecretion (achlorhydria in many) and peptic ulceration, but merely classified them as a variant of the Zollinger-Ellison syndrome. I have found that certain of my colleagues still hold to this view. I, however, would agree with your leading article that these cases should be classified as a completely separate entity, since the only feature they have in common with "proper" cases of the Zollinger-Ellison syndrome is non-β cell tumours of the pancreas, which do not even secrete gastrin (it has been suggested that they produce gastric inhibitory polypeptide¹³), and some have been reported to respond to steroids,⁹ which are of no use in "proper" cases. The suggested name of W.D.H.W. syndrome⁹ seems to me as good as any, especially as the letters remind one of the cardinal features of the condition.

I should like to put forward a tentative classification of the spectrum of Zollinger-Ellison syndrome cases encountered. As the syndrome which Zollinger and Ellison actually documented included pancreatic lesions I suggest that cases of D cell hyperplasia be termed type I Z-E syndrome and those with tumours, whether in pancreas proper or ectopic pancreatic tissue, be termed type II. The cases with gastric lesions could conveniently be termed pseudo-Z-E syndrome type I (G cell hyperplasia) and type II (tumour of G cells). Cases with the clinical features of classical Zollinger-Ellison syndrome and undefined pathology would remain unclassifiable.

It is to my mind important to attempt some sort of classification in view of the different treatments applicable to the different types (for example, vagotomy and antrectomy for pseudo-Z-E syndrome type I¹³). I should be interested to see readers' comments.—I am, etc.,

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- 1 Zollinger, R. M., and Ellison, E. H., *Annals of Surgery*, 1955, 142, 709.
- 2 Howe, C. T., *Scottish Medical Journal*, 1965, 10, 307.
- 3 Gregory, R. A., Grossman, M. I., Tracy, H. J., and Bentley, P. H., *Lancet*, 1967, 2, 543.
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- 7 Polak, J. M., Stagg, B., and Pearse, A. G. E., *Gut*, 1972, 13, 501.
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- 13 Pedersen, R. A., and Brown, J. C., *Gastroenterology*, 1972, 62, 393.

Results of Renal Transplantation

SIR,—We are grateful to Dr. S. C. Farrow and his colleagues (31 March, p. 800) for pointing out an error in our results. One of the patients who rejected was in fact omitted in error from the published actuarial survival curve. In addition, one of our patients rejected at the end of 12 months and Dr.

Farrow has set down this patient as not surviving for one year. In our data we have included her as a one-year survival. Accepting these differences our one-and-two-year survival figures would be 82% and 80% instead of our published figures of 85% and 82%. In fact, 10 months have now passed since the figures were compiled, and one further graft has been lost (one of the live-donor transplants). All of the remaining grafts are still functioning and have done so for at least 12 months, giving an actual overall one-year survival of 85%. If the cadaver transplants are taken alone, then the comparable figure is 82%. Two-year graft survival figures calculated on the actuarial method of Barnes will give us an overall two-year survival of 80% based on 12 of the 33 grafts which have now passed the two-year mark. The mortality for this group remains at 6%.

While we have no doubt that Dr. Farrow's method of predicting graft survival may be valid, it requires time to be tested. It also suffers from the disadvantage that it compares individual results with pooled data. As we all know, pooled transplant data suffer from a number of disadvantages, including the fact that immunosuppression, tissue typing, etc. vary considerably from centre to centre. Barnes's method, in contrast, does not suffer from these disadvantages and has stood the test of time, something which Dr. Farrow's method has yet to do. In this connexion it may be relevant to state that according to his computer our projected one-year cadaver graft survival should be 71% when in fact the actual survival is 82% for this group of patients. Although an 11% error may not seem much, it means a great deal when the case for and against transplantation is being argued.—We are, etc.,

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Malaria Reference Laboratory

SIR,—As mentioned in the *B.M.J.* (31 March, p. 811) the Malaria Reference Laboratory has been transferred from its previous location at Horton Hospital, Epsom, Surrey, to the London School of Hygiene and Tropical Medicine. This move marks the end of a historic period and calls for a recollection of the role played by this unit for almost half a century.

In 1917-18 many thousands of soldiers and sailors returning home from war service in the tropics had periodic relapses of malaria contracted abroad. In south-east England the infection was transmitted to local mosquitoes, over 500 cases of indigenous malaria occurred in several counties, and especially Kent and Essex. To deal with this problem a special laboratory was set up in 1918 at the Manor War Hospital, Epsom, under Col. S. P. James, with Mr. P. G. Shute as his assistant. A series of surveys followed by appropriate control measures dealt most successfully with the outbreaks of introduced malaria, which ceased in 1921. Sir Ronald Ross was a frequent visitor to the laboratory and his advice was often sought.

In 1923 malaria therapy became increasingly used for the treatment of general paralysis, and in 1925 a special unit was

established for this purpose at Horton Hospital under the aegis of the Ministry of Health. After the retirement of Col. James the successive directors of this unit were Sir Gordon Covell (1946-65) and Professor P. C. C. Garnham (1965-72). In 1948 Mr. P. G. Shute, O.B.E., became the assistant director of the Malaria Reference Laboratory with which he had been associated from its very beginning.

During the 48 years of the existence of this laboratory some 13,000 patients in the U.K. and abroad have been therapeutically infected with one or other of the four species of malaria parasite, and over 100,000 anophelid mosquitoes have been used. Various laboratory methods currently used in malariology were initiated at Horton, and the masterly use of these techniques by Mr. Shute and Miss Mary Maryon, his collaborator for 37 years, won for the unit an international fame and the distinction of being appointed by the World Health Organization in 1960 as its regional reference laboratory.

The scientific achievements of the Horton Malaria Reference Laboratory are too numerous for them all to be quoted in this note. Characterization of strains of malaria parasites (such as the Madagascar strain of *Plasmodium vivax*), confirmation of the specificity of *P. ovale*, differential infectivity of strain of *Anopheles atroparvus*, studies on the longevity of human plasmodia in man, investigation of relapse patterns, collaboration in the discovery of the tissue phase of *P. vivax*, and advances in the immunology and chemotherapy of malaria are the main subjects to be found in some 250 papers that came from the laboratory during nearly half a century of its existence. There are few research establishments in this country or abroad which can match it in the quality and quantity of scientific output or practical contribution to tropical medicine. There are even fewer units that have given so much for such a modest outlay.

The achievements of the Horton laboratory form one of the memorable chapters in the history of the world's fight against malaria.

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An Academy of Medicine

SIR,—The letter from Sir Thomas Holmes Sellors and others (24 March, p. 737) merits the special attention of the medical profession in Great Britain and Ireland. As a supporter of the concept of a British academy of medicine as outlined in the letter, since the immediate postwar years, I believe that the reasons for the institution of such a body have become more potent and valid from year to year. In your leading article on this subject (24 March, p. 690) you clearly agree that the proposal offers something different from the provisions of the B.M.A., the General Medical Council, the Joint Consultants' Committee, and the Conference of Postgraduate Deans and that it deserves examination and debate. Let us hope that such action may be expedited.—I am, etc.,

E. K. BLACKBURN

Sheffield

Contraceptives on the N.H.S.

SIR,—The provision of contraceptives on prescriptions may not be the forward-looking step that it appears to be at first sight. It in no way provides for the needed adequate free family planning service, which should include provision for education quite apart from regular medical examinations when the pill, intrauterine device, or cap is used. Patients in areas well served by special clinics and local authority schemes (for example, Lambeth and Aberdeen) would appear to be going to suffer by the proposed legislation.

The ability of general practice to meet the needs of a contraceptive service are limited by the lack of experience and knowledge of many G.P.s, the existence of a substantial group who are disinclined on religious or moral grounds to participate in such prescribing, and the number of doctors who feel that they are already overburdened in other areas of health care without adding to their work load (whatever fee the Government might envisage).

I would have been more reassured if Sir Keith Joseph had announced that the Government proposed to set up a free comprehensive family planning service based on existing clinics (such as those of the Family Planning Association and local authorities). I hope that as a profession we can make the Government aware of all the problems involved so that yet more ill-conceived legislation does not follow premature and confused Ministry announcements.—I am, etc.,

ADRIAN HART

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Sudden Whitening of Hair

SIR,—The brief discussion of this "phenomenon" in your leading article (3 March, p. 504) is interesting in that you say that various theories have been put forward to account for it. In the course of research over many years for material for a book on faith healing I was time after time confronted with individuals and various groups trying to establish causes for such happenings before establishing that they actually happened. During this period I came across two men whose hair was stated to have turned white overnight, and on investigation both these claims turned out to be entirely without foundation (unless the one who was an albino could be said to have "foundation" for the story).

Dr. Jelinek¹ has added a graceful contribution to this mythology, but could it not once and for all be stated that hair does not whiten suddenly? Just in case I may be thought too dogmatic may I say I am prepared to look into any adequately documented history of such an occurrence.—I am, etc.,

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¹ Jelinek, J. E., *Bulletin of the New York Academy of Medicine*, 1972, 48, 1003.

Soaking Beds

SIR,—The "soaking beds" described by Dr. J. W. C. Leech (3 March, p. 553) have been observed by nurses since 1948, when the "great deliverance" from the menace of bedsores arrived on the hospital scene. I refer