Relapsing Polychondritis and Pulseless Disease

SIR,—We read with interest the recent letter from Dr. D. A. Rajapakse and Professor E. G. L. Bywaters (24 November, p. 488) concerning immunological investigations in relapsing polychondritis. We should, however, like to correct one misquotation concerning the findings reported by Hughes *et al.*,[1] who, by means of a standard indirect immunofluorescent technique using fetal cartilage as a substrate, detected positive diffuse fluorescence throughout the cartilage matrix with sera from only two of 12 (not from all 12) patients with rheumatoid arthritis.

We have examined sera from a further 104 patients with probable, definite, or classical rheumatoid arthritis as defined by the American Rheumatism Association[2] and detected cartilage matrix fluorescence in 10 (9·6%). Positive results have been found in only one of 102 normal blood donors and in two of 149 patients with a variety of diseases. Of eight patients with relapsing polychondritis tested, three had positive matrix fluorescence and one other showed a weakly positive reaction.[3]—We are, etc.,

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Disinfectant Contamination

SIR,—During routine bacteriological monitoring of an operating theatre suite vessels containing the disinfectant Resiguard (Aspro-Nicholas) at a dilution of 1 in 160 were found to be contaminated with a Gram-negative bacillus identified as *Alkaligenes faecalis*. Resiguard, used for disinfecting floors and furniture between operating sessions, was made up fresh daily in 1 gallon (4·4 l.) amounts, but the containers were not sterilized at the same time. Standard loopfuls of the contents inoculated onto blood agar and incubated resulted in confluent growth of the organism, suggesting that bacterial multiplication had taken place in the disinfectant.

A series of 2·5 ml volumes of serial dilutions of Resiguard in buffered distilled water, tap water (as used in the theatre), and Ringer's solution were inoculated with 0·1 ml of an 18-hour broth culture of the *Alkaligenes* strain. After 5 and 10 minutes contact time standard loopful subcultures were made onto solid recovery medium and results read after overnight incubation. The organism survived 10 minutes' contact with Resiguard at a dilution of 1 in 20 in all tests. The addition to all serial dilutions of methyl alcohol at a final concentration of 10% failed to kill the organism in 10 minutes at a Resiguard dilution of 1 in 100. The Nicholas Research Institute, which kindly checked our results, found that Resiguard at a concentration of 1 in 80 with 6% isopropyl alcohol also failed to sterilize an inoculum of 10^8 organisms/ml over any reasonable time scale. There was no loss of resistance to Resiguard after 20 sub-cultures of the organism on disinfectant-free medium.

Examination of the tap water used to prepare Resiguard in the operating theatre failed to reveal the presence of this organism and swabs from the taps were also negative. However, the same strain of *Alkaligenes faecalis* was found in the theatre drains, gulleys, floors, and windowsills. Sterilizing the containers when fresh Resiguard is prepared has eliminated this organism not only from the containers but also from the other areas of the theatre, and one can only presume that the organism was being spread throughout the environment by the contaminated disinfectant.

As we are not aware of any published literature relating to the survival of vegetative bacteria in Resiguard at in-use dilutions, we feel that our experience is worth reporting.—We are, etc.,

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Glucagon Therapy in Acute Pancreatitis

SIR,—With respect to your leading article (1 December, p. 503) on glucagon therapy in acute pancreatitis, there are several points which we feel warrant comment. Though the mortality in acute pancreatitis has been reported[1 2] as approximately 25%, several authors have recorded much lower figures. Lukash[3] in the U.S.A. and Louw, Marks, and Bank[4] in South Africa have all recorded figures of less than 9%. A recent prospective survey in Glasgow[5] conducted over a two-year period has revealed an overall mortality of 11·5% in 78 patients. For those patients treated conservatively without either the use of protein (Trasylol) or glucagon, the mortality was 6% (four deaths in 67 cases). It may well be that more seriously ill cases are included in some treatment series than others but it seems that an overall mortality of 25% may be somewhat high for the modern conservative management of acute pancreatitis.

Conservative treatment is not directed simply towards the "control of circulatory collapse, relief of pain and prevention of secondary infection" but includes monitoring for the well-known and often insidious associated acute renal failure, coagulation abnormalities and, more recently, the use of high flow oxygen to counteract the hypoxia which often occurs.[6]

Though glucagon may well prove to be of value in the management of acute pancreatitis the report[7] that the infusion of glucagon caused a decline in serum amylase levels is a poor reason for advocating its use. The rate of decline of serum amylase was indeed no faster with glucagon therapy than would have been expected without it. Further it was implied that the severity of an acute attack is equated with the level of serum amylase and this is far from proved. Indeed in very serious attacks serum amylase levels may rise very little.

The results with aprotinin used in a double blind trial[8] are extremely interesting but reservations must nevertheless be expressed at the 25% mortality reported in the control group and the fact that the treated patients fared no better than those managed without the use of aprotinin in some other centres.[3 5] While a controlled clinical trial would certainly seem to be indicated in the assessment of the effects of these agents on acute pancreatitis account will have to be taken of the difficulties of assessing severity of disease and response in acute pancreatitis.—We are, etc.,

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The Solitary Thyroid Nodule

SIR,—The leading article on the solitary thyroid nodule (10 November, p. 310) was rich in valuable practical viewpoints on an old problem. I noted with special interest, however, that it reported a wave of enthusiasm on the continent for fine needle biopsy of thyroid lesions but commented that the degree of accuracy of this method is not high enough to justify its general use.

Living in one of the corners of this continent I have the impression that the "wave of enthusiasm" belongs to history and that, at least in my country, fine needle puncture has become an indispensable routine measure in the diagnosis of thyroid disease. I would be unwilling to manage a patient with a thyroid swelling without the information obtained by cell samples from the abnormal tissue, but I have to accept the fact that most distinguished colleagues in England and the U.S.A. perform admirably without access to this piece of information. The reasons for this difference in attitudes are certainly complex; outside the international congress halls clinical medicine has always a strong local flavour.

It is true that the accuracy of cytological diagnosis of thyroid malignancy is far from absolute; the same is true of histological diagnosis, but for natural reasons histology will yield more cancers from the surgical specimen than one could expect from fine needle puncture before operation. This comparison is much more difficult than is usually thought, since histological diagnosis is an artificial exercise from the clinical point of view. If there were 23 unsuspected cancers in 365 thyroidectomies in the Mayo Clinic (certainly after the application of all types of diagnostic aids except cytology) one may suspect that they correspond to some of the cancers (about 1% of the samples) detected by fine needle puncture in the department of medicine where I am working.

Though thyroid cancer is after all not a first rank clinical problem lymphoid thyroiditis is. The diagnosis of Hashimoto's disease is little more than guesswork without the thyroid cell sample which always provides a reliable diagnosis. Lymphoid