but personally, looking through the bronchoscope or at bronchotomy, I have never seen anything whiter than a light pink. The majority have been red, some very red, emphasizing their vascularity.

I would like to comment on one or two other points. The statement that the mass that projects into the bronchial lumen is only a small part of a more extensive tumour needs modification. It may be and usually is, and is manifestly so in large tumours, but in some cases the tumour does not extend through the whole thickness of the bronchial wall. I have on three occasions resected a tumour via a bronchotomy without excising the whole thickness of the bronchial wall. Two of the patients are free from recurrence 14 and 16 years later and the third was lost sight of after four years when he emigrated.

Finally, I am in no way qualified to express an opinion on the histology, but I have the impression that the boundary between bronchial adenoma and adenocarcinoma may be hazy and that pathologists do not always speak with the same voice. A tumour which some would classify as an adenoma may be classified as an adenocarcinoma by others. The histological report in the three cases mentioned above was "benign bronchial adenoma," and I see no reason to suspect that the reports were incorrect.

It seems to me that there is still need for further study before we can approach the treatment of bronchial tumour with complete confidence. If we regard all of them as malignant or potentially so we shall avoid the surgical error of treating a malignant lesion as though it were a benign one, but will we avoid the surgical misfortune of treating a benign lesion as though it were a malignant one?—I am, etc.,

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Haematology and Biochemistry of Ankylosing Spondylitis

SIR,—Dr. M. J. Kendal and others (28 April, p. 235) state that the relatively high values of haemoglobin and serum albumin in their patients were unexpected. It is, however, well known that patients with ankylosing spondylitis have much less tendency to anaemia than rheumatoid patients and that, when present, the degree of anaemia is seldom severe. For example, in their study of 212 patients Wilkinson and Bywaters[1] found mild anaemia in only 12. Admittedly, however, some authors (for example Lefcovitz and Thomas)[2] have noted a higher incidence. In practice a moderate degree of anaemia in spondylitis always warrants a search for complicating factors, especially gastrointestinal bleeding from drug therapy.

Slight elevations of serum alkaline phosphatase may occur in ankylosing spondylitis especially when radiological changes are advanced.[3] However, it is not generally accepted that this can be the result of bony ankylosis, as is suggested. The elevations are always very slight, and indeed in the present series the mean level of alkaline phosphatase (13·33 units) is only at the upper limit of normal and (as is pointed out) does not differ significantly from the mean alkaline phosphatase (11·77 units) of the rheumatoid group.

While it is true that the E.S.R. does not correlate with the degree of, and is not often a reliable guide to, disease activity, it is misleading to say that this investigation is "of little value" in ankylosing spondylitis. Even moderate elevations of the E.S.R. may be helpful in the diagnosis of the early case. Moreover, the authors give the normal range of the E.S.R. as 0-12 mm/1 hr. Most rheumatologists take a higher figure as the upper normal limit—up to 15 mm/1 hr in young males and 20 mm/1 hr in young females (even higher upper limits of normal are accepted in the elderly). Therefore the statement that "the E.S.R. was moderately raised in most patients" must be regarded with some reserve. In a few severe cases of ankylosing spondylitis the E.S.R. is very high and these patients do often have considerable pain; they are ill, wasted, and often respond poorly to conventional treatment with anti-inflammatory drugs or deep *x*-rays. A continued high E.S.R. can therefore be of some prognostic significance.

Regarding serum protein electrophoresis in spondylitis, it is worth adding that when immunoelectrophoresis is carried out in addition to conventional electrophoresis a much higher frequency of abnormalities is found. In 10 consecutive patients of mine paper electrophoresis showed abnormalities in only two, whereas immunoelectrophoresis showed abnormalities of one or more of IgA, IgG, and IgM immunoglobulins in half the group. From the point of view of early diagnosis this would seem to merit further study. When IgG antiglobulins are considered, elevated levels are found in the majority of patients as in other varieties of seronegative polyarthritis,[4] but this is a more difficult investigation to carry out routinely.—I am, etc.,

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1 Wilkinson, M., and Bywaters, E. G. L., *Annals of the Rheumatic Diseases*, 1958, **17**, 209.
2 Lefkovits, A. M., and Thomas, J. R., *Annals of Internal Medicine*, 1958, **49**, 89.
3 Mowbray, R., Latner, A. L., and Middlemiss, J. H., *Quarterly Journal of Medicine*, 1949, **18**, 187.
4 Howell, F. A., Chamberlain, M. A., Perry, R. A., Torrigiani, G., and Roitt, I. M., *Annals of the Rheumatic Diseases*, 1972, **31**, 129.

Amyloidosis and the Kidney

SIR,—It is a pity that your leading article on amyloidosis and the kidney (12 May, p. 322) conveyed the impression that the body organs are equally affected in primary and secondary amyloidosis, discounting the traditional view that this is not so. The assertions may be true for the kidney, heart, and alimentary tract but they certainly do not apply to skin.

Clinical involvement of the skin in secondary amyloidosis is rare, whereas it is common in the primary form and in association with myeloma. About a quarter of these patients have cutaneous lesions which are sufficiently characteristic to allow a firm clinical diagnosis in anticipation of biopsy confirmation. Translucent brown papules about the eyelids are often haemorrhagic. Sometimes larger but similar nodules appear on the face and lips. Diffuse infiltration of the fingers and hands (sometimes inducing haemorrhagic pseudobullae) leads to an appearance which may be mistaken for scleroderma.

These and other physical signs may lead to recognition of the primary and myeloma-associated forms of amyloid by the dermatologist in patients with a variety of chronic systemic symptoms which have evaded previous diagnosis.—I am, etc.,

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Supporting Service for the Mentally Handicapped

SIR,—We wish to give our fullest support to Dr. E. B. McDowall (26 May, p. 481) in his demand for a single responsible body to provide properly co-ordinated services for the mentally handicapped.

This is what is done in most other countries which have organized provision for medical care. Unless we follow their example the present trend of fragmentation of the provision of services will lead to an overall decrease in care as the mentally handicapped take their place at the foot of the priorities in each group providing these services—whether social, educational, or medical.

Care for the mentally handicapped requires to be comprehensive and prolonged, and to ensure this a specific responsible authority is required.—We are, etc.,

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Chairman

Lennox Castle Hospital,
Lennoxtown, Glasgow

D. A. PRIMROSE
Secretary,
Western Regional Division of Mental Deficiency

The Royal Scottish National Hospital,
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Treatment of Status Asthmaticus

SIR,—I read with great interest your leading article (9 December, 1972, p. 563), on the treatment of status asthmaticus. Though the administration of hydrocortisone (as well as aminophylline) is essential for the treatment, its use results in some untoward effects, some of which have never before been reported.

I have now finished a study of 31 patients admitted in status asthmaticus and treated with intravenous hydrocortisone. The first group of 15 patients included patients who had previously been on steroid treatment for chronic asthmatic bronchitis, and the initial intravenous dose of 200 mg of hydrocortisone sodium phosphate did not give any side effects. The second group of 16 patients included patients who had never been on steroid treatment before, and the administration of 200 mg of hydrocortisone sodium phosphate induced side affects which included paraesthesiae,[1] macular rash, tremor of the arms and legs, and neausea and vomiting, as shown in the table. The duration

Reaction	No. of Patients*
Paraesthesiae	13
"Pins and needles"	10
Itching	5
Heat and chills	2
Numbness	10
Tightness	4
Formication	1
Tremor	5
Nausea and vomiting	3
Macular rash	1

*Most patients developed more than one symptom

of the intravenous injection of 200 mg hydrocortisone ranged from 30 sec. to 1 min. and the duration of the reactions from 1-5 min. The sites affected were the upper and lower limbs, the perineal area, and the face. A second dose of hydrocortisone, when necessary, did not give any of the above reactions.

Some of the above symptoms (nausea, transient itching, localized muscular weakness, bronchospasm, and vertigo) have been described after a local injection of hydrocortisome acetate.[2] Though the cause of the above reactions has not been identified,[3] the role of idiosyncrasy should not be overlooked.

I am most grateful to Dr. D. Pearson for permission to report the results of this study.

—I am, etc.,

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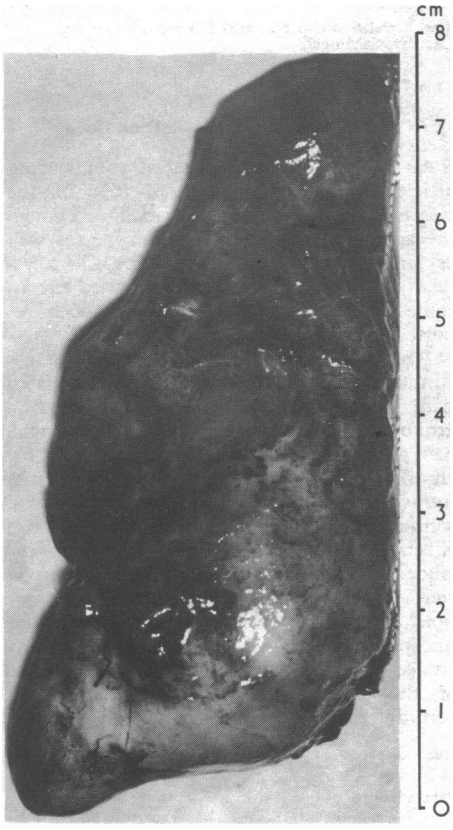
1 Bartrop, D., and Diba, Y. T., *Lancet*, 1969, 1, 529.
2 Kendall, P. H., *Annals of Physical Medicine*, 1958, 4, 170.
3 Martindale, *Extra Pharmecopoeia*, 26th edn., p. 529. London, The Pharmaceutical Press, 1972.

Actinomycosis

SIR,—I was interested in your epidemiology report on actinomycosis (10 February, p. 365). The report does not mention intra-abdominal actinomycosis, and I felt that the accompanying photograph of an actinomycotic appendix might be of interest to your readers.



The patient was a 28-year-old single girl who had a four-day history only and her symptoms and signs were quite subacute, apart from a white cell count of 17,900/mm³ with 85% neutrophils. There was no history of actinomycosis in other sites. At operation the appendix was seen to be very large and thickened and surrounded by the omentum. No free pus was present, but on separating the omentum two or three small sinuses were seen in the greatly thickened mesoappendix. These excluded a very small amount of pus which was cultured. Removal of the appendix was straightforward apart from the fact that the stump could not be buried and was merely ligated and cauterized. Apart from the induration around the stump of the appendix the caecum appeared to be normal. Pus from the appendix showed no growth after 48 hours. The histology report was as follows: "Acute suppurative appendicitis and peritonitis. Actinomycoses organisms are noted in the lumen of the appendix and surrounded by polymorph infiltrate. These are considered to be the probable etiological agent."

The patient had no postoperative complications and was not treated with any antibiotics. Two and a half months after operation she is asymptomatic.—I am, etc.,

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Australia

Research Investigations in Adults

SIR,—I must take exception to a generalization by Professor L. J. Witts in the tape-recorded discussion (28 April, p. 220). In response to the question "Do they have ethical committees in, say, university physiology laboratories?" he replied, "Not yet, but sooner or later they will have to."

Such observations may have relevance to Professor Witts's immediate parish, but are certainly out-dated so far as world-wide physiological opinion is concerned. In most North American universities prior approval by a committee on human experimentation has for many years been a requirement of grant support, experimentation, and publication. The views of the applied physiologist have in fact led those of the clinicians by several years, and a detailed discussion of the issues raised by Professor Witts and his associates will be found in at least one undergraduate textbook. (*Alive, Man! The Physiology of Physical Activity*. Springfield, Illinois: C. C. Thomas, 1972.)

This underlines rather forcibly Dr. Roger Williams's point on the need to check what is going on elsewhere in the world—whether pontificating or experimenting.—I am, etc.,

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Medical Staff Dining-rooms

SIR,—With reference to recent correspondence on medical staff dining-rooms, surely Dr. R. C. Redman's (26 May, p. 485) reasons for an exclusive dining-room are negated by the last few words in his letter, "improved interdepartmental relations and co-operation—something that cannot be valued in strictly economic terms."

Dr. M. W. Rout (28 April, p. 244) and Dr. Redman must appreciate that there are other departments in hospitals which deal with patients and hospital staff and which also benefit from "improved inter-departmental relations."—I am, etc.,

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Lead in Drinking Water

SIR,—In a recent article (7 April, p. 21) Dr. M. D. Crawford and Mr. D. G. Clayton report differences in the mass of lead present in human rib bone between individuals residing for more than 10 years in towns supplied with hard drinking water and those living in soft water areas. Comment is also made concerning the mass of lead in domestic drinking water sampled from taps first thing in the morning, which will contain any residues accumulated in the pipes overnight. The data presented in table I of their paper are not accompanied by any indication of the statistical spread of the results but would appear to illustrate that the burden of lead in rib from soft water areas is greater than that found for hard water areas. As part of a large study relating the chemical composition of man with his environment, published recently,[1] data were presented for the same groups of towns for human rib and are given together with the data of Dr. Crawford and Mr. Clayton in the table.

Dr. Crawford's paper gives no information concerning the method of sample preparation, which could be of some importance to the following discussion. My work was specifically concerned with the lead present in inorganic bone and not the system bone + cartilage + marrow + blood. Unless rather unusual precautions are taken, the significance of the lead content of bone can be open to doubt; some contributory factors are as follows: (1)—Differences exist in the mass of lead in cortical and trabecular bone; in particular there appears to be a significant increase in the level of lead on trabecular surfaces. Papers describing a means of studying this phenomenon have been published.[2 3] (2)—In many rib samples, particularly those taken from individuals more than about 35 years of age, the extent of trabeculation and open cavities can vary considerably, hence also the mass of occluded bone marrow which will contain lead. (3)—Any study concerning elements in bone must consider features of the age distribution. For subjects between about 40 and 50 years old, most of the bone surface will be resting bone with some areas resorbed and only a little depositing. For samples of rib from individuals greater than the age of 50 years, bone is undergoing drastic processes of resorption which will have a profound effect upon the level of chemical elements.

Dr. Crawford and Mr. Clayton provide data in their fig. 2 for lead in dried (not ashed) samples of rib from London and Glasgow which quite clearly will give exceptionally high values for the lead content of ashed rib, even for a hard water area such as London—44 p.p.m. dry weight, which exceeds the lead values for ashed rib from soft water areas in the more recent study.

In my opinion, though current studies describing the mass of lead in bone from soft and hard water areas are of interest, present sampling combined with adequate