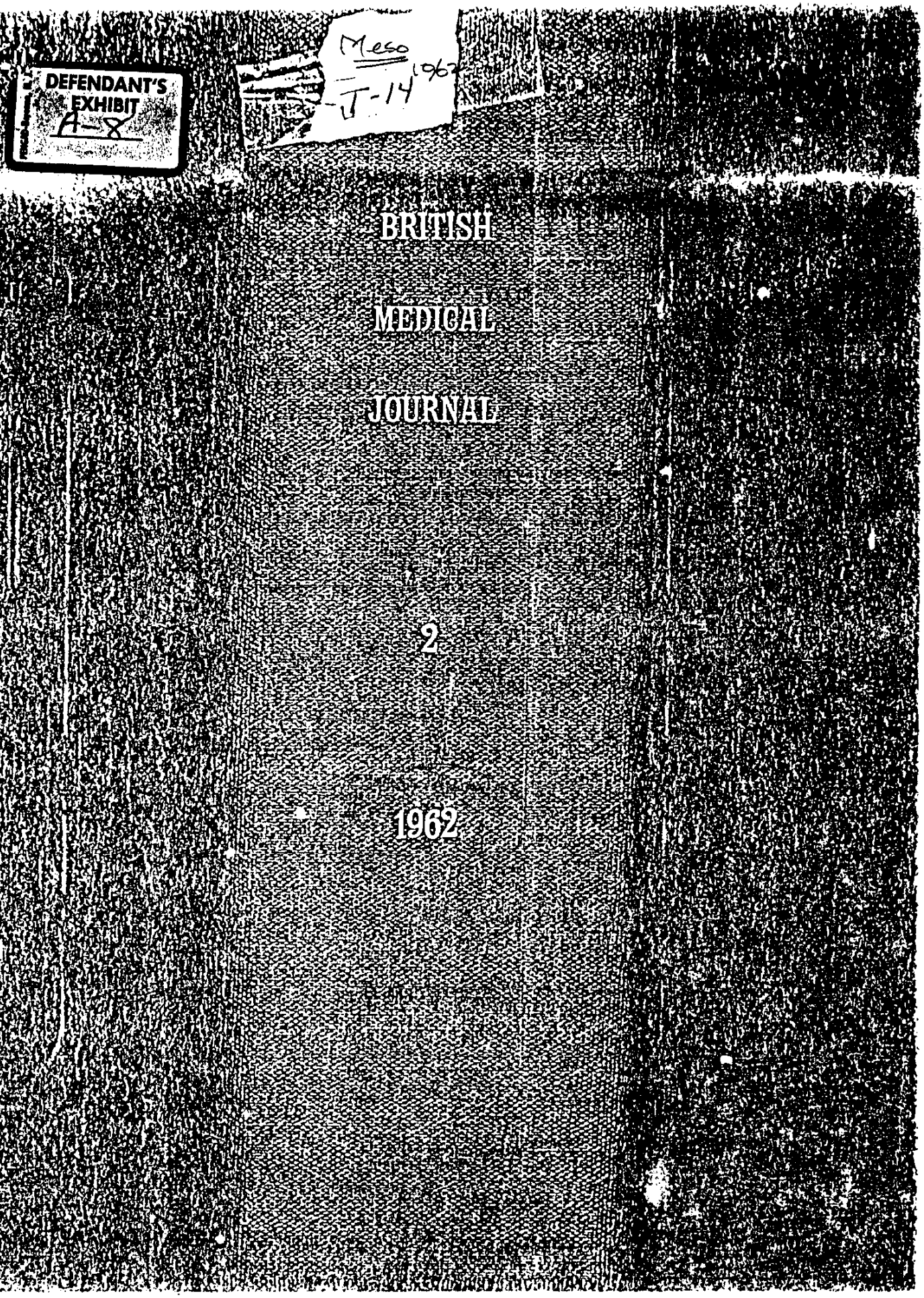
DEFENDANT'S
EXHIBIT
A-8
Meso
J-14
1962
BRITISH
MEDICAL
JOURNAL
2
1962

35664

British medical journal.
v.2, 1962

BRITISH MEDICAL JOURNAL

THE JOURNAL OF THE BRITISH MEDICAL ASSOCIATION

Editor

HUGH CLEGG, M.A., M.D., F.R.C.P.

Deputy Editor

J. W. P. THOMPSON, M.A., M.B., D.P.H.

VOLUME II, 1962

JULY TO DECEMBER

PRICE 12s. 6d.

LONDON

BRITISH MEDICAL ASSOCIATION

TAVISTOCK SQUARE, W.C.1

with good grace. Are the teachers asking for medical scientists rather than doctors? Are the medical journals of to-day enjoying the prestige which seems unreasonably to accumulate around the science rather than the art and practice of medicine? And do not the contributors feed the journals for the prestige necessary to gain a laboratory, an appointment, or a grant? And yet what little gain the patient gets from it all. I do not say that research is unnecessary: only that as it thrives it does so at the expense of the doctors who practise medicine. This is probably inevitable, but this thriving plant of research must not overwhelm the others. Medical scientists often have no medical degree. It is not necessary. It may not even be desirable in the future. What will be desirable in the future is a popular form of service named family doctoring, which is aided in its organization, its recruitment, and its advances by a powerful medical press.—I am, etc.,

London E.17. J. H. S. MORGAN.

Fluoridation of Water

SIR,—We hear that the incidence of dental caries in this country is alarmingly high, and that the understaffed dental profession is having difficulty in keeping pace with the problem. A recent report[1] states that in those drinking fluoridated water all their lives the incidence of caries amongst them is 66% less than in other communities. It also states that in fluoridation the dental, medical, chemical, engineering, and legal aspects have been solved to the satisfaction of those qualified to judge. If these statements are true, what are we waiting for? Fluoridation of the water supply in all parts of the country would seem to be an excellent way of dealing with an urgent problem, and might not the B.M.A. take a lead in the matter?—I am, etc.,

Warminster, Wilts. D. LONGBOURNE.

REFERENCE

[1] Trulson, M. F., Clancy, R. E., and Stare, F. S., *Practitioner*, 1962, 189, 510.

Blankets and Hospital Infection

SIR,—We were disappointed to read in your leading article (August 4, p. 314) that "particles of wool from blankets make a large contribution to the dust of a hospital ward." Published analyses[1-3] have all shown that air-borne fibres are essentially cotton.

Wool blankets have suffered unjustly from errors which have gained widespread credence through frequent repetition, and it is perhaps opportune to draw attention to other popular misconceptions. The claims that the population of airborne bacteria or the incidence of wound sepsis can be reduced by a change from wool have now been disproved,[4-6] but dubious economic assertions are now taking their place. Calculations based on a wool blanket life of 60 wash cycles[4] are unrealistic. This value was obtained in an unreplicated test[7] using blankets that lost almost 9% of their weight during shrinkproofing. Usual industrial losses are below 2% and articles losing 4% are regarded as severely damaged.[8] Other shrinkproof wool blankets bought under contract by institutions have lasted 300 and 350 high-temperature (boiling) wash cycles.[9 10] High-temperature laundering of wool is a practical laundry procedure used on a scale large enough to cause a demand for an Australian Standard method.[11] Available figures for thermal insulation of blankets[12 13] show that wool blankets are warmer than alternatives, both before and after laundering.

The variance of medical opinion between British and Australian sources is due in part to the different weaves traditionally used for blankets in the two countries.[9 10] Precautions are necessary to obtain a satisfactory shrink-resist finish on a British plain weave blanket with dissimilar warp and weft yarns, but this can be done. Alternatively twill weave blankets of Australian type can be woven and shrink-resistance thus simplified.

Since your journal's opinion on public-health matters is widely followed, we must advise workers in this field to be careful to avoid condemnation of established practice by inadequate test. A newspaper announcement condemning the use of wool blankets in home nursing[14] on the basis of a test[15] involving so-called "wool" blankets which were later shown to be essentially a rayon/cotton mixture[16] is an illustration of this point. Many hospital "woollens" are actually mixed fabrics[17] with properties very different from shrink-resistant pure wool.—We are, etc.,

The Royal Melbourne Hospital, Melbourne, Australia. D. C. COWLING. T. A. PRESSLEY.

REFERENCES

[1] Pressley, T. A., *Lancet*, 1958, 2, 712.
[2] Rubbo, S. D., Pressley, T. A., Stratford, B. C., and Dixson, S., ibid., 1960, 2, 397.
[3] Larkin, J. M., Bridson, E. Y., Grieve, W. S. M., and Gibson, J. W., *J. clin. Path.*, 1961, 14, 80.
[4] Newcastle Regional Hospital Board Working Party, *J. Hyg. (Lond.)*, 1962, 60, 85.
[5] Williams, R. E. O., Noble, W. C., Jevons, M. P., Lidwell, O. M., Shooter, R. A., White, R. G., Thom, B. T., and Taylor, G. W., *Brit. med. J.*, 1962, 2, 275.
[6] Rountree, P. M., Loewenthal, J., Tedder, E., and Gye, R., *Med. J. Aust.*, 1962, 2, 367.
[7] Newcastle Regional Hospital Board, *Report on Shrinkage of Woollen Blankets*, 1957. Newcastle upon Tyne.
[8] Moncrieff, R. W., *Wool Shrinkage and its Prevention*, 1953, p. 218. National Trade Press, London.
[9] Pressley, T. A., and Morris, F. P., *Med. J. Aust.*, 1962, 1, 43.
[10] James L., *Aust. National Drycleaner and Launderer*, 1962, 13, No. 8.
[11] Standards Association of Australia, *Australian Standard No. CL2. Laundering of Shrink-resistant Wool Blankets*, 1962. Sydney.
[12] Pierce, F. T., and Rees, W. H., *J. Text. Inst.*, 1946, 37, T181.
[13] Rowlands, R. J., *Text. Res. J.* In press.
[14] *Wool Science Review*, 1961, No. 20, 45. International Wool Secretariat, London.
[15] Sunday Times Medical Panel, *Sunday Times*, 1958, October 12.
[16] Schwabacher, H., Salsbury, A. J., and Fincham, W. J., *Lancet*, 1958, 2, 709.
[17] Lennox, F. G., ibid., 1959, 2, 63.
[18] Harwood, F. C., Powney, J., and Edwards, C. W., *Brit. med. J.*, 1944, 1, 615.

Mesotheliomas and Asbestos Dust

SIR,—Investigations in South Africa,[1 2] and recent inquiries in Great Britain and elsewhere,[3 4] have indicated that there may be an association between diffuse mesotheliomas of the pleura and peritoneal cavities and exposure to asbestos dust. There appears to be no correlation between the severity of any pulmonary asbestosis and the occurrence of these tumours. In a number of cases the exposure to asbestos dust appears to have been minimal, and the only histological evidence of asbestos exposure is the presence of a few asbestos bodies and fibres in the lung tissue. However, a detailed occupational history has, in nearly all cases, revealed some contact with asbestos fibre.

More information is required on this subject to establish what proportion of these uncommon tumo

occur in people who have been exposed at some time to asbestos dust. A histochemical method has been developed for differentiating between pleural mesotheliomas and peripheral pulmonary adenocarcinomata.[5]

We would appreciate, therefore, information concerning any patient in whom this type of tumour has been diagnosed; and suggest that this information be sent to the Director, Medical Research Council, Pneumoconiosis Research Unit, Llandough Hospital, Penarth, Glamorgan, South Wales, where we are compiling a register of these cases.—We are, etc.,

W. J. SMITHER,
Chairman,
Asbestos Research Council.

Cape Insulation & Asbestos Products Ltd., Barking, Essex.

J. C. GILSON.

Medical Research Council, Pneumoconiosis Research Unit, Llandough Hospital, Penarth, Glam.

J. C. WAGNER.

REFERENCES

1 Sleggs, C. A., Marchand, P., and Wagner, J. C., *S. Afr. med. J.*, 1961, 35, 28.
2 Wagner, J. C., Sleggs, C. A., and Marchand, P., *Brit. J. industr. Med.*, 1960, 17, 260.
3 Keal, E. E., *Lancet*, 1960, 2, 1211.
4 Konig, J., *Arch. Gewerbepath. Gewerbehyg.*, 1960, 18, 159.
5 Wagner, J. C., Munday, D. E., and Harington, J. S., *J. Path. Bact.*, 1962, 84, 73.

Loss of Protein in the Gut

SIR,—Your recent leader (September 29, p. 841) discussing protein-losing enteropathy mentioned the possible value of ^{51}Cr albumin in the diagnosis of this condition. While there is no doubt that this substance is theoretically superior to ^{131}I PVP we have found that its preparation is scarcely justifiable economically.

The method described by Waldmann,[1] in which free chromium is removed by passage through MBI—amberlite resin—results in the loss of 80%–90% of the original radioactivity. There is, however, free chromium still present after this procedure, and dialysis for 24 hours to remove it is advisable. The proportions of chromium and albumin used initially were those suggested by Gray and Sterling.[2] Using this modification approximately 85%–93% of the original radioactivity is lost in preparation, a procedure which, it must be added, takes about two days to complete.

It will interest us to know what experience others have had.—We are, etc.,

C. E. ASHCROFT.
O. P. GALPIN.

Manchester Royal Infirmary, Manchester 13.

REFERENCES

1 Waldmann, T. A., *Lancet*, 1961, 2, 121.
2 Gray, S. J., and Sterling, K., *J. clin. Invest.*, 1950, 29, 1604.

Long-term Anticoagulant Therapy

SIR,—The results of the trial of long-term anticoagulant therapy in cerebrovascular disease by Hill, Marshall, and Shaw (October 20, p. 1003) are in agreement with those of most British workers in this field. The authors rightly stress the dangers of this treatment and its failure to prevent recurrence of cerebral infarction, and my own much smaller series confirms this, although the difference in the fate of the control groups is remarkable.

Out of 201 patients admitted to Ashford Hospital, Middlesex, in 1956, 1957, and 1958 with the diagnosis of recent atheromatous cerebral infarction, only 32 were found who were under 70 years of age, had experienced a previous stroke, had a diastolic blood-pressure of below 110 mm. Hg, and had no contraindication to anticoagulant therapy. These 32 were divided at random into 16 controls and 16 on anticoagulant therapy, and a preliminary report of the results was given at the Dundee conference, 1960.[1] This showed six deaths, six recurrent infarctions, one minor recurrence, and three asymptomatic in the controls; with four deaths, three recurrent infarctions, three minor recurrences, and six asymptomatic in the treated group—very little difference. The trial was stopped and a recent follow-up (now from four to six years) shows eight deaths, five recurrent infarctions, two minor recurrences, and one asymptomatic in the controls; with seven deaths, three recurrent infarctions, four minor recurrences, and two asymptomatic in the treated group. One of the treated deaths was due to cerebral haemorrhage, and of the 15 total deaths 10 were due to extracranial causes—fairly evenly distributed between the groups.

This selection of patients who have had at least two cerebral infarcts probably explains in part the difference in morbidity and mortality between my series, where half the patients were dead in each group from four to eight years after their second or third stroke, and Hill, Marshall, and Shaw's surprising figure of only one cerebrovascular death out of 65 patients in the control group with the trial lasting up to four years. The other reason for this discrepancy may be that the average of their series was 57 years, and of mine 66 years, so that the normal survival rates would be quite different.

It will, however, be a pity, as the authors suggest, if these results are interpreted as showing that anticoagulants should never be given for any length of time in cerebrovascular disease, because the results of using them as a prophylaxis against recurrence of cerebral embolism and against continuation of disabling transient ischaemic attacks are well established.—I am, etc.,

A. BARHAM CARTER.

Neurological Unit, Ashford Hospital, Middlesex.

REFERENCE

1 *Thrombosis and Anticoagulant Therapy*, 1960, p. 64, edited by W. Walter. Livingstone, London.

"Iatrogenic"

SIR,—Mr. Aleck W. Bourne criticizes the use of the word "iatrogenic" (September 1, p. 610). He says that the word is derived from the Greek ἰατρος, a doctor, and γεννάω, I produce or create. He argues that "iatrogenic" as an adjective means something which produces a doctor and not something which a doctor produces.

Every philologist knows that most Greek words like those of other languages have several meanings usually. Therefore the Greek noun[1] γενέτης means both the begetter and the begotten. Indeed the adjective γεννητος means begotten or produced. Therefore I submit that the adjective "iatrogenic" is a very appropriate word to describe a disease that a doctor has produced.—I am, etc.,

B. J. O'DRISCOLL.

University College, Galway.

REFERENCE

1 *A Lexicon Abridged from Liddell and Scott's Greek-English Lexicon*. Oxford, 1871, at the Clarendon Press. Impression of 1958, p. 140.