

5 were now above it. The adjusted serum total calcium was 2.44 (0.44) mmol/l (adjusted reference range 2.20–2.60).

The reference range for serum albumin, measured in 70 healthy Ghanaian women, is 31–49 g/l, but 10 of our patients had a low serum albumin (23.7 [4.0] g/l). The eleventh had an albumin of 47.2 g/l. The adjustment of calcium values in the 10 women was 0.41 (0.10) mmol/l, which is greater than the span of the reference range. The low serum albumin values may have been due to the normal fall of 5–10 g/l found in pregnancy² and to proteinuria. Failure to adjust total calcium in such patients will be seriously misleading.

Department of Molecular Medicine,
School of Medical Sciences,
University of Science and Technology,
Kumasi, Ghana

J. J. FLEMING
W. K. B. A. OWIREDU

Department of Surgery,
Komfo Anokye Teaching Hospital,
Kumasi

B. NKUM

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ASTHMA AND RHINITIS ASSOCIATED WITH
LYCOPodium SPORES ON CONDOMS

SIR,—A 45-year-old man, with a long history of rhinitis and asthma in the spring, on exposure to birch pollen and timothy, began to experience the same symptoms when using condoms. He found that he had a reaction only when he used condoms with a very smooth surface and not if they were coated with a gel. Under the microscope the condoms were found to be covered with small particles resembling pollen and scanning electronmicroscopy revealed that they were spores of lycopodium from clubmoss, which is used to create a smooth and less adherent surface.

Radioallergosorbent tests on serum revealed positive reactions to birch, timothy, and lycopodium, with negative reactions to daisy, dandelion, and hazel. A scratch test on the inside of the forearm was positive and itching occurred when the lycopodium on the outside of a condom was applied but not when the plain rubber on the inside was used. When using lycopodium-free condoms he had no more symptoms and there seems little doubt that they were caused by lycopodium spores used as a lubricant.

I have since seen a couple both of whom were sensitive to pollen and complained of rhinitis when using condoms. Almost all condoms in Sweden are covered with lycopodium spores when they arrive in bulk from the manufacturer. Some are later covered with a gel which can inhibit the spreading of lycopodium dust.

Department of Dermatology,
University Hospital,
S-751 85 Uppsala, Sweden

LENNART JUHLIN

CORTICOSTEROID-INDUCED OSTEOPOROSIS AND
HORMONE IMPLANTS

SIR,—Mr Studd and his colleagues (Feb 11, p 339) seem to overstate their case when they suggest that oral or percutaneous oestrogen therapy is an essential adjunct for women on long-term corticosteroids. Their conclusions come from only four patients. They produce no convincing explanation why oestrogens should reverse or prevent the adverse effects of corticosteroids on bone. Oestrone and oestradiol levels are reduced in postmenopausal women receiving corticosteroids,¹ perhaps due to a direct effect of corticosteroids on oestrogen secretion,² but many other mechanisms could be involved in glucocorticoid osteoporosis.³ Decreased bone formation may be especially important, especially in women.⁴ Corticosteroids may directly inhibit osteoblasts⁵ but an indirect effect is also possible, via reduced testosterone levels.⁶

Such observations raise the possibility that some adverse effects of corticosteroids could be abrogated by anabolic steroids or androgens. In corticosteroid-induced osteoporosis anabolic steroids increase osteocalcin levels^{7,8} and forearm⁹ and total bone mass.⁸ The patients reported on by Studd and colleagues were also receiving testosterone implants so the increase in bone mineral density may

have been due to an anabolic effect on bone formation and have had little to do with oestrogen therapy, which does not prevent corticosteroid-induced osteoporosis in mice.¹⁰

It may well be that postmenopausal women who are to start corticosteroid therapy should be on hormone replacement, especially if they have pre-existing low bone mass. However, there is no proof that corticosteroid-induced bone loss is prevented by such add-on therapy, and omens for such a hypothesis are not good.

Rheumatology Department,
City Hospital,
Aberdeen AB9 8AU

DAVID M. REID

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PLASTIC OR PARAFFIN?

SIR,—We would like to add our experience to your thoughtful editorial of Jan 21 (p 139) on the advantages of plastic-embedded media in histology. We provide a histopathology service for a 570-bed hospital with an annual work load of nearly 10 000 surgical specimens. For the past eight years we have routinely embedded all small specimens in plastic media; these specimens include endoscopic and transbronchial biopsy samples, bone-marrow aspirates, needle biopsy specimens from liver, prostate, and breast, small portions of urinary bladder, lymph node biopsy material, and CT-guided needle biopsy specimens. In the past year we processed 3800 blocks of tissue in this manner.

The process is especially valuable for delicate tissue specimens such as gastrointestinal mucosa and pulmonary parenchyma or where cytological detail is an integral part in the diagnostic work-up (as in lymph node pathology and in grading of bladder, prostate, and breast neoplasms).

We have avoided the major drawbacks mentioned in your editorial by using the 'Polaron' plastic embedding medium first developed by DuPont. Thanks to the shorter polymerisation time for this medium, specimens submitted to the laboratory by 1700 hours are ready for histological interpretation the following morning. Our costs are nearly the same as those for paraffin processing. By using standard fixatives and processing fluids we are able to keep material costs to 55 cents per block. With allowance for technician's time plastic-embedded material can be processed at \$4.10 per block, only slightly more than the cost for our standard paraffin product.

We are surprised that medical centres have been so slow to adopt this new technique. In our experience, pathologists are keen to promote change when the impact is primarily on technicians and supporting staff but are reluctant to alter their own entrenched practices. The greater detail to be had from plastic-embedded tissue requires an interpretative adjustment by the pathologist, but that is easily done. We have now reached the point where plastic embedding has become an indispensable daily diagnostic tool.

Department of Pathology,
Kennestone Hospital,
Marietta, Georgia 30060, USA

T. C. KINGSLEY
R. KING