were enlarged and infiltrated. The urogenital, endocrine, reticuloendothelial, and central nervous system were macroscopically normal. In the chest there were bilateral small straw-coloured pleural effusions. Both lungs were rather voluminous but firm, with a granular appearance and texture.

Histological examination of the formalin-inflated lungs revealed diffuse and focal interstitial fibrosis. Haematoxylin-van Gieson staining showed collagen in considerably increased amounts in the interstitium, with occasional areas showing even greater collagen deposition. Intra-alveolar fibrosis, as seen in organisation of proteinaceous exudates, was not evident. Some oedema was also seen. Occasional foci of plump expanded cells, probably of alveolar origin, were present, projecting into alveolar lumina. There was no evidence of *Pneumocystis* or fungi. A single very small focus of secondary adenocarcinoma was identified in one of numerous sections examined.

These findings confirm that this patient had interstitial fibrosis. As other aetiological factors have been excluded, we feel that the interstitial fibrosis developed as a result of therapy with 5-fluorouracil and mitomycin C.

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Practical problems with the Clinitest

SIR,—Recent reports in the *BMJ* (31 March, p 859) and elsewhere^{1,2} have highlighted the potential hazard to the management and control of diabetes in patients with congenital colour vision defects who use the Ames Clinitest. With congenital colour vision defects in 8% of the male population this represents a hazard to one in 12 male diabetics. Since diabetics are known to have acquired colour vision defects associated with their diabetes,³ we recently conducted a study⁴ aimed at determining how well diabetic patients in general coped with colour matching on the Clinitest. The study, conducted during May of this year, produced some surprising findings.

Although diabetics were found to make significantly more errors on the test than a control group with normal colour vision (the diabetics systematically overestimated their sugar concentration for concentration levels $\frac{1}{4}$ %, $\frac{1}{2}$ %, $\frac{3}{4}$ %, 1%), the control group also made errors in the same direction on the test. In particular, diabetics presented with a nominal $\frac{1}{2}$ % concentration of dextrose solution matched on average $1\frac{1}{2}$ Clinitest steps further towards the red (that is, indicating a higher sugar concentration). Furthermore, 14 out of 36 diabetic matches were in error by two Clinitest steps, so that a prepared solution of $\frac{1}{2}$ % sugar concentration (actually measured at 0.49%) was read as a 1% concentration. The control group of normal subjects, however, matched the same $\frac{1}{2}$ % solution on average at the $\frac{3}{4}$ % concentration level. So while the diabetic matches were one and two Clinitest steps away from the expected nominal test match, they were only up to one step further in error than the match achieved from subjects with normal colour vision. These matching errors, which were all of overestimation, occurred for prepared solutions in which the

measured dextrose concentrations were always marginally less than the nominal figure. The errors in matching were comparable whether made under typical domestic tungsten lighting (CIE illuminant A) or artificial daylight (CIE illuminant C).

Analysis by reflectance spectrophotometry of the prepared solutions and coloured reference papers showed that the solutions were indeed slightly redder than their nominal paper reference colours, thus substantiating the "errors" made by the control group of subjects with normal colour vision. Furthermore, for each concentration the solution-paper colour combination had characteristically different spectral reflectance curves (that is, were metameric), indicating that the matches are particularly vulnerable not only to changes in the type of illuminant being used but more importantly to changes in an observer's colour vision mechanisms.

We draw attention to these findings and ask whether their clinical implication for diabetic control is sufficient to warrant a thorough re-evaluation of the test.

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¹ Taylor, W O G, *Transactions of the Ophthalmological Society of the United Kingdom*, 1972, **92**, 95.

² Thompson, D G, Howarth, F, and Levy, I, *Lancet*, 1978, **1**, 44.

³ Lakowski, R, Aspinall, P, and Kinnear, P, *Ophthalmic Research*, 1972, **4**, 145.

⁴ Aspinall, P, Hill, A R, and Cameron, D, in *Proceedings of Fifth Symposium of the International Research Group on Colour Vision Deficiencies*, London, 1979 (in press).

The grumbling appendix

SIR,—It is interesting that this myth should still need a leading article (4 August, p 294)—interesting also that your recent references mostly come from America. In Britain the incentives for retaining the diagnosis "chronic appendicitis" are nowadays seldom tangible, but they are nevertheless difficult to resist. For, however stringent the chief may be in eliminating lily-white appendicectomies he is up against the enthusiasm of young men keen on surgery, and of young patients and parents who are even keener on surgery. We can all remember our early days, when such an alliance enabled us to get our appendicectomy time down to single figures! So let us not be too hard on these young men who make the most of their opportunities, or indeed on the young patients for making full use of a syndrome which has solved many a conflict for them.

But what are we to tell the anxious parents? Firstly, we must say, there is no present evidence of disease, acute or chronic, in the appendix. Secondly, the child may nevertheless develop an acute appendix at some time, like anyone else. Thirdly, they will ask "What, then, is the trouble?" and it is no use saying, "There is nothing wrong." Patients and parents will be reassured if we offer a reasonable physical explanation; for these cases I found one very acceptable (and frequently true) label: "Inflamed internal glands—he'll grow out of it."

There is one point which will help in trying to sort out the rare genuine interval case from the spurious: the pain of onset in acute

appendicitis is typically in the mid-epigastrium, not at the umbilicus. But don't let our patients get to know this.

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Doctors and children's teeth

SIR,—With reference to the letter by Professor P J Holloway (11 August, p 390) on the subject of fluoride tablet dosage, we have been using the dosage (up to 2 years 0.5 mg/day, 2-10 years 1 mg/day) first recommended in your columns¹ and no adverse reaction report (yellow card) has been sent to the DHSS in respect of dental fluorosis.

Higher dosages are commonly used in Australia and have not led to complaints from either dentists or members of the public. Only one paper dealing with dosage has been published in the *British Dental Journal* in recent years.² This actually suggested a higher dosage, 0.75 mg, for the ages of 16-24 months while reducing the dosage in the first eight months to 0.25 mg. Since Professor Holloway admits that greater protection is given by the larger dose we are prepared to take the very remote risk of a claim from a member of the public for causing fluorosis in view of the fact that our sales average 40 million tablets a year.

It is not irrelevant to remark that tablets of 0.25 mg, while available at many dentists, are not in general stocked by pharmacies.

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¹ *British Medical Journal*, 1956, **1**, 1312.

² Geddes, D A M, Jenkins, G N, and Stephen, K W, *British Dental Journal*, 1973, **134**, 426.

Disposable insulin syringes

SIR,—Dr Arnold Bloom and colleagues (2 June, p 1467) report the satisfactory use of "disposable" insulin syringes on more than one occasion. The availability of modern syringes is also of assistance in relation to the precision of administering insulin. The British Standard insulin syringes, with a Luer needle, have a "dead space" of about 0.05 ml, which contains 4 units of 80 U/ml insulin. Thus, if one mixes two insulin types into a single syringe, there can be considerable inaccuracy, particularly if one of the doses of insulin is small. In addition, the large dead space also causes an unnecessarily large air bubble in the syringe on drawing up insulin, and patients have to be instructed in the techniques of injecting the bubble back into the insulin bottle. When the syringe is washed after use insulin to the value of 1.5p may be discarded.

We have recently undertaken a clinical trial of 1-ml disposable syringes (Becton-Dickinson, sterile-fluid-path, Plastipak syringes) in 17 children and 22 adult insulin-dependent diabetics. Instead of the usual 20 marks/ml, the syringes were compatibly marked with 80 marks/ml for use with only 80 U/ml insulin. The dead space of about 0.01 ml meant that the air bubble on drawing up the insulin was small enough to be ignored. Twenty-two patients had an insulin dose less than 10 U, but none had difficulty in measuring the small doses, the syringes being marked at 2-unit intervals. One of the fears relating to the