mention of asbestos-like bodies in describing the effects in the lung of inhaled glass wool fibres in two animal species, including a species which consistently forms asbestos bodies when exposed to asbestos dust. There are few reports in the literature on the effects of glass fibre in man, but Murphy,[5] in a human case of "fibre-glass pneumoconiosis," describes and illustrates numerous uncoated glass fibres in the lung tissues without mentioning asbestos-like bodies. It is also likely that, as used in industrial conditions, much of the dust derived from glass fibres consists of particles too large to be respirable.[4] For these reasons I consider it is very unlikely that any of the "asbestos bodies" in my series can be attributed to glass fibres.

The role of diatomaceous earth is likewise doubtful. Vigliani and Mottura[6] describe in detail the post-mortem histological appearances in the lungs of a man exposed to pure calcined diatomite dust without mentioning asbestos-like bodies. That they were aware of the possibility of finding such bodies is clear from their reference to the work of Nordmann,[7] who described asbestos-like bodies in lung tissue at necropsy in patients exposed to diatomaceous earth and quartz dust. Nordmann, however, is careful to distinguish between "pseudoasbestos bodies" present in two of his six cases and "true asbestos bodies" found in another two cases known to have been exposed to asbestos as well as diatomaceous earth. Some of the "pseudo-asbestos" bodies he illustrates do not resemble asbestos bodies.

Filamentous aluminium silicate has been shown in hamsters to evoke the formation of asbestos-like bodies, but I do not know of any human case in which the presence of such bodies has been attributed to this material. Furthermore, I understand that aluminium silicate has only been used as an insulator in this country for about the last three years (and then only in small quantities for specialized purposes), and thus it also seems unlikely to be an important source of confusion in my survey. None of the histological sections in my series showed any evidence of haemosiderosis of elastic fibres.

By contrast, there is strong circumstantial evidence that many of the bodies I found are in fact true asbestos bodies. More than half of the patients for whom an occupational history is available had either worked with asbestos or had worked in an environment where incidental inhalation of asbestos at some time is virtually certain to occur. The fact that such evidence has been elicited by the comparatively crude methods employed in obtaining retrospective occupational histories from relatives of deceased patients suggests that occupational exposure is probably a more important source of asbestos fibres in the population than the general environmental contamination by asbestos suggested by previous writers.—I am, etc.,

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Genetics of Finger-prints

SIR,—The erudite and amusing article on finger-prints by Professor L. S. Penrose (11 May, p. 321) contains an idea which may prove to be of major importance to our understanding of the mode of action of chromosomes.

After presenting his findings that total ridge count of the fingers varies in inverse proportion to the number of sex chromosomes present the author states (p. 324): "The queer thing is that the effect is produced by the whole chromosome, or a large segment, rather than by its constituent genes." A few years ago I put forward a similar view to account for the process of sex differentiation: "there seems to be strong evidence that the inheritance of sex . . . is controlled not by individual genes but by whole chromosomes or at least large parts of chromosomes."[1] There is increasing evidence that the biological difference between males and females is basically a quantitative one,[2–5] and Professor Penrose's findings appear to support the view that genetically determined quantitative variation can be affected by entire chromosomes or large chromosomal sections.

Professor Penrose's studies emphasize the value of dermatoglyphics, for here we are dealing with a quantitative variable which is fixed in embryonic life and is subsequently unaffected by either environment or hormones. For this reason the sex chromosome constitution may be reflected more directly in ridge counts than in variables of more obvious biological and medical significance.—I am, etc.,

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Crohn's Disease and Carcinoma of Colon

SIR,—With reference to the article by Drs. A. D. Perrett, S. C. Truelove, and G. R. Massarella (25 May, p. 466), in which the authors report a possible association between the two diseases, they give seven references to reports of cases in which carcinoma of the small intestine developed in relation to pre-existing Crohn's disease of the ileocaecal junction. They omitted, however, reports by Martinelli and Belluci,[1] Steele and McNeely,[2] Almond *et al.*,[3] and Davis and Caley.[4] Since carcinoma of the small intestine is very rare, such reports would seem highly significant in pointing to the pre-malignant nature of Crohn's disease. In addition, Hughes,[5] case records of Massachusetts General Hospital, Case 43292,[6] and Wyburn-Mason[7] have reported cases in which Crohn's disease in the ileocaecal region developed into malignant lymphoma. One of my surgical colleagues also had a male patient of 55 years of age who developed intestinal obstruction due to a lesion in the upper part of the ileum, which was resected with end-to-end anastomosis. Sections of the lesion showed the typical changes of Crohn's disease. Seven years later intestinal obstruction recurred and laparotomy now showed a tumour at the site of the anastomosis. Histologically this proved to be a reticulosarcoma. Such observations lend weight to the suggestion that Crohn's disease may be a premalignant condition.—I am, etc.,

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Guanethidine and Diabetes

SIR,—Diabetes and hypertension are often found in the same patient. Although many patients with diabetes must have received guanethidine for control of coexistent hypertension, we have been unable to trace any direct reference to the effect of guanethidine on clinical severity or insulin dosage in diabetic patients. An inquiry to the manufacturers of guanethidine revealed the same lack of published data in this regard. This prompted us to record briefly the following case, where a striking increase in insulin requirements and a rise in blood sugar levels was noticed when guanethidine was discontinued in a diabetic patient.

A 43-year-old housewife was a known diabetic for the last 14 years. Her fundi showed advanced diabetic retinopathy, and she had gradually lost vision over the last three years with development of retinitis proliferans in the right eye. She has been taking soluble insulin 40 units twice daily since 1964, and was quite well controlled. She developed the nephrotic syndrome, with proteinuria, oedema, hypercholesterolaemia, and her blood pressure, which was 150/90 in 1964, gradually rose to 210/120 in June 1967. She was started on bendrofluazide 5 mg. daily and methyldopa 250 mg. three times a day, which was replaced by guanethidine 10 mg. twice daily in December 1967. She needed a little less insulin after this, and was well controlled on soluble insulin 40 units in the morning and 30 units in the evening. She was admitted on 1 March 1968 with a history of several fainting attacks over the previous week. Her blood pressure on admission was 200/100 lying, and 120/80 standing. Her blood sugar was 220 mg./100 ml. It was thought that the fainting might have been due to postural hypotension due to guanethidine, and this was discontinued on 5 March. A progressive increase in glycosuria was noted over the next few days, and she complained of increased thirst while on the same insulin dosage. Her insulin was increased by 8 units on 11 March. The blood sugar levels were found to have gone up in spite of this. On 13 March the blood sugar was 429 mg./100 ml. at 10 a.m. and over 500 mg. at 3 p.m. Insulin was increased by 8 units. On 16 March the blood sugars were 327 mg./100 ml. at 10 a.m. and 478 mg./100 ml. at 3 p.m. Insulin was further increased by 8 units on 19 March to 52 units in the morning and 42 units in the evening. Blood sugars on 20 March were 244 mg./100 ml. at 10 a.m. and 378 mg./100 ml. at 4 p.m. On 22 March these were 347 mg./100 ml. at 10 a.m. and 468 mg./100 ml. at 3 p.m.

Thus within a few days of discontinuing guanethidine the requirements of insulin had increased considerably. Since there was no