specific infection, seems a convincing argument for routine testing by general practitioners of the sexually-active women patients on their lists. Even if a large majority of women examined should prove to be free from any form of genital infection, routine screening programmes—wherever carried out—offer the only means of eventually controlling sexually-transmitted disease.—We are, etc.,

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¹ Pariser, H., Farmer, A. D., and Marino, A. F., *Southern Medical Journal*, 1964, 57, 688.

² Benfield, J. M., *Community Medicine*, 1971, 126, 168.

Diagnosis of Appendicitis

SIR,—I was interested in the simple and practical points Mr. G. D. F. McFadden (13 November, p. 430) mentions in connexion with the diagnosis of appendicitis. But he states that he has failed to see in print that coughing often causes pain in acute appendicitis. In recent editions of *Short Practice of Surgery* this test has been described, and in the last edition reference is made on page 984.¹

The other points mentioned in the letter are also very useful guides in the diagnosis of acute appendicitis.—I am, etc.,

MCNEILL LOVE

Brickendon, Hertford

¹ Bailey, H., and McNeill Love, R. J., *Short Practice of Surgery*, 15th ed. London, H. K. Lewis and Co., 1971.

Cavernous Chylangioma of Jejunal Mesentery

SIR,—Mr. E. S. Field's account (3 April, p. 27) reminds me of a girl of 12 whom I explored in 1961 for suspected appendicitis. A loop of plum-coloured, fibrin-coated ileum had to be resected as it was clearly in jeopardy. The related mesentery was a mass of chylous vessels which were obstructing venous drainage. On opening the bowel, the mucosa presented a remarkable white papillary surface like the head of a cauliflower—the engorged villi were visible to the naked eye.

Alas! Ill-advised deep-freezing spoiled the specimen for posterity, but the appearances still seem worth recording.—I am, etc.,

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The Lungs in Ankylosing Spondylitis

SIR,—Your leading article (28 August, p. 492) now accepts upper lobe fibrosis as an occasional feature of this systemic disorder. It would be premature to assume that this is part of the primary pathological process in view of the non-specific lung histology. Furthermore, such fibrosis is not necessarily of ominous significance, since in one case I reported¹ bilateral upper lobe fibrosis has existed for 21 years with little pulmonary disability except for an intercurrent aspergilloma.

Manometric studies have shown a very high incidence of impaired oesophageal motor

function in patients with ankylosing spondylitis.² One may speculate that these may predispose to aspiration pneumonitis, especially in the upper lobes, where mechanical ventilation is defective. The association of such pulmonary abnormalities and ankylosing spondylitis calls for a careful radiographic and manometric assessment of oesophageal motor function, even when symptoms do not point to a primary oesophageal disorder.—I am, etc.,

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Gastroenterology Unit,
Wellington Hospital,
Wellington, New Zealand

¹ Scobie, B. A., *Australasian Annals of Medicine*, 1970, 19, 131.

² Scobie, B. A., in *Proceedings of the Third Asian-Pacific Congress of Gastroenterology*, Melbourne, Vol. 1, p. 263, 1968.

Diazoxide and Hypertension

SIR,—Over the past five years intravenous diazoxide has been our first choice for the emergency treatment of severe hypertension with renal failure. We confirm the satisfactory results now reported by Drs. J. E. F. Pohl and H. Thurston (16 October, p. 142).

Since these authors presented their work at the Medical Research Society¹ we have also used diazoxide orally for long-term management. It has been given to patients suffering from severe renovascular hypertension, many of whom had severe postural hypotension while on treatment with conventional hypotensive agents. By using diazoxide in addition to methyldopa and or bethanidine it has been possible to obtain control with a small dose (100–200 mg daily) and we have not so far encountered hyperglycaemia requiring tolbutamide therapy. With this combination it has also been possible to reduce the dose of methyldopa and bethanidine and thereby reduce the incidence of side effects.—We are, etc.,

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D. K. PETERS

Department of Medicine,
Royal Postgraduate Medical School,
London W.12

¹ Pohl, J. E. F., and Thurston, H., *Clinical Science*, 1971, 40, 11.

Anticoagulant Interactions

SIR,—May I be allowed to clarify some points of detail in the leading article (16 October, p. 128) "Anticoagulant Interactions"?

The question of chloral hydrate-warfarin interactions is at present very much debated. Sellers and Koch-Weser's work indicated that a chloral hydrate metabolite, trichloroacetic acid, and not chloral hydrate itself caused warfarin displacement from albumin binding sites.^{1–3} A similar chloral hydrate induced potentiation of warfarin has now been reported by Weiner,⁴ though this observation was made only on a single patient.

Recently, however, several groups of workers have failed to observe any potentiation of warfarin by chloral hydrate.^{5–7} Griner and his co-workers stating that "the clinical significance of any interaction between warfarin and chloral hydrate is negligible for patients receiving long-term therapy with warfarin under the conditions of this study." The last few words of this statement are perhaps the most significant. Sellers and

Koch-Weser⁸ have countered these results by asserting that the potentiation occurs soon after chloral hydrate administration and will only be transient, but still of clinical significance.

One interesting factor which arises in comparing these studies is that Sellers and Koch-Weser's work was carried out using healthy volunteers, while the authors who failed to observe any potentiation of warfarin by chloral hydrate carried out their studies on patients who were already receiving long-term warfarin therapy for various conditions. The single case observed by Weiner does, however, fail to fit this pattern.

The fatal haemorrhage which Cucinell *et al.*⁹ suggested was caused by chloral hydrate reducing the anticoagulant effect was observed with a combination of chloral hydrate and bishydroxycoumarin. Here there is always the possibility of a different mechanism of interaction.—I am, etc.,

P. F. COOPER

Uxbridge, Middx

¹ Sellers, E. M., and Koch-Weser, J., *New England Journal of Medicine*, 1970, 283, 827.

² Sellers, E. M., and Koch-Weser, J., *Annals of the New York Academy of Sciences*, 1971, 179, 213.

³ *British Medical Journal*, 1971, 1, 360.

⁴ Weiner, M., *Annals of the New York Academy of Sciences*, 1971, 179, 226.

⁵ Griner, P. F., Raisz, L. G., Rickles, F. R., Wiesner, P. J., and Odoroff, C. L., *Annals of Internal Medicine*, 1971, 74, 540.

⁶ Udall, J. A., *Annals of Internal Medicine*, 1971, 75, 141.

⁷ Breckenridge, A., Orme, M. L'E., Thorgerisson, S., Davies, D. S., and Brooks, R. V., *Clinical Science*, 1971, 40, 351.

⁸ Koch-Weser, J., and Sellers, E. M., *Annals of Internal Medicine*, 1971, 75, 141.

⁹ Cucinell, S. A., Odessky, L., Weiss, M., and Dayton, P. G., *Journal of the American Medical Association*, 1966, 197, 366.

Hazards of Colostomy Closure

SIR,—Your leading article on the "Hazards of Colostomy Closure" (13 November, p. 380) offers much needed wisdom on this procedure, which is all too often regarded as easy and safe when compared with resection of the colon. May I draw your attention to another hazard of colostomy closure which can be disastrous; this is the development of postoperative infective diarrhoea. Patients who have previously had no such trouble may develop this owing to the activation of organisms present in the bowel at the time of operation. The most dangerous of these in our experience in *Clostridium welchii*. To prevent these hazards a swab should be taken from colostomies prior to closure, or a routine single dose of penicillin given on the day of operation.—I am, etc.,

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Hamster-hair Hypersensitivity

SIR,—May I suggest that the onset of asthma in Dr. J. A. Wilson's Case 3 (6 November, p. 341), as well as the failure to improve when the hamsters were removed, was due to coincidental disease of the adrenals. Dr. Green and I¹ reported two patients with Addison's disease who presented with asthma, and postulated that their asthmatic tendency had been unmasked by deteriorating adrenal cortical function.

The "striking adrenal cortical atrophy" found at necropsy may have been due to Addison's disease rather than to cortico-