be more frequent than in normal individuals matched for age and sex. However, a marked degree of fingertip ridge flattening accompanied by white lines, which were usually plentiful, was seen on all fingers in only five patients (3 women and two men). Moreover, in the two males, fingertip changes, including patchy ridge damage, were clearly occupational in origin, and in the females, in whom all prints were readable, appearances were consistent with age and housewifery in two (aged 69 and 71) and long-standing dryness of the hands in the other (aged 51).

These findings seem to be in general agreement with those of Dr. David[4] who did not find ridge atrophy in dermatitis herpetiformis patients. In addition, in eight patients with dermatitis herpetiformis examined by one of us (R.M.) in whom the rate of uptake of tritiated precursor compounds in the epidermis has been examined no difference has been detected when compared with normal controls. This would indicate a normal rate of synthesis of macromolecules within the epidermis and suggests a normal rate of epidermal replication.

We think it unlikely that fingerprint observation in dermatitis herpetiformis will prove useful as a measure of jejunal pathology in this disease.—We are, etc.,

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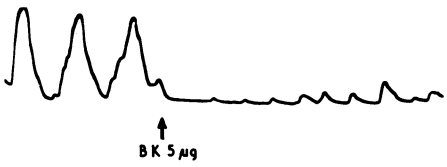
1 Marks, J., Shuster, S., and Watson, A. J., *Lancet*, 1966, **2**, 1280.
2 Shuster, S., and Marks, J., *Systemic Effects of Skin Disease*, London, Heinemann, 1970.
3 Brow, J. R., Parker, F., Weinstein, W. M., and Rubin, C. E., *Gastroenterology*, 1971, **60**, 355.
4 David, T. J., in Proceedings 4th International Congress of Human Genetics, Paris, 1971, Amsterdam, *Excerpta Medica*, in press.

Uterine Hypotonia

SIR,—Further to the correspondence concerning uterine hypotonia (24 July, p. 251 and 11 September, p. 637) Landesman[1] reported relaxation and cessation of activity of the human uterus in the presence of bradykinin, and Serneri[2] showed a relationship between bradykinin and fibrinolysis.

In 1969 while working at the University of Bradford I repeated the work of Landesman (Fig.) and presented my findings to the Blair Bell Research Society.[3] I also suggested[4] that the presence of bradykinin released as a by-product of the activation of the coagulation system was the cause of uterine atony which occurs with severe antepartum haemorrhage and amniotic fluid embolism. This atony had been described previously by Scott and Reader[5] as being of greater import than the coagulation defect itself.



Any substance that interrupts the activation of the coagulation system, such as aprotinin (Trasylol) or aminocaproic acid (Epsikapron), will prevent the release of fibrinogen degeneration products[6] and more importantly the release of kinins and thus will improve the condition.—I am, etc.,

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1 Landesman, R., Campbell, W. L., and Wilson, K., *Nature*, 1963, **197**, 1208.
2 Serneri, G. G. N., Ferrini, P. L. R., Paoletti, P., Panti, A., and Valva, G. d'A., *Thrombosis et Diathesis Haemorrhagica*, 1965, **14**, 508.
3 Senior, J. B., and Spencer-Gregson, R. N., *Journal of Reproduction and Fertility*, 1969, **18**, 551.
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Mental Deficiency Nursing

SIR,—Mrs. Jean Patey (2 October, p. 50) presents a point of view which is held by many parents and relatives of patients in hospitals for the mentally handicapped.

It is the experience of hospitals that patients who are settled, clean, and happy in hospital (the critics say "institutionalized") often fail to be accepted, become dirty, and present a nuisance outside hospital. With routine supervision patients function in ways which suggest to the visitor that they do not need to be in a hospital.

It is usual and natural for young people to leave home after adolescence, and if the mentally handicapped are to follow a normal pattern of living they too should go away from their parental homes. At present a reduction in hospital places with little immediate expansion in community provision compels many mentally handicapped people to remain at home.

Providing hospitals for the mentally handicapped with better facilities and more staff is expensive, and the argument that these hospitals are not necessary will appeal on economic grounds. Scandinavian services for the mentally handicapped, which claim to be a model, have residential institutions which are hospitals given other names. In planning for the mentally handicapped the doctors, nurses, and parents associated with hospitals will be the least consulted, because they could be imputed to hold biased views.

The organizations which adopt an anti-hospital attitude are composed of only a minority of the parents of the mentally handicapped. A survey of the wishes of the parents and relatives of patients in hospitals for the mentally handicapped would probably show a majority in favour of hospital care.—I am, etc.,

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Agranulocytosis Associated with Trimethoprim-sulphamethoxazole

SIR,—Drs. B. Hulme and D. S. Reeves (11 September, p. 610) report leucopenia associated with a combination of trimethoprim and sulphamethoxazole during immunosuppressive therapy with prednisolone and azathioprine after renal transplantation. They warned against the use of trimethoprim-sulphamethoxazole soon after cadaveric renal transplantation, but they left the pathogenetic mechanism of leucopenia open. In two patients we recently observed agranulocytosis in association with the use of trimethoprim and sulphamethoxazole, suggesting an immunological reaction caused by the sulphonamide component.

A 64-year-old woman received sulphamethoxazole for a urinary tract infection during a period from 10 to 24 January 1971, and thereafter ampicillin. This was changed to Eusaprim, a combination of trimethoprim and sulphamethoxazole, on 2 February. On the next day she was febrile, and a rash and a disappearance of neutrophils was noted on 8 February. The treatment with Eusaprim was discontinued, and a spontaneous remission took place seven days later.

The urinary tract infection of a 67-year-old woman was treated with Eusaprim during the period from 13 to 17 November 1970, and the white blood count remained normal. A new course of treatment was started on 25 November with sulphamethoxazole, but stopped on the following day as she became febrile and neutropenic. A remission took place over three days during treatment with hydrocortisone.

The course of the disease in both of our patients was similar; they had received sulphamethoxazole alone or in combination with trimethoprim two weeks earlier, and the new treatment was followed by a rapid neutropenic and febrile reaction. The clinical picture was typical of an immunological reaction. In earlier reports on agranulocytosis due to trimethoprim and sulphamethoxazole[1 2] the recovery was more delayed than in our cases, and there was morphological support for marrow toxicity.[1]

It is evident that in the combination of trimethoprim and sulphamethoxazole it is the sulphonamide component which causes agranulocytosis, probably both immunological and toxic. No evidence is available to support the view that the combination with trimethoprim would cause agranulocytosis more often than the sulphonamide component used alone.—We are, etc.,

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1 Evans, D. I. K., and Tell, R., *British Medical Journal*, 1969, **1**, 578.
2 Paulley, J. W., *British Medical Journal*, 1970, **2**, 364.

Chromosome Breakage and Ultrasound

SIR,—Mr. I. J. C. Macintosh's letter concerning chromosome breakage and ultrasound (18 September, p. 703) has prompted my response.

The use of Schlieren photography to examine the shape of Doppler ultrasonic patterns is fraught with the possibility of misinterpretation. I fear that the blame for this rests upon us, the manufacturers, who have promulgated this technique. The Schlieren depicted by Mr. Macintosh in his letter is obtained by alternately vibrating both the transmitting and the receiving crystals. Obviously, when any Doppler ultrasonic unit is used in vivo, this is not the fact. Only one crystal is used for transmission and the other for receiving. Thus, if one takes a Schlieren photograph of a Doppler ultrasonic unit as it is used in actual practice, the picture is as seen in the Figure. Examination of this readily points out that there is no focal point at all in the Doptone fetal pulse detector. The beam is