value and therefore any improvement in homeostatic control would have been obvious during pregnancy. Thus our argument that the shape of the curve may be a more important indicator of homeostatic control than any actual level reached appears to be supported by these cases.

It is probable that factors controlling fasting blood glucose concentrations are separate from those affecting homeostatic control of a glucose load. Thus a curve indicating poor homeostatic control can occur from a low as well as a high fasting concentration. The converse can also be true, but usually patients with a very high fasting concentration have curves of abnormal shape. It has long been appreciated that the fasting glucose concentration is lowered during pregnancy and this change appears to be independent of the progressive changes in blood glucose concentration which occur after an ingested glucose load.²

All that seems to have changed in the cases reported by Drs. Sheldon and Coleman is the fasting level; while this is a phenomenon worthy of further research, it would seem unwise to regard this change alone as indicative of "remission" during pregnancy, particularly if by this is meant a reduction of risk to the fetus.—We are, etc.,

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Enteric-coated Potassium Chloride— A Continuing Hazard

SIR,—Small-bowel ulceration due to enteric-coated potassium chloride tablets was first described 10 years ago.^{1,2} Since that time there have been many reports confirming the original observations³ and similar lesions have been demonstrated in animal experiments. Other, apparently safer, forms of potassium chloride have been developed. In particular, small-bowel ulceration has not yet been described with wax-cored slow-release tablets or effervescent tablets containing potassium chloride.

The continuing risk of small-bowel ulceration was recently shown in a patient who had taken an overdose of Hydrosaluric-K tablets (hydrochlorothiazide 25 mg with potassium chloride 572 mg in an enteric-coated core). His total intake of potassium chloride was 15–20 g. Twenty-four hours after admission he developed signs of peritonitis. At laparotomy he had multiple punctate ulcers and an area of haemorrhagic necrosis in the mid-ileum which necessitated a fairly extensive small-bowel resection.

The dangers of enteric-coated potassium are widely known and simple enteric-coated potassium chloride tablets must rarely be prescribed. However, enteric-coated potassium is still present in some widely used diuretic/potassium mixtures, and the readily available prescribing information makes no reference to this fact in the case of the preparation implicated in this instance.^{4,5}

We should like to draw attention to this continuing hazard and question the justification for the continuing manufacture and sale of compound tablets of this type.—We are, etc.,

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Shigella sonnei Septicaemia in a Child with Acute Monocytic Leukaemia

SIR,—Dr. Eileen E. M. Moore's account (5 January, p. 22) of *Shigella sonnei* septicaemia in a neonate prompts me to report the development of fatal septicaemia and meningitis in a child who was exposed to *Sh. sonnei* during initial treatment for acute monocytic leukaemia.

A 3½-year-old girl presented at another hospital with a month's history of pallor and transient febrile episodes. Blood examination suggested, and bone marrow aspiration confirmed, a diagnosis of acute monocytic leukaemia. She was transfused and given prednisone by mouth. On transfer to a children's ward at Hammersmith Hospital six days later she was much improved, with a voracious appetite and no gastrointestinal symptoms. Cultures of ears, nose, throat, and urine showed no pathogens. She was nursed in a single room but not strictly confined to it. The day after admission she began treatment with a combination of eight antileukaemic drugs,¹ and after the first five-day pulse of this therapy the peripheral blast cell count had fallen from 18,000/ μ l to nil; the neutrophil count was 400/ μ l.

The day after the patient's admission *Sh. sonnei* was isolated from the stools of another child in the ward. Although this child was immediately discharged, our patient developed fever and severe diarrhoea on her fifth hospital day and *Sh. sonnei* was isolated from three successive stools and two rectal swabs. Cultures of urine and cerebrospinal fluid at the onset of fever were sterile. Initial treatment with phthalylsulphathiazole by mouth was changed after 24 hours to oral chloramphenicol because of persisting fever up to 39.5°C. On the ninth hospital day, despite 48 hours of chloramphenicol treatment, she was drowsy, febrile, and very ill, and *Sh. sonnei* was found in two separate blood cultures. Intravenous gentamicin and cephalothin were substituted for chloramphenicol and a transfusion was given of 1×10^{11} granulocytes from a compatible donor with chronic granulocytic leukaemia. The temperature fell to 38.5°C and she improved slightly, but that evening aspirated a quantity of vomitus and suffered cardiac arrest.

Necropsy disclosed multiple petechial haemorrhages, enteritis with subserosal bleeding, and consolidation of the lower lobes of both lungs. The cerebrospinal fluid was cloudy and patches of exudate were present on the surface of the brain. Histological sections showed a paucity of polymorphonuclear cells consistent with the neutropenia present before death.

Dysentery due to *Sh. sonnei* is usually a brief illness which subsides without antibiotics: the organism appears to be confined to the lumen and mucosa of the bowel. Dr. Moore's patient and other reported patients with *Sh. sonnei* septicaemia^{2,3} were neonates or young children, though one adult

case has been reported.⁴ Septicaemia complicated by meningitis has been reported once only, in a 3-day-old infant.⁵ Our patient was older than in most previously described cases but had significant neutropenia attributable to her acute monocytic leukaemia and also to its treatment. The fatal outcome of this usually benign infection in a patient with reduced body defences emphasizes that infectious disease in leukaemia patients often fails to follow usual clinical patterns. Rare and serious complications are so common that it seems unwise to follow "accepted" methods of management derived from experience in treating less vulnerable individuals.—I am, etc.,

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Effect of Levodopa on Tremor in Benedikt's Syndrome

SIR,—Recent advances in our knowledge of the regional biochemistry of the brain, particularly that of the biogenic amines, have made possible a new understanding of the pathogenesis of Parkinsonism, and levodopa has been used successfully in the treatment of this disease.^{1,2} From experience with Parkinsonism it was expected that levodopa therapy might be effective for the patient with Benedikt's syndrome and improve some, if not all, of the manifestation of the disease. We wish to report a case of Benedikt's syndrome treated successfully with levodopa.

A 44-year-old man was admitted in February 1973 with the chief complaint of involuntary movement of the left arm which had appeared six years after a stroke in July 1963. This attack had produced oculomotor palsy on the right side and hemiparesis and hemianaesthesia on the left side. A few months after the stroke there had been a partial recovery, but he suffered relapses in May 1967 and July 1969, after which the involuntary movement developed. Cinematographic and electromyographic studies showed that the involuntary movement was a rhythmic, coarse tremor with an average frequency of 2.6–3.1 cycles per second. Though this tremor was present at rest, it became more intense with movement of the involved limb. It disappeared during sleep and increased with emotional stress, cold, and fatigue. The patient was given levodopa in doses of 200 mg three times a day and the dose was gradually increased to 1,000 mg three times a day. Marked improvement of tremor was noted within a few weeks and the efficacy of the drug was ascertained by means of a cross-over study. The other neurological signs were not altered.

It has been suggested by clinical observations and animal experiments that the mechanism of tremor in Benedikt's syndrome is associated with the cerebellofugal pathway and the rubrospinal tract. On the other hand Poirier³ pointed out that experimental lesions in the ventral tegmentum of the midbrain producing sustained postural tremor in the monkey were associated with a decreased concentration of dopamine and