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11 000/μl, and diarrhoea lasting more than 7 days were significantly more common with *C coli* infection. Other indices were not significantly associated with the species (or biotype) (table).

We cannot explain the apparent disagreement between our findings and the Yugoslavian study. We serotyped all strains with sera obtained by immunising rabbits with 52 campylobacter reference strains kindly provided by Dr Lior[2] and found that the distribution of serogroups among the index children and the others was similar. In other words, there was not a virulent serogroup of *C coli* which accounted for severity of *C coli* enteritis in our study group. *C coli* was isolated in 15% of cases from Tuscany but in 35% in Popović-Uroić's series, and almost all our children were under 3 years of age while in the Yugoslavian study 46% of patients were over 5. Differences in the environmental circulation of the two *Campylobacter* spp in the two countries, together with differences in age, may explain the disparity.

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NATALE FIGURA
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SUBCUTANEOUS APOMORPHINE IN PARKINSON'S DISEASE

SIR,—Dr Stibe and colleagues (Feb 20, p 403) report on the benefit of continuous subcutaneous infusions of apomorphine hydrochloride in parkinsonian patients with response oscillations to oral levodopa. Stibe et al have also successfully used 'Penjects' in some cases.

We have treated four men and three women aged 41–68 (mean 57) years with intermittent subcutaneous injections of apomorphine for up to 6 months, using a penject system (Disetronic AG, Burgdorf, Switzerland). The pen contains 3·5 ml ampoules of apomorphine hydrochloride solution (10 mg/ml), and doses can be pre-set in multiples of 0·05 ml from 0·05 to 0·75 ml. The patients had had Parkinson's disease for 11 years on average (range 2½ to 15 years) and the daily levodopa doses ranged from 800 to 2500 mg (mean 1430 mg). All patients had three, four, or five off periods daily that were refractory to conventional treatment, including sustained-release levodopa. In five patients off periods were accompanied by prominent dystonia (painful feet in three, jaw spasms in one, and laryngeal stridor in one).

As Stibe et al found, subcutaneous apomorphine reversed off periods in all our patients 5–15 min after an injection, and the effect usually lasted 1·5–2·5 h. The doses required to produce an "on" effect were larger than those reported by Stibe et al and ranged from 2·5 to 7 mg (mean 4·7 mg). The average time "off" per day was reduced from 4·9 to 1·8 h but there was no significant reduction in the daily dose of levodopa. 30–60 mg oral domperidone daily prevented nausea from the apomorphine injections, and there were no laboratory abnormalities except slight eosinophilia in some. So far no psychotic side-effects have been observed.

Our patients found that they could quickly interrupt off phases and painful off-period dystonia, thus greatly improving the scope of social activities open to them; this social improvement may not always be reflected fully in the number of hours "on". For example, a 43-year-old woman with a 5-year history of Parkinson's disease had responded well to levodopa for 3 years when wearing-off reactions and very painful off-period foot dystonia appeared. Disabling foot cramps lasted up to 3 h and were especially severe in the early morning and late afternoon or evening. After some devastating experiences with dystonic spasms during visits to the theatre this woman had stopped going out in the evening. Her off-period dystonia proved very responsive to 2·5 mg apomorphine subcutaneously—and, with a penject in her handbag, she now feels free to go to concerts and the theatre and out for dinner.

In our experience subcutaneous apomorphine has been as practical and reliable a means of interrupting off-period dystonia as intravenous levodopa.[1] In contrast to earlier reports[2,3] neither we nor Stibe et al found differences in the effects of apomorphine on tremor and on akinesia/rigidity. The intermittent subcutaneous administration of apomorphine seems more convenient and simpler than the use of minipumps, and we agree with Stibe et al that this approach may be sufficient for many patients with oscillating Parkinson's disease including, in our opinion, some who are now on pumps.

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GROWTH IN CHILDREN TREATED FOR ACUTE LYMPHOBLASTIC LEUKAEMIA

SIR,—We agree with the conclusions of Dr Clayton and colleagues (Feb 27, p 460). Controversy has raged for years over the contribution of growth hormone deficiency to growth failure in children treated with prophylactic cranial irradiation for acute lymphoblastic leukaemia (ALL). Although we agree with Kirk et al[1] that biochemical growth hormone deficiency is prevalent among patients receiving 2400 cGy of irradiation, the suggestion that growth hormone treatment is appropriate in most cases is contrary to our experience. We have reviewed 181 patients with ALL who were alive six years or more from diagnosis and who were diagnosed between 1970 and 1979.[2] We compared two groups: group I (n = 137) was those in first remission who had received a single course of cranial or craniospinal radiation (2400 cGy) and group II (n = 44) was those who had relapsed and required further radiation treatment (cranial, craniospinal, testicular, or total body irradiation). We did not compare growth by the height standard deviation score but, because endocrine data were included in a general survey of morbidity within the two groups, we found that 20% of group I had clinically significant endocrine morbidity. This included a high proportion of patients with early puberty leaving only 1 out of 137 who required replacement therapy for isolated growth hormone deficiency. Some degree of growth retardation was, however, common and we agree with Clayton and colleagues' suggestion that those children below the tenth centile whose growth is persistently poor, especially where growth hormone deficiency is demonstrated, would benefit from a trial of growth hormone. In contrast was the high frequency of endocrine morbidity (68%) in group II, where replacement therapy is normally required.

We have also done growth hormone studies in response to insulin-induced hypoglycaemia in 9 of 26 patients with early puberty who had received 1800–2400 cGy of prophylactic cranial irradiation. Of these, 8 had an inadequate growth spurt and 7 had a blunted or frankly subnormal response to hypoglycaemia (unpublished). In this situation, growth retardation and growth hormone insufficiency assume great importance when final height is compromised by an early inadequate growth spurt. Our practice has therefore been to treat with a gonadotropin-releasing-hormone analogue and growth hormone.

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