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Professor Hughes and Dr Long comment:
We were interested to read of Dr Schober's experience of using the Insuflon cannula in 49 young diabetics. The circumstances in which he used the device were entirely different to those operating during our study. The comparison is therefore not a valid one other than to comment that the device did not appear to be either effective or acceptable when tried out in a summer diabetic camp setting. We did have problems ourselves until we learned how to apply consistently the adhesive patch, such that our diabetic children were then able to participate in a wide range of activities.

We re-emphasise the value of this type of indwelling cannula device for the administration of insulin to newly diagnosed diabetics and for those children with needle phobia. Clearly, further studies are required to determine whether such a device would be of practical benefit in a wider range of circumstances.

γδ T cells in human breast milk

SIR,—We previously reported that the proportion of lymphocytes bearing the γδ T cell receptor is significantly higher in human colostrum than in either autologous or heterologous blood samples.[1] Additional studies revealed that the great majority of colostral γδ T cells react with a monoclonal antibody (δTCS1) which recognises actively motile cells that express non-covalently bound γδ chains.[2] To characterise further the phenotype of this small, but numerically important, T cell subset, we have now examined eight milk lymphocyte suspensions with a more complete panel of monoclonal antibodies directed against T cell related antigens.

Direct and indirect immunofluorescence staining techniques followed by two colour cytofluorimetric analysis showed that only a few (15–35%) milk γδ T cells (TCR δ1$^+$) were CD8$^+$, whereas nearly all were CD4$^-$, CD5$^-$, and CD7$^-$. In contrast, an overwhelming preponderance (85–95%) of colostral γδ T cells displayed the HML-1$^+$ phenotype. This is the reverse of situation encountered in the bloodstream, where HML-1$^+$ T lymphocytes are virtually absent.

Although the origin of colostral T cells is still a matter of conjecture, the fact that the phenotypic pattern of milk γδ T lymphocytes is similar, if not identical, to that of the intestinal intraepithelial counterpart[3] suggests that these cells might originate in the gut associated lymphoid system and home to the mammary gland late in pregnancy and throughout lactation. This selective homing process by immune system cells has been well documented in experimental animal models.[4]

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Segmental colonic transit time in Duchenne muscular dystrophy

SIR,—We read with interst the recent paper by Korman *et al* concerning the orocaecal transit time in Duchenne muscular dystrophy.[1] In contrast to reports of gastric hypomotility and intestinal pseudo-obstruction, the authors found a normal orocaecal transit time. Therefore the genesis of patient's constipation is not due to impaired small bowel motility. Colonic motility had not yet been studied in Duchenne muscular dystrophy.

For this reason, we recently studied segmental colonic transit time in 12 patients, aged 8 to 18 years (mean 12·3 years). Eight of them were confined to a wheelchair. Gastrointestinal symptoms were noted and segmental colonic transit time was performed according to a method previously described[2]: 20 markers were given at breakfast time to the patients for three consecutive days and plain film of the abdomen was taken at the fourth and seventh days.

Ten children had at least one criteria of constipation: less than three stools per week (n=5), difficulties in defecation (n=7), and hard stools (n=3). Ten had gastrointestinal symptoms: abdominal pain (n=7) proctologic abnormalities (n=5), encopresis (n=2), and abdominal distension (n=2). Results of segmental colonic transit time are reported in the table. Seven of 12 children with Duchenne muscular dystrophy had an abnormal colonic transit time: three had stagnation of markers in the rectosigmoid area and four had an abnormal transit time in all the colonic segments. No relationship was found between colonic transit time and either gastrointestinal manifestations or gravity of muscular dystrophy.

Our results show that impairment of colonic transit time is frequent in Duchenne muscular dystrophy. Immobility, weakness of abdominal wall muscles and smooth muscle involvement of the colon[4] might explain the high frequency of constipation in these patients.

Segmental colonic transit time in 12 patients with Duchenne muscular dystrophy

Age (years)	*Right colon* (hours)*	*Left colon* (hours)*	*Rectosigmoid* (hours)*	*Total**	*Constipation*	*Other gastrointestinal symptoms*
8	6	1	13	20	+	+
9·5	2	1	41	44	−	+
10	30	35	10	75	+	+
11	14	6	8	28	+	+
11	2	1	28	31	+	+
12	24	20	41	85	−	+
13	12	14	47	73	+	−
14	1	11	29	41	−	+
14	13	1	41	55	+	−
15·5	59	61	0	120	+	+
16	4	4	30	38	+	+
18	50	37	7	94	+	+

*Upper limits of normal range of colonic transit time in French children are right colon, ≤18 hours; left colon, ≤20 hours; rectosigmoid, ≤34 hours; and total, ≤62 hours.[3]
Abnormal values underlined.
Plus (+) and (−) signs indicate present or absent.

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Surfactant treatment for premature babies—a review of clinical trials

SIR,—In the review article on surfactant treatment,[1] Survanta (Abbott Laboratories) is described as a frozen aqueous suspension. The current formulation of Survanta, recently approved for commercial use in the United States, is a suspension requiring refrigeration only. Before administration, Survanta vials are warmed to room temperature.

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1 Morley CJ. Surfactant treatment for premature babies—a review of clinical trials. *Arch Dis Child* 1991;66:445–50.

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