tion is to be prevented, the natural choice being ampicillin. It may be wise to supplement this with cloxacillin in case the condition is caused by a penicillinase-producing staphylococcus, but in patients who are allergic to penicillin the combination of chloramphenicol and lincomycin has been suggested.[2]

The point needs stressing that acute epiglottitis, uncommon though it is, occurs in adults as well as young children, and though it may start innocuously it is a rapidly progressive illness, with a high mortality from laryngeal obstruction and toxaemia. The diagnosis should be considered in an adult who presents with acute sore throat and minimal pharyngeal signs, but who has dysphagia, respiratory distress, and some voice change. The need for early tracheostomy, once symptoms of laryngeal obstruction have appeared, should be considered.

[1] Rainer, E. H., *Journal of Laryngology and Otology*, 1971, **85**, 493.
[2] Gorfinkel, J. H., Brown, R., and Kabins, S. A., *Annals of Internal Medicine*, 1969, **70**, 289.
[3] Vetto, R. R., *Journal of the American Medical Association*, 1960, **173**, 990.
[4] Johnstone, J. M., and Lawy, H. S., *Lancet*, 1967, **2**, 134.

Rickets in the Premature Baby

It is unwise to add medicine to an infant's feed, because he may leave some of the feed and therefore receive an inadequate dose of the medicine. Now a group of doctors[1] at the Sick Children's Hospital, Toronto, have shown that it is unwise to rely on vitamin-D-fortified milk to prevent rickets in premature and other infants of low birth weight, because the infants may leave some of the feed or otherwise take too little milk, so that the intake of the vitamin is inadequate. It had been the hospital custom to give vitamin D, 400 units per day, in addition to the milk formula, which contained 450 units of vitamin D per litre. In a six-month period four infants (weighing 680 g, 870 g, 935 g, and 1,300 g respectively, three of them small for dates) developed rickets. It was then found that as a result of the small feeds which they were taking they were receiving approximately 50 IU of vitamin D per day for the first month, and that only when they reached 2,500 g in weight did they take enough to obtain 300 IU per day.

It was noted that in the U.S.A. and Canada most fluid and evaporated milk contained approximately 450 IU of vitamin D per litre of reconstituted milk. In Great Britain the prophylactic and therapeutic doses of vitamin D are not accurately known, but it is commonly thought that 150-300 IU of the vitamin per day is probably adequate for prophylaxis and 300 to 500 IU for treatment. Unfortunately the question of the correct dosage is not simple. Paediatricians are afraid of overdosage, with resultant hypercalcaemia, and a quantity of vitamin D safe for one child may be toxic for another. M. Seelig[2] has suggested that American children may be receiving too much vitamin D for safety. Thirty-six years ago J. M. Lewis[3 4] showed that 90 units of crystalline vitamin D in milk was more effective in the treatment of rickets than 900 units in oil; and that 10 to 15 drops of vitamin D in oil were necessary to cure rickets, while only one drop was necessary if it was incorporated in milk. Others subsequently made similar observations. Hence it would seem that the prophylactic dose of vitamin D in oil may be different from that in fortified milk.

We have to strike a balance between risking the development of hypercalcaemia on the one hand and rickets on the other. Many doctors believe it is probably unnecessary and possibly unwise to give additional vitamin D to full-term babies fed on evaporated or dried milk or cereals —all of them in Britain fortified with vitamin D. But the work of P. K. Lewin and colleagues in Toronto[1] indicates that the daily intake of vitamin D by small babies of low birth weight may be inadequate because of the low quantities of fortified milk which they are able to take.

[1] Lewin, P. K., Reid, M., Reilly, B. J., Swyer, P. R., and Fraser, D., *Journal of Pediatrics*, 1971, **78**, 207.
[2] Seelig, M., *Clinical Pediatrics*, 1970, **9**, 380.
[3] Lewis, J. M., *Journal of Pediatrics*, 1935, **6**, 362.
[4] Lewis, J. M., *Journal of Pediatrics*, 1936, **8**, 308.

Reflux and Hernia

An exciting and provocative hypothesis about the anti-reflux mechanism of the stomach has recently been published and will cause consternation among the traditionalists, though it may not change the form of practical methods of treating the symptoms and complications of reflux for some time to come.

S. Cohen and L. D. Harris[1] found that when intra-abdominal pressure was increased the pressure within the lower oesophageal sphincter also increased. Increase in pressure in the lower oesophageal sphincter always exceeded the increase in intra-abdominal pressure (measured as intragastric) in a group of patients without symptoms of reflux, whether they had a hiatus hernia or not. On the other hand, increase in pressure in the lower oesophageal sphincter was always less than the increase in gastric pressure in another group of patients who had symptoms of reflux, again regardless of whether they had a hiatus hernia or not. The resting or baseline pressure in the sphincter of all the patients with symptoms of reflux was less than the pressure in those without symptoms.

The authors draw several conclusions: firstly, that the common sliding hiatus hernia does not by itself make the sphincter weaker; secondly, the pressure surrounding the sphincter does not affect its strength; thirdly, the sphincter becomes stronger as intragastric pressure increases, perhaps by a reflex process; and fourthly, the ability to become stronger is not affected by the sphincter's location above or below the diaphragm. We may also conclude that the ability to become stronger is related in some way to the resting pressure in the sphincter and not to the increase in intra-abdominal pressure. The weaker the sphincter initially, the less the increase in pressure as intragastric pressure rises, until intragastric pressure equals or exceeds sphincteric pressure and reflux occurs. The fault seems to lie within the sphincter. If it contracts well in the resting state, it will always overcome intragastric pressure by contracting even harder when challenged. If it is inherently weak, its response will be correspondingly inadequate. S. J. Rosenberg and Harris[2] suggest that incompetence of the lower oesophageal sphincter is not a primary defect of the muscle but is related to decreased stimulation by endogenous gastrin. They compared the dose response curves to pentagastrin of the lower oesopha-