dependent on the distance of the segment studied from the blind loop.

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Serum Phospholipid Levels in Growth Hormone Deficient Children. C. G. Theodoridis, P. H. W. Rayner, E. C. Albutt, and G. W. Chance. (*Department of Paediatrics and Institute of Child Health, University of Birmingham*). Serum total phospholipids were measured by Bartlett's (1959) method in 10 growth hormone deficient children aged 3–18 years and in 21 short stature children of similar age-group who were endocrinologically and metabolically normal.

The mean serum total phospholipid levels obtained from the HGH deficient children—273 ± 20 mg./100 ml. (range 250–315 mg./100 ml.)—were much higher than the mean obtained from the non-growth hormone deficient children—205·4 ± 22 mg./100 ml. (range 160–250 mg./100 ml.). This difference was statistically significant ($p < 0·001$). This difference could not be accounted for by thyroid deficiency as only 3 HGH deficient patients had associated TSH deficiency.

Despite raised serum total phospholipids the percentage distribution of serum phospholipid phosphorus between the 4 phospholipid fractions (separation was done by thin layer chromatography) was similar to that of the controls (Table).

TABLE

	GH Deficient Children	Control Children
Lysophosphatidyl choline	9·4±1·5	9·5±1·2
Sphingomyelin	20 ±0·4	20·4±0·5
Phosphatidyl choline	65·5±3	65·1±0·9
Phosphatidyl ethanolamine	5·1±1·9	5·0±0·7

A 20% fall in serum total phospholipid was observed in 3 children with isolated growth hormone deficiency, who received growth hormone treatment for 3 months.

In summary, raised total phospholipid levels are demonstrable in children with HGH deficiency. The administration of exogenous HGH leads to a fall of serum total phospholipids. There is no change in the percentage distribution of serum phospholipid phosphorus between the 4 phospholipid fractions in these children.

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Vitamin E Therapy in A-β-lipoproteinaemia. D. R. P. Muller, J. T. Harries, and June K. Lloyd (*Institute of Child Health, London*). A-β-lipoproteinaemia is a rare inborn error of metabolism characterized by the absence of β-lipoprotein from the blood, acanthocytosis of the red cells, and steatorrhoea (present from birth), and an ataxic neuropathy and pigmentary retinopathy which develop later and are slowly progressive.

We have investigated the vitamin E status of 6 children with a-β-lipoproteinaemia by estimating serum concentrations of vitamin E and red cell haemolysis. Initially serum vitamin E was undetectable in all of the children, and haemolysis raised (> 6%) in the 5 in whom it was estimated. Oral administration of large doses of vitamin E resulted in rapid correction of the abnormal haemolysis. Serum vitamin E remained undetectable for approximately 6 months in 4 children who received doses varying between 25 and 75 mg./kg. per day, whereas in 2 children who received even larger doses (100 mg./kg. per day) serum vitamin E was detectable after 1 and 3 months, respectively. The maximum level achieved by oral therapy was 0·24 mg./100 ml. (normal — 0·44 mg./100 ml.) which approached the peak levels of 0·3 mg./100 ml. obtained in 2 children who received a large, single intramuscular load.

In 4 children oral therapy has now been given for longer than 3 years, and the retinal and neurological abnormalities present in 2 children have been studied sequentially by tests of motor nerve conduction and retinal function; one child has shown no deterioration and the other a definite improvement. As the natural history of the condition is towards progressive deterioration, and as no other form of therapy was introduced during administration of vitamin E, it seems likely that this vitamin has contributed to the improvement.