ferment galactose, raffinose, inulin, erythrol, mannitol nor dulcitol. Neither does it liquefy gelatin nor coagulated blood serum. Hydrogen sulphide and indole are not formed. Litmus and methylene blue are strongly reduced, but nitrates are not.

The organism is pathogenic for mice, guinea pigs, rabbits and rhesus monkeys, freshly isolated cultures generally being fatal within 72 hours after intravenous or intracerebral inoculation. Evidence of meningeal irritation and incoordination is frequently prominent. Rabbits seem most susceptible. The most characteristic gross lesions are minute (0.5-1 mm.), yellowish, opaque miliary lesions which may be located in the meninges, brain, kidney, adrenal, heart muscle, spleen and liver. The adrenal may be markedly congested. Microscopically there are small foci of necrosis, surrounded and invaded by proliferating histiocytes and leucocytes and frequently interstitial hemorrhage. In the central nervous system, the lesions are found particularly in the leptomeninges and the adjacent nervous tissue. The organisms are easily cultivated from infected tissues.

Further studies on the properties of the organisms are in progress. The evidence at hand indicates that systematically it falls in the so-called diphtheroid group. We suggest for it the name *Corynebacterium parvulum.*

7423 P

Phosphatase Activity of the Serum and Tissues in Osteogenesis Imperfecta.

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Robison[1] has pointed out that the enzyme phosphatase is an important factor in the calcification of bone. Kay[2] has more recently summarized the evidence for its activity in connection with studies on a wide variety of clinical conditions. Since osteogenesis imperfecta is an outstanding example of defective bone formation for which no pathogenesis has as yet been discovered, a comprehensive study of the various metabolic factors which might be involved in this condition was begun by the writer in 1931. The present report deals with the phase of that study which concerns the phosphatase activity of the blood serum and fixed tissues.

[1] Robison, R., *Biochem. J.*, 1923, **17**, 286.

[2] Kay, H. D., *J. Biol. Chem.*, 1930, **89**, 235, 249.

Kay reported the serum phosphatase activity to be slightly increased in some cases of osteogenesis imperfecta, while Bodansky and Jaffe[3] found it to be normal in 6 cases.

Periodic determinations of the serum phosphatase by the method of Kay have now been made in 4 cases of osteogenesis imperfecta under a variety of experimental conditions. In one case the fixed tissue phosphatase has also been studied postmortem.

Repeated determinations in the 4 cases showed values almost identical with those found for normal control subjects and for a miscellaneous group of patients in the same age period. Variations in the ordinary constituents of the diet were found to have no definite effect on the serum phosphatase values. Following the administration of vitamin D in relatively enormous doses in the form of viosterol 10,000X, however, the phosphatase activity of the serum was uniformly reduced. Parathormone administration over a period of several days had a similar but less marked depressing effect on the enzyme.

The sudden death of a 7-year-old girl with osteogenesis imperfecta, at a time remote from any unusual medication and when no demonstrable infection was present, made possible a study of the phosphatase activity of the blood serum and fixed tissues a few hours postmortem. The serum values were normal. Figures found for the various tissues were as follows:

Tissue	Units Phosphatase*
Liver	0.12
Muscle	0.17
Brain	0.14
Kidney	0.28
Intestine	
Duodenum	0.00
Jejunum	0.09
Ileum	0.40
Cecum	0.21
Colon	0.39
Bone	
Ulna, upper end	0.83
Ulna, lower end	0.50
Ulna, shaft	0.65
Periosteum and subperiosteum	0.00
Rib	0.76
Callus rib	1.05

* Expressed in mg. phosphorus liberated by 1 gm. of tissue in 3 hours at 38°C. and at pH 8.8.

[3] Bodansky, A., Jaffe, H. L., *Proc. Am. Soc. Biol. Chemists*, 1934, 11.

The most striking feature of the results was the almost complete absence of phosphatase in the periosteum and sub-periosteal structures, where it is normally abundant. This observation suggests a relationship between the extremely defective calcification of the cortex and the lack of phosphatase. That phosphatase may be mobilized from other sources under special conditions, however, is indicated by the greatly increased activity in the region of callus formation.

The extremely low phosphatase activity found in the duodenum may be of significance in relationship to defective phosphorus absorption. Normally the duodenum shows a higher phosphatase activity than other segments of the gut.

7424 P

Prophylactic Vaccination Against Intracranial Complications Following Pneumococcus Type III Mastoiditis.

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Inasmuch as intracranial complications following pneumococcus Type III mastoiditis are known to occur, as a rule, not earlier than several weeks after the initial infection, serious attempts to induce a state of active acquired immunity should receive special consideration. The use of prophylactic vaccination was suggested by Kolmer and Amano on the basis of their work in rabbits in which immunization with pneumococcus vaccines elicited protection against experimental meningitis.[1] Barach found that type-specific active acquired immunity appeared within 6 days after intradermal or intravenous injection of pneumococcus type-heterologous vaccines in cases of lobar pneumonia.[2]

Accordingly, in September, 1931, we undertook to immunize every case of pneumococcus Type III mastoiditis admitted to the Otological service of The Mount Sinai Hospital as soon as the bacteriological diagnosis was made, whether that be before or after the operation. The following method of vaccination was employed:

[1] Kolmer, J. A., and Amano, K., *Arch. Otolaryngology*, 1931, **14**, 125; 1932, **15**, 547.

[2] Barach, A. L., PROC. SOC. EXP. BIOL. AND MED., 1928, **25**, 558; *J. Exp. Med.*, 1931, **53**, 567.