vation of the parathyroids is the retention of phosphorus and decrease or an absence of this element in the urine. The decreased excretion of calcium, which follows, may be the result of the parathyroid extirpation primarily or it may be due to the increased phosphorus retention and the retained calcium is thus deposited in the tissues as the insoluble salt.

The relation of hypoparathyroidism to abnormal calcification such as in Paget's disease, juvenile sclerosis, renal sclerosis, otosclerosis, and general arteriosclerosis is worthy of consideration. The association of a low serum calcium and low calcium excretion in otosclerosis has been frequently observed by otologists; and the occurrence of cataracts in parathyroid tetany in human beings and experimental animals has been noted by many observers. The association of extreme calcification of the vascular system and renal disease has been noted clinically and pathologically and it is possible that the parathyroid plays a rôle in this syndrome.

The rôle of the parathyroids and high phosphorus diets, such as meat, in general arteriosclerosis of the middle aged can only be speculated upon at present.

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Note on Individual Differences in Human Blood.

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1. Previously an agglutinable property of human blood designated as P was described, which could be demonstrated by the use of immune agglutinins from rabbits,[1] and it was mentioned[2] that reactions of a more or less similar specificity occur with a certain type of irregular human isoagglutinins. Inasmuch as these 2 reagents are not always easily available it seems worth mentioning that similar results can readily be obtained with various absorbed normal animal sera, *e. g.*, those of horses, pigs and rabbits. In horse serum the agglutinins were found rather frequently.

2. An anti-dysentery immune serum from a goat was shown by Eisler[3] to contain agglutinins for human blood which can be ab-

[1] Landsteiner, K., and Levine, Philip, *J. Exp. Med.*, 1928, **47**, 757.

[2] Landsteiner, K., and Levine, Philip, *J. Immunol.*, 1930, **18**, 91.

[3] Eisler, M., *Z. f. Immunitätsf.*, 1930, **67**, 38.

sorbed by dysentery bacilli (Shiga). Using this serum, kindly supplied to us by Eisler, we were able to confirm fully these interesting observations. Furthermore we found that after absorbing the serum with cells of the sub-group A^1 the supernatant fluid agglutinated most cells of sub-group A^2, O, and probably B, distinctly more strongly than those of sub-group A^1, in a similar way as certain exceptional human sera.[4] Whether this is a singular occurrence or can be reproduced with other dysentery sera is being investigated.

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The Oligodynamic Haemolytic and Haemagglutinative Properties of Some of the Heavy Metals.

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An accidental observation that $HgCl_2$ in high dilution was powerfully lytic for erythrocytes suspended in an isotonic medium led to the collection of data concerning the effect of many of the commoner salts in concentrations ranging from 0.01M to 0.0000003M. Progressively doubled dilutions in 0.85% NaCl solution were mixed with equal quantities of washed human cells in 2% suspension, placed, with a fragility test, in a water bath at 37°C. for 1 hour and read finally after some 20 hours' refrigeration at 8°C. The effect of the active salts was decidedly more in evidence at the 20 hour than at the 1 hour reading. The positive results recorded do not pretend to quantitative accuracy because of the considerable variations sometimes observed in repeated tests with cells of slightly different fragility.

Some salts were tested also with sheep and with rabbit cells; the latter were rather more sensitive to lysis and were involved in a peculiar phenomenon that was not observed with the other 2 types of erythrocytes. In the weakest partially effective lytic dilutions of the mercury salts the unhemolyzed cells were gathered in a tough film.

The hemolytic effect of the mercurials was surprisingly little diminished by the presence of even an excessive quantity of serum in the isotonic diluting medium.

[4] Landsteiner, K., and Levine, Philip, *J. Immunol.*, 1929, **17**, 8, 11.