apy. The data suggest that excess of calcium, rather than the agent or disease process producing it, is ultimately responsible for the defect.

The concentrating defect was not attributable to an increased solute excretion, which showed negligible changes with improvement.

All subjects but one showed normal sodium conservation with urinary sodium less than 9 mEq per day at this intake, suggesting that the concentrating defect does not depend upon gross failure of sodium transport.

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CORRECTION

On page 590 of the paper entitled "Isozymes of lactic dehydrogenase in human tissues" by Elliot S. Vesell and Alexander G. Bearn (J. clin. Invest. 1961, **40**, 586), lines 12 and 13 of the Summary should read: "Leukocytes and serum showed highest activity in peak 4."