clinical manifestations of the disease. There is no increase in lipids within the kidney. There was no real acidosis, and the only suggestion at one time during the patient's course of some abnormality in terms of renal function was a diffuse aminoaciduria, which rapidly cleared.

R. BAEHNER (Children's Hospital Medical Center, Boston, Massachusetts): BAGGIOLINI, HIRSCH, and DE DUVE [J. Cell Biol. *40:* 529, 1969] have shown differences in subcellular distribution patterns for acid phosphatase, in rabbit leukocytes, using different chemical substrates. Have you assayed for acid phosphatase using substrates such as β-glycerophosphate or phenolphthalein instead of p-nitrophenylphosphate?

Dr. NADLER: We have used both of those, and essentially the same thing is found. One of the differences is that the β-glycerophosphatase is essentially confined to lysosomal fractions at all times, and there is no detectable activity in this patient using that particular method.

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