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**Biologic Decay Periods of Chloride in Man with Use of Long-Life Cl^{36}
as Tracer.* (18371)**

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From the data obtained in tracer studies with radiochloride,[†] Cl^{36} ($t_{1/2} \approx 2 \times 10^6$ yrs.), it was possible to estimate the biologic decay periods of this element for human subjects. A knowledge of the biologic decay rates of chloride in man is of importance in the understanding of the physiologic turnover and exchange rates in normal subjects, in patients with edematous states, including congestive heart failure, and in individuals internally contaminated as a result of military and

peaceful applications of nuclear energy. From previous investigations with Na^{22} in man and Cl^{36} in dogs, estimations of biologic decay rates of chloride in normal man with relatively constant compartment size were made (2-5). The observed biologic decay rates agree with the estimated values.

Cl^{36} (10,000,000 CPM or approximately

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TABLE I.

Subject	Mean values in days			Injected Cl ³⁶ recovered, %
	C _{.5} *	E _{.5} †	U _{.5} ‡	
Control, 501	15	16	17	96
Mild congestive heart failure, 502	19.5	38	48	69
Severe congestive heart failure, 503	45	65	169	48

* C_{.5} = Time in days required for concentration of Cl³⁶ in serum to reach one-half at any time after equilibrium of distribution.

† E_{.5} = Time in days required for one-half of total Cl³⁶ administered to be eliminated from body by all routes (Feces, urine, vomitus, and pleural and ascitic fluids were collected in these studies).

‡ U_{.5} = Time in days required for one-half of total Cl³⁶ administered to be eliminated in urine.

50 μ c) as NaCl in water was administered intravenously to 3 female subjects: Subject No. 501, 31 years old with previous hyperthyroidism controlled with propylthiouracil; Subject No. 502, 62 years old with arteriosclerosis, essential hypertension, mild diabetes and mild congestive heart failure, who was made to compensate or allowed to decompensate, as desired, during the course of the study; Subject No. 503, 59 years old with hypertensive and arteriosclerotic heart disease with severe congestive heart failure, who died during the thirty-seventh day of study. The other 2 subjects were studied for 70 continuous days.

Blood was collected once daily under oil and immediately centrifuged; the serum was assayed as described previously(1,3,5). Urine and stools were collected as excreted and were also assayed. Any vomitus or other fluids removed, such as ascitic or pleural fluid, were likewise studied in order to determine the total elimination of Cl³⁶ from the body.

The mean biologic decay periods for the entire study for each subject are presented in Table I. Curves for the decrease in concentration of Cl³⁶ in the serum, urinary excretion (percentage of Cl³⁶ not excreted in the urine) and total recovered elimination (percentage of injected Cl³⁶ not recovered) for Control Subject No. 501 are shown in Fig. 1. The relationships of changes in rates to changes in intake of Cl³⁵ are evident. The importance of these influences as well as the importance of changes in compartment size have been discussed previously for radiosodium(2,5) and

have been applied here. The biologic decay periods for Cl³⁶ varied considerably, being influenced by intake of chloride, variations in size of the chloride compartments, state and progress of the congestive heart failure, and drugs (mercurial diuretics, desoxycorticosterone acetate, bromide, sodium, and cathartics). These and other considerations will be presented in detail elsewhere.

INFLUENCE OF INTAKE ON RATES OF ELIMINATION AND ON CONCENTRATION OF CL³⁶ IN SERUM

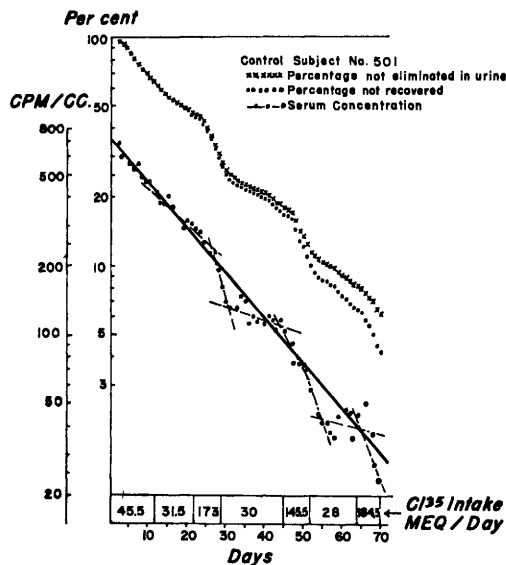


FIG. 1.

Graph of changes in concentration of Cl³⁶ in the serum (CPM/cc), rate of urinary excretion of Cl³⁶ (percentage of injected Cl³⁶ not eliminated in the urine), and rate of elimination of total recovered Cl³⁶ (percentage of injected Cl³⁶ not recovered) in Control Subject No. 501. Influences of variations in intake of chloride upon these curves are indicated.