Absorption of Iron from the Gastrointestinal Tract

A COMPARATIVE STUDY OF THE ORAL IRON TOLERANCE TEST IN HUMAN BEINGS USING STABLE AND RADIOACTIVE IRON

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THE SIGNIFICANCE of changes in serum iron levels after the ingestion of large doses of stable iron has been questioned as a valid index of iron absorption.[1,2,3,4] It is apparent that not only the rate of absorption, but also the rate of removal of iron from the blood determines the serum levels.

Previous work in this laboratory has utilized an oral iron tolerance test with stable iron in an endeavor to study iron absorption in states of health and disease.[5] This study was undertaken in an attempt to evaluate the accuracy of the Ramsey method[6] for the determination of serum iron by comparing the recovery of stable iron in the serum after oral administration with the recovery of simultaneously administered radioactive iron in the serum. Furthermore, it was desired to ascertain whether or not these values for serum recovery bear any relationship to the total absorption of Fe^{59} as determined both by stool analysis and recovery of Fe^{59} in the hemoglobin.

METHODS

An iron tolerance test was performed. In most instances stable iron was given in the form of ferrous ammonium sulfate and radioactive iron as ferric chloride (Fe^{59}). In normal subjects the absorption of ferric stable and ferric radioactive iron was studied with and without the addition of ascorbic acid. In a small series ferrous stable and ferrous radioactive iron (Fe^{59}, as ferrous gluconate) were given for comparison with the other groups. Stable iron in the amount of 1 mg per kilogram of body weight and radioactive iron in tracer doses of the order of 10 to 15 microcuries (0.001–0.016 mg elemental iron per microcurie) were given orally. The radioactive iron was purchased from the Abbott Laboratories.

All subjects were given the iron (both stable and radioactive) by mouth, after a fast of 10 to 16 hours. They were allowed no food until after the completion of the test. Specimens of venous blood were drawn immediately preceding the test and hourly thereafter for four hours. Serum iron levels were determined by the Ramsey method.[6] Values of 100–170 μg per 100 ml were considered normal fasting levels in our laboratory. Latent iron-binding capacity was determined by the method of Rath and Finch,[7] using a Klett colorimeter (Model #800-3). Estimations of per cent recovery of stable iron in the serum were calculated from the difference at each hour between the serum iron and the fasting serum iron, multiplied by the plasma volume, and divided by the total dose of stable

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iron. Plasma volume was estimated by means of the hematocrit and an assumed blood volume of 84 ml per kilogram in males, and 78 in females.[8,9]

Radioactivity in the samples was measured in the following ways. Standard for the test dose was a 2-ml aliquot taken from a 100-ml volumetric flask containing the test dose of ferric or ferrous radioactive iron as the chloride or the gluconate. This aliquot was immediately made up to 100 ml with water acidified with concentrated sulfuric acid to a pH between 1 and 2. This was necessary to prevent precipitation of iron and its consequent adherence to the glassware. Five-ml aliquots of the acidified solution were counted for radioactivity in a gamma-ray scintillation well-type counter (Nuclear-Chicago Model #DS 3). Counting of radioactivity was performed to greater than 2 per cent statistical accuracy on samples of over 1000 counts per minute and to at least 5 per cent statistical accuracy on samples of less than 1000 counts per minute. Background averaged 128 counts per minute.

Specimens of 5 ml of serum were pipetted into iron-free tubes and measured for radioactivity prior to chemical analysis. Measurements of stable iron were subsequently carried out on these same aliquots of sera. Blood was taken into a bottle containing dry potassium oxalate, a 5-ml sample was pipetted immediately into a measuring tube, and, after hemolysis had occurred by freezing and thawing, it was read for radioactivity. In estimating the activity of the blood, the blood volume assumption was as above. Blood was drawn three times weekly in the second and third weeks after the tracer dose was given in early subjects (Table IIa, b, c), and after one to two months in later ones (Tables Ia, b, c, and IId, e).

The feces passed each day were collected and weighed. Each specimen was homogenized in an electric blendor and transferred in 5-ml aliquots to a weighed tube for counting. The weight of each 5-ml aliquot was determined, and total recovery of radioactivity estimated on this basis. Fecal collections were continued until the activity of the stools on two successive days measured less than 1 per cent of the administered dose. Later stool collections were limited to seven days; as in most earlier subjects all appreciable radioactivity was excreted in this interval.

Iron absorption studies were performed on 34 subjects. Twelve subjects were considered as normal controls; one of these was recovering from a spontaneous pneumothorax. Five subjects convalescent from coronary occlusion (three weeks post coronary occlusion) and one subject with axillary vein thrombosis and pulmonary infarction, all of whom appeared hematologically normal, were originally selected as controls, but this group will be considered separately as their absorption data differed somewhat from those of the healthy controls. Seventeen other patients with a wide variety of diseases were studied by the iron tolerance test.

RESULTS AND INTERPRETATION

In the healthy group several combinations of iron salts (in reduced and unreduced states, with and without ascorbic acid as a reducing agent) were administered. Three subjects received both ferric stable and ferric radioactive iron without ascorbic acid (Table Ia); in this group only meager absorption of iron was demonstrated, either in serum or hemoglobin (in fact radioactivity was too low for satisfactory counting to statistical accuracy), and recovery of administered Fe_{59} in the feces was maximal. A comparable group of controls (Table Ib) who received the same combination of iron salts plus 500 mg of ascorbic acid administered simultaneously showed excellent iron absorption, even at one hour, and the recovery of Fe_{59} in the stools was proportionately low (Fig. 1). Comparably good results were obtained in two control subjects receiving both stable and radioactive ferrous iron (Table Ic). Four normal controls were given stable ferrous iron and radioactive ferric iron (Table Id); in this group recovery of stable iron in the serum was as large as that of the reduced ferric (Table Ib) and the ferrous series (Table Ic) (Fig. 2). However, recovery of radioactive iron in serum was consistently less than that of stable iron, and recovery of radioactive iron in hemoglobin and its estimated absorption by the stool technique

were somewhat less than in the other control groups (Table Ib, c) in which the tracer was given in the reduced state. It has been shown that a large quantity of ferrous iron is capable of reducing ferric iron,[10] and the demonstrable absorption of the isotope in the series shown in Table Id confirms this phenomenon. However, the tendency to a smaller recovery of Fe^{59} than of stable iron in the serum samples suggests that the reduction of the iron to the ferrous state was only partial, at least during the four-hour period devoted to serum collection.

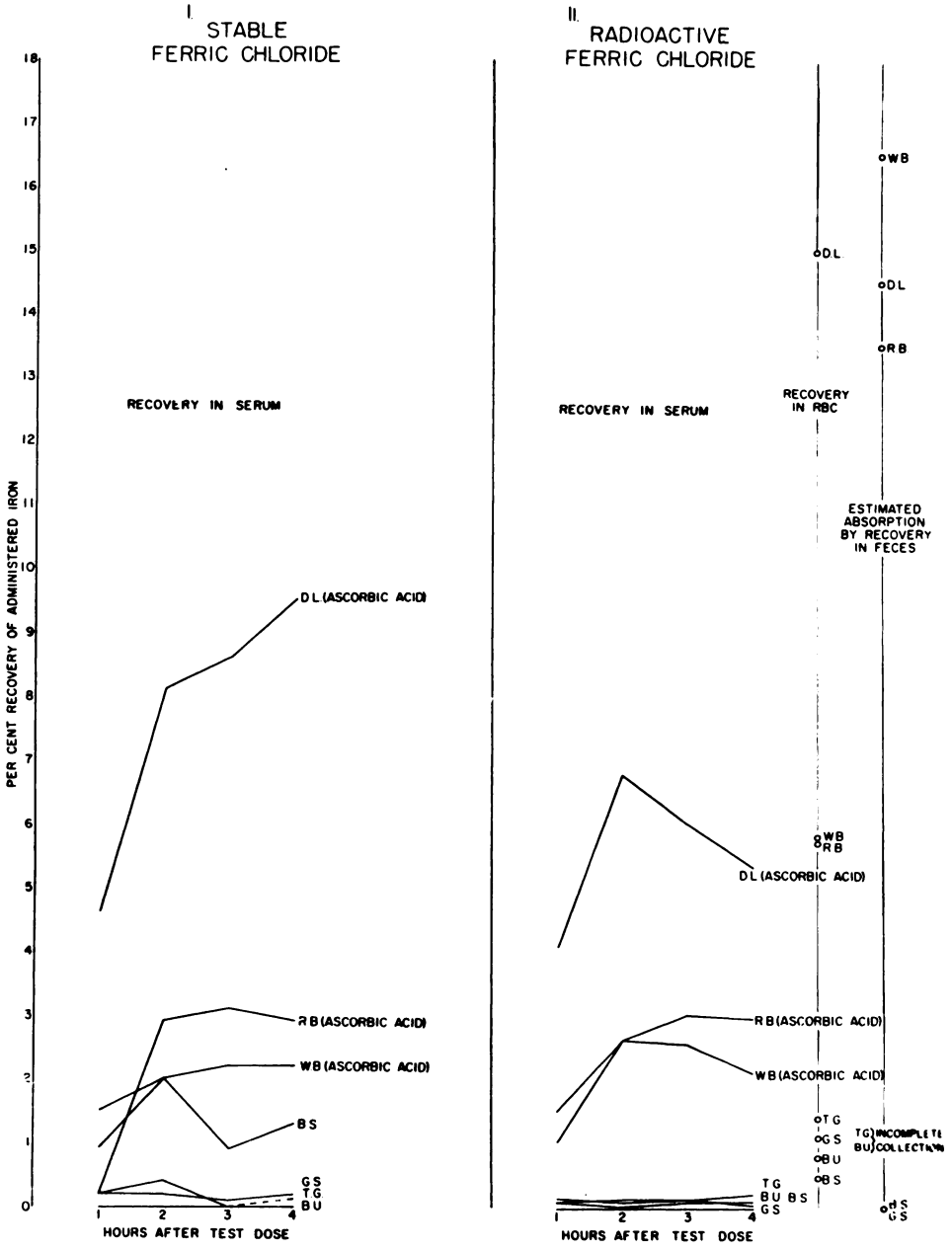

Fig. 1. Response of normal subjects to ferric stable and radioactive iron, with and without simultaneously administered ascorbic acid.

TABLE I. Normal Subjects

Subject	Age	Sex	Wgt.	Hct.	Latent iron-binding capacity	Fasting serum Fe		% Recovery of iron, hours 1	2	3	4	Maximum rise serum Fe	% Recovery of Fe^{59} Stool	Blood	Serum	Diagnosis
						μg/100 ml						*μg/100 ml*				
a. Ferric Stable, Ferric Fe^{59}																
B. U.	31	M	56	48	250	125	Isotope	0.12	0.07	0.10	0.11	2	89.0*	0.8	0.1	Normal
							Stable	0.9	2.0	0.9	1.3					
B. S.	27	M	78.4	48	200	110	Isotope	0.17	0.12	0.14	0.08	45	113.0	0.5	0.2	Normal
							Stable	0.9	2.0	0.9	1.3					
T. G.	24	M	68	48	200	200†	Isotope	0.11	0.15	0.14	0.21	10	98.8	1.4	0.2	Normal
							Stable	0.2	0.4	0	—					
b. Ferric Stable, Ferric Fe^{59} + Ascorbic Acid																
D. L.	27	M	77	48	225	165	Isotope	4.0	6.8	6.0	5.3	115	85.0	15.1	6.8	Normal
							Stable	4.6	8.1	8.6	9.0					
R. B.	27	M	73	42	—	160	Isotope	1.1	2.6	3.0	3.0	70	86.0	5.7	3.0	Normal
							Stable	0.2	2.9	3.1	2.9					
W. B.	25	M	63.6	43	200	165	Isotope	1.5	2.6	2.6	2.1	50	83.0	9.6	2.6	Normal
							Stable	1.5	2.0	2.2	2.2					
c. Ferrous Stable, Ferrous Fe^{59}																
P. K.	19	M	57	49	200	125	Isotope	2.5	3.1	2.9	2.4	95	88.9	9.9	3.1	Spontaneous pneumothorax (no anemia)
							Stable	2.0	3.5	4.6	4.6					
M. D.	20	M	75	46	250	140	Isotope	3.1	3.3	2.8	2.0	90	95	13.3	3.1	Normal
							Stable	3.1	3.3	3.5	4.0					
d. Ferrous Stable, Ferric Fe^{59}																
E. H.	30	M	66	46	200	160	Isotope	3.0	3.2	2.4	0.3	135	91	8.0	3.2	Normal
							Stable	3.5	5.9	4.4	—					

TABLE I. Normal Subjects—*cont'd.*

Subject	Age	Sex	Wgt.	Hct.	Latent iron binding capacity	Fasting serum Fe		% Recovery of iron, hours				Maximum rise serum Fe	% Recovery of Fe^{59}			Diagnosis
								1	2	3	4		Stool	Blood	Serum	
					μg/100 ml	*μg/100 ml*						*μg/100 ml*				
d. Ferrous Stable, Ferric Fe^{59}																
B. H.	34	M	89	42	250	140	Isotope	2.3	3.0	2.7	0.9	180	92	6.3	3.0	Normal
							Stable	2.2	7.9	5.7	—					
D. B.	32	M	80	45	—	130	Isotope	0.2	0.4	0.2	0.2	140	94	4.0	1.2	Normal
							Stable	1.3	2.2	2.2	—					
H. E.	26	M	66	45	—	160	Isotope	0.8	1.4	1.1	0.5	130	95	2.4	1.4	Normal
							Stable	1.7	5.7	4.6	4.0		Mean 7.0% absorption	Mean 5.2%		

* Incomplete collection.
† Slightly higher than our usual "normal" range; unexplained.

In summary, normal subjects showed a prompt rise in serum iron concentration after the ingestion of ferrous stable iron, followed by the appearance of radioactive iron in the hemoglobin with a mean of 5.2 per cent (Table Id) if the isotopic iron was administered in its ferric form simultaneously with a large quantity of ferrous stable iron; and the total absorption based on stool analysis gave a mean value of 7 per cent (Table Id). Better absorption of isotopic iron was demonstrated when the tracer was either given in the reduced form or was reduced simultaneously with stable iron by a large dose of ascorbic acid.

The iron tolerance test using ferrous stable iron and ferric radioactive iron was employed in the majority of patients studied, as the ferrous isotope was not available to us until later in the study. Though there were several exceptions, the tendency to greater recovery of stable iron than of radioactive iron in the serum occurred in the patients as well as in the normal subjects, and can probably be explained by incomplete reduction of the radioactive iron. The absorption of iron in these patients varied from greater than the controls in subjects with evidence of iron deficiency (Table IIa)(Fig. 2), to very poor absorption in several patients (Table IIb). The bulk of the patients appeared to absorb iron as well as the controls (Table IIc); in several of this group normal hematocrits were observed, but a depression of the fasting serum iron and a slight elevation of the latent iron-binding capacity (L.I.B.C.) suggested iron deficiency[7] (S. C., M. S., J. Su.), and in these the degree of absorption was only slightly greater than or the same as for the rest of the group. This was not a constant observation, however, as one patient (R. B.) with higher than normal latent iron-binding capacity and lower than normal fasting serum iron demonstrated evidence of slightly less absorption.

No definite correlation of the type of disease process and the degree of iron absorption can be made from this small series, and hence no statistical analysis has been attempted. Several specific cases are worthy of mention, how-

TABLE II Patients

Subject	Age	Sex	Wgt.	Hct.	Latent iron-binding capacity	Fasting serum Fe		% Recovery of iron, hours 1	2	3	4	Maximum rise serum Fe	% Recovery of Fe^{59} Stool	Blood	Serum	Diagnosis
						μg/100 ml						μg/100 ml				
a. Ferrous Stable, Ferric Fe^{59}																
N. H.	63	F	50	32	325	52	Isotope	8.2	7.2	6.1	5.1	173	76	—	8.2	Pancreatitis CVA ASHD, (hypochromic anemia)
							Stable	3.7	6.1	9.3	8.0					
S. W.	81	F	55	40	400	80	Isotope	10.2	11.5	10.3	8.7	370	70	26.8	11.5	Carcinoma of esophagus (hypochromic anemia)
							Stable	7.0	15.9	17.1	17.3					
T. C.	50	F	57	30	400	45	Isotope	7.4	5.8	4.6	3.7	195	78	24.5	7.3	Lupus erythematosis (hypochromic anemia 2° to menstrual blood loss)
							Stable	6.3	10.7	10.1	8.5					
b. Ferrous Stable, Ferric Fe^{59}																
F. O.	51	F	84	35	100	105	Isotope	0.8	0.4	0.2	<0.1	100	98	—	0.8	Myelogenous leukemia (chronic) (transfusions)
							Stable	1.5	0.8	0.0	0.0					
A. G.	53	F	36	39	150	150	Isotope	0.6	0.6	0.5	0.5	70	98	1.7	0.6	Sprue syndrome
							Stable	5.7	5.7	5.7	2.4					
F. H.	58	M	55	50	100	240	Isotope	0.4	0.3	0.4	0.4	0	98	2.2	0.4	Hemochromatosis (biopsy of liver)
							Stable	0.0	0.0	0.0	0.0					
P. P.	49	M	88	50	200	220	Isotope	0.6	0.9	0.9	1.0	25	98	—	1.1	Diabetes, cirrhosis, gangrene (no hemachromatosis)
							Stable	0.8	1.1	0.8	0.6					
J. F.	29	F	58	38	225	65	Isotope	0.5	0.5	0.3	0.1	95	*	—	0.5	Hypochromic, mycrocytic anemia (?thalassemia minor)
							Stable	3.1	3.1	0.7	0.2					
c. Ferrous Stable, Ferric Fe^{59}																
M. O.	55	F	40	30	150	80	Isotope	3.1	3.1	2.8	2.0	110	*	—	1.3	Chronic nephritis, uremia
							Stable	6.6	6.0	6.0	6.0					
M. C.	72	F	54	40	150	70	Isotope	2.4	2.3	2.0	1.3	92	95	—	2.4	Laennec's cirrhosis (compensated)
							Stable	3.3	4.2	4.4	3.7					
J. M.	70	M	55	20	0	190	Isotope	3.5	1.5	1.2	0.7	45	91	—	3.4	Agnogenic myeloid metaplasia (hemolytic
							Stable	3.0	0.8	0.7	0.7					

Subject	Age	Sex	Wgt.	Hct.	Latent iron-binding capacity	Fasting serum Fe		% Recovery of iron, hours 1	2	3	4	Maximum rise serum Fe	% Recovery of Fe^{59} Stool	Blood	Serum	Diagnosis
						μg/100 ml						μg/100 ml				
H. F.	45	F	57	30	150	150	Isotope	3.9	2.0	2.2	4.3	70	88.5	12.2	3.9	Lupus erythematosis, and septicemia
							Stable	2.2	3.8	3.8	1.6					
S. C.	27	F	53	41	225	90	Isotope	2.5	3.0	3.7	2.0	130	89	12.0	3.7	Infectious mononucleosis, myocarditis
							Stable	3.2	8.2	8.2	7.6					
M. S.	51	F	77	36	375	45	Isotope	5.2	5.0	4.7	3.9	150	*	—	5.2	Diabetes, femoral thrombophlebitis
							Stable	3.2	7.2	8.0	7.7					
A. B.	67	F	78	36	—	120	Isotope	2.7	3.5	3.2	2.3	175	*	—	3.5	Muscular dystrophy
							Stable	3.5	8.7	8.7	7.5					
J. Su.	63	M	88	32	350	110	Isotope	0.4	0.7	0.5	0.3	130	90	8.6	3.4	Hypochromic anemia—?, 2° to peptic ulcer
							Stable	1.1	6.3	6.3	6.3					
R. B.	53	M	50	39	325	40	Isotope	1.0	2.5	3.0	3.0	110	92	—	2.6	Rheumatic heart disease, chronic CHF
							Stable	4.1	5.6	5.4	5.1					
							d. Ferrous Stable, Ferric Fe^{59}									
J. P.	47	M	85	46	—	180	Isotope	1.5	1.7	1.3	0.7	90	96	3.5	1.7	Coronary occlusion (no anemia)
							Stable	2.7	4.1	3.9	0.5					
J. W.	41	M	61	43	150	180	Isotope	0.3	0.3	0.2	0.1	40	99	0.6	0.3	Axillary vein thrombosis, pulmonary infarction (no anemia)
							Stable	1.9	1.9	1.7	0.0					
							e. Ferrous Stable, Ferrous Fe^{59}									
C. T.	64	M	55.6	47	200	135	Isotope	1.0	1.3	1.1	1.9	40	87.3*	—	1.3	Coronary occlusion (3 weeks), ASHD (no anemia)
							Stable	1.8	1.8	1.3	1.1					
J. A.	50	M	79.6	49	200	80	Isotope	0.5	0.5	0.4	0.3	40	94.4	—	0.5	Coronary occlusion (3 weeks) (no anemia)
							Stable	0.7	0.4	1.5	1.8					
J. M.	44	M	61.6	45	—	75	Isotope	0.6	1.2	1.1	0.8	30	—	9.2	1.2	Coronary occlusion (4 weeks) (no anemia)
							Stable	0.0	0.9	1.3	0.9					

* Incomplete collection. CVA—cerebrovascular accident. ASHD—arteriosclerotic heart disease. 2°—secondary to. CHF—congestive heart failure.

ever. One patient (F. H.) with hemochromatosis (proved on biopsy) appeared to absorb less iron than the controls; this is not consistent with recent reports in the literature.[11,12] One patient (J. Su.) with hypochromic anemia and evidence of iron deficiency by fasting serum iron level and latent iron-binding capacity appears to have had delayed absorption, as the serum recoveries are low, but the stool and hemoglobin recoveries are consistent with the other iron-deficient patients. This discrepancy may have been due to delayed gastric emptying, as this patient had a peptic ulcer and may have had some degree of duodenal obstruction,

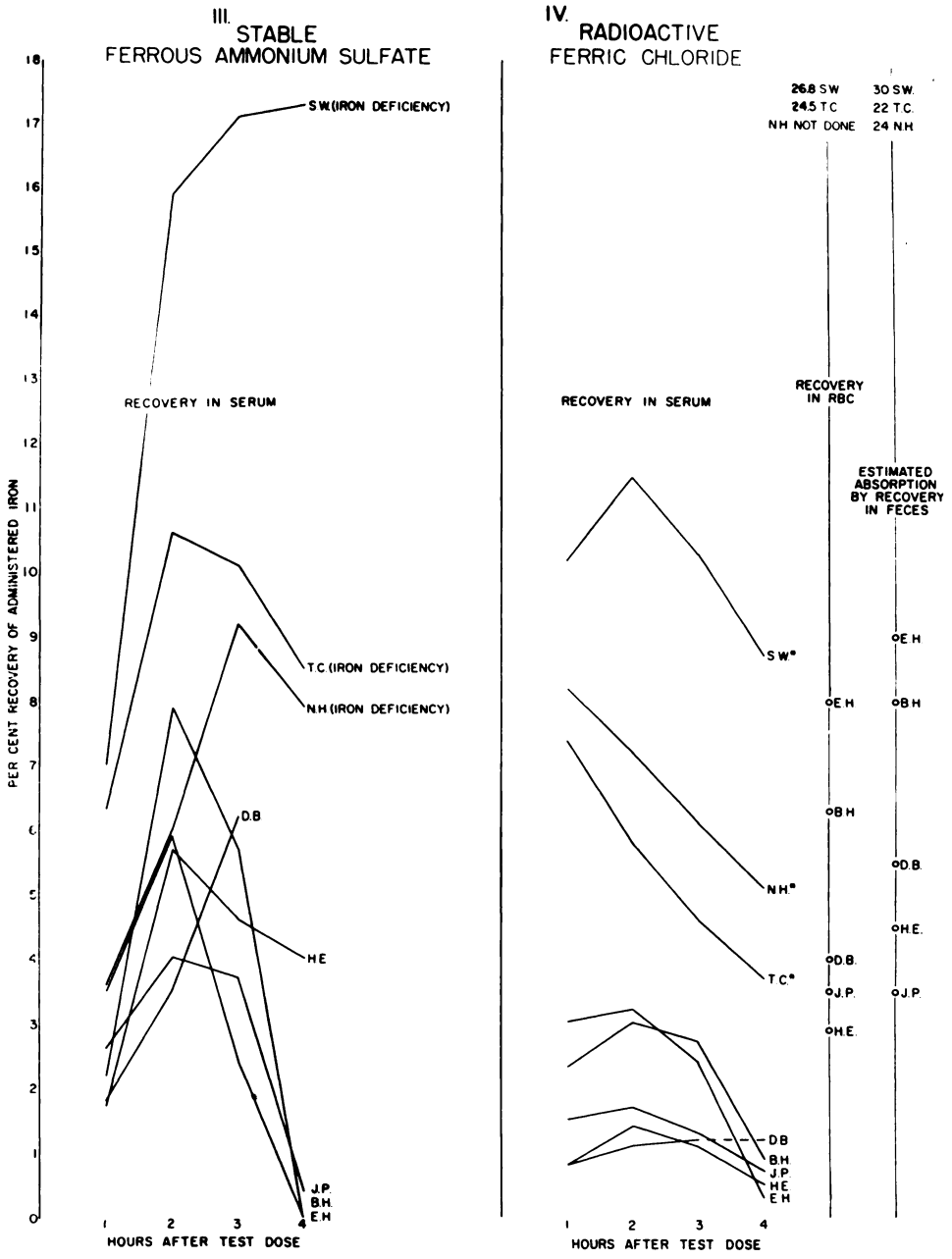

Fig. 2. Response of normal subjects to simultaneously administered stable ferrous iron and radioactive ferric iron.

although there was no definite clinical evidence of this. An interesting finding in the group of patients with coronary occlusion and the one patient with axillary vein thrombosis and pulmonary infarction (Table IId, e), originally selected as controls, was evidence of generally poor iron absorption, consistently in the serum recoveries and frequently in the hemoglobin recovery of Fe_{59} or by the stool analysis, although two patients (J. P. and A. M.) showed normal hemoglobin recovery.

DISCUSSION

In evaluating the type of absorption study employed in this series, several points become apparent. The heights and shapes of serum iron tolerance curves, both stable and isotopic, vary greatly from person to person regardless of the initial fasting serum iron level or the hematologic picture (Figs. 1 and 2), but the shape of the stable and radioactive curves on single individuals are of generally similar contour.

Throughout all the studies the recovery of serum iron, stable or radioactive, was considerably less than the recovery in hemoglobin or the estimation of absorption by stool analysis. This is consistent with previous work in the literature,[1,3,4,13] and the reason for the difference has already been commented upon. As others have pointed out,[12] the administration of the large doses of iron necessary to study changes of stable iron in the serum is unphysiologic, whereas the minute quantity of elemental iron necessary for evaluation of absorption with a radioactive tracer allows a more physiologic procedure. It is interesting to observe in the present study that in the serum the recoveries of stable iron were generally more similar to the tracer recoveries when both iron salts were administered in the same state of reduction (or were reduced simultaneously from the ferric state) than when a ferric tracer was given with a large quantity of stable ferrous iron. As a ferrous tracer is now available, there is no reason to employ the large amount of reduced stable iron as a carrier or to rely on other reducing agents, such as ascorbic acid, for maximal absorption.

Since the serum recoveries are 30 to 40 per cent less than the later hemoglobin recovery of ingested iron, there is little value in carrying out the serum procedure, except in an attempt to evaluate the rapidity of iron absorption shortly after its administration. Such a study might be valid for healthy subjects, but in disease states no real evidence of total iron absorption can be obtained from serum or plasma measurements, as the rate of iron removal to storage tissues may vary widely.[14]

Accordingly, for the study of iron absorption in patients with various disease states the radioactive tracer technique, allowing evaluation of eventual iron uptake in hemoglobin and its total absorption by stool analysis, provides a more accurate and physiologic method. The stool analysis technique has been shown to be more reliable in the study of disease, however, because certain conditions, such as fever or infection, may impair the synthesis of hemoglobin and render the estimation of iron absorption by hemoglobin analysis unsatisfactory.[15]

SUMMARY

An iron tolerance test, employing both stable and radioactive iron, has been evaluated in a series of healthy controls and in a series of patients with a variety of disease. The data for hourly serum recoveries of iron over a four-hour period, later recovery in hemoglobin of radioactive iron, and estimation of total iron absorption by stool analysis of radioactive iron are presented for both series. The effect of reduction of iron to the ferrous state on its absorption has again been demonstrated, and the relatively poor absorption of ferric iron has been substantiated. By the present tolerance test, increased absorption in iron-deficient patients has been demonstrated. The relative inaccuracy of serum values as an indication of iron absorption has been emphasized, and it is concluded that a radioactive ferrous iron tracer technique provides a more useful and reliable measurement of iron absorption than does the stable iron tolerance test.

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REFERENCES

1. HAHN, P. F., BALE, W. F., LAWRENCE, E. O., and WHIPPLE, G. H.: Radioactive iron and its metabolism in anemia. *J. Exper. Med.* 69: 739, 1939.
2. SAYLOR, L., and FINCH, C. A.: Determination of iron absorption using two isotopes of iron. *Am. J. Physiol.* 172: 372, 1953.
3. WHIPPLE, G. H., and ROBSCHEIT-ROBBINS, F. S.: I. Iron and its utilization in experimental anemia. *Am. J. Med. Sc.* 191: 11, 1936.
4. MOORE, C. V., ARROWSMITH, W. R., WELCH, J., and MINNICH, V.: Studies in iron transportation and metabolism: IV. Observations of the absorption of iron from the gastro-intestinal tract. *J. Clin. Investigation* 18: 553, 1939.
5. GRACE, W. J., DOIG, R. K., and WOLFF, H. G.: Absorption of iron from the gastrointestinal tract. J. CLIN. NUTRITION 2: 162, 1954.
6. RAMSEY, W. M.: The determination of iron in the blood plasma or serum. *Biochem. J.* 53: 277, 1953.
7. RATH, C. E., and FINCH, C. A.: Chemical, clinical and immunological studies on the products of human plasma fractionation. XXXVIII. Serum iron transport. Measurement of iron-binding capacity of serum in man. *J. Clin. Investigation* 28: 79, 1949.
8. GIBSON, J. G., 2ND, and EVELYN, K. A.: Clinical studies of the blood volume. IV. Adaptation of the method to the photoelectric microcolorimeter. *J. Clin. Investigation* 17: 153, 1938.
9. GREGERSON, M. I.: A practical method for the determination of blood volume with the dye T-1824. A survey of the present basis of the dye-method and its clinical application. *J. Lab. & Clin. Med.* 29: 1266, 1944.
10. BADENOCH, D. M., and CALLANDER, S.: Iron metabolism in steatorrhea. *J. Hemat.* 9: 123, 1954.
11. ALPER, T., SAVOG, P. V., and BATHWELL, T. H.: Radio iron studies in a case of hemochromatosis. *J. Lab. & Clin. Med.* 37: 665, 1951.
12. PETERSON, R. E., and ETTINGER, R. H.: Radioactive absorption in sideroses (hemochromatosis). *Am. J. Med.* 15: 518, 1953.
13. MOORE, C. V., MINNICH, V., and WELCH, J.: Studies in iron transportation and metabolism: III. The normal fluctuations of serum and easily split-off blood iron in individual subjects. *J. Clin. Investigation* 13: 543, 1939.
14. DUBACH, R., MOORE, C. V., and MINNICH, V.: Studies in iron transportation and metabolism: V. Utilization of intravenously injected radioactive iron for hemoglobin synthesis and an evaluation of radioactive iron for studying iron absorption. *J. Lab. & Clin. Med.* 31: 120, 1946.
15. DUBACH, R., CALLANDER, S., and MOORE, C. V.: Studies in iron transportation and metabolism: VI. Absorption of radioactive iron in patients with fever and with anemias of varied etiology. *Blood* 3: 526, 1948.