

60–190 ng%). The diagnosis of toxic diffuse goiter (Graves' disease) was made and the patient was started on propylthiouracil, 200 mg q.i.d. along with propranolol hydrochloride, 20 mg q.i.d. To our surprise, 6 wk later, the patient was still clinically toxic, and discussion with relatives revealed that she was not taking her thyroid medications regularly. They indicated that they could not control her behavior adequately to assure us that she would take her medications conscientiously. For that reason, we elected to treat this patient more definitively with I-131. A urine pregnancy test was ordered and the patient presented a water-like liquid for study. A second request for a urine specimen produced a yellow fluid more typical of urine. A radioiodine uptake from a 5- μ Ci I-131 capsule was <1% at 24 hr. However, the number of counts over the thyroid area was over 2.5 times our normal background reading. We repeated the uptake and even more closely observed the patient ingesting the radioiodine capsule. The uptakes at 1, 2, 4, and 24 hr, however, were all <1%. Again, all of these counts were significantly greater than background. There was no significant tracer uptake over the ovaries. The 24-hr urine iodine was 84 μ g (normal 150–700 μ g/24 hr).

A 24-hr urine showed that <1% of the radioiodine was present. This point confused us greatly. If the radioiodine was present to only a slight degree in the thyroid and not in the urine, where could it be? We then suspected that the low uptake results were factitious. To test this hypothesis we repeated the patient's uptake, administering liquid I-131. The uptake was then 45% at 1 hr and 64% at 24 hr. Twenty-nine mCi of liquid I-131 was then administered orally. The patient's goiter promptly regressed, as did her hyperthyroidism.

One of us (SSS) has previously served as physician at a federal narcotics hospital. Whenever possible, all medications in that institution were administered either in liquid form or by the parental route in an effort to avoid the problem of contraband medication. Patients given capsules or tablets would sometimes put the medication between the cheek and gum and later remove this medication for the "market place." We guessed that if the current patient had carried out a similar maneuver, enough absorption could occur to provide trace amounts of radioiodine over the thyroid area, and this could explain our previously confusing results. The prompt and intense radioiodine uptake observed after administration in liquid form provided support for our suspicion. In retrospect, we might have measured uptake levels over the stomach and mouth after apparent capsule ingestion. This could be a practical approach if liquid radioiodine is not available.

Although the patient repeatedly refused to admit that she did not swallow the entire capsule, this seemed to be the most reasonable explanation. The manufacturer of the I-131 capsule in question, assured us that no reports of failure of I-131 uptake have been reported because the material was given by capsule. Although we have administered many thousands of I-131 capsules in our laboratory, we have never seen a serious problem of capsule malabsorption, so such events, if any, must be exceedingly rare. We must conclude that this patient in all likelihood surreptitiously ejected the I-131 capsule at some time after putting it in her mouth.

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Accepted Gallium-67 Decay Data

The industrial and government members of the Atomic Industrial Forum Research Associate Program have agreed to the following decay data for gallium-67.

The AIF-sponsored Research Associate Program is supervised

TABLE 1. Ga-67 EC DECAY* (78.26 H 3)
I (MIN) = 0.10%*

Radiation type	Energy (keV)	Intensity (%)	$\Delta(q\text{-rad}/\mu\text{Ci}\cdot\text{hr})$
Auger-L	0.99	168	9
Auger-K	7.53	61	4
ce-K-1	81.607 5	0.224 9	0.0004
ce-K-2	83.652 5	28.7 13	0.0511
ce-L-2	92.117 5	3.52 15	0.0069
ce-M-2	93.175 6	0.516 22	0.0010
ce-K-3	174.918 10	0.40 5	0.0015
X-ray L	1	0.8 4	≈ 0
X-ray K α_2	8.61578 5	16.8 12	0.0031
X-ray K α_1	8.63886 5	32.9 23	0.0061
X-ray K β	9.57	6.7 5	0.0014
γ 1	91.266 5	3.07 10	0.0060
γ 2	93.311 5	38.3 12	0.0760
γ 3	184.577 10	20.9 6	0.0823
γ 4	208.951 10	2.37 7	0.0105
γ 5	300.219 10	16.8 4	0.107
γ 6	393.529 10	4.70 14	0.0394
γ 10	887.693 15	0.145 5	0.0027

3 weak γ 's omitted ($\sum I\gamma = 0.13\%$)

* National Council on Radiation Protection and Measurements, Handbook of Radioactivity Measurement Procedures, NCRP Report No. 58, November 1, 1978.

and administered by the National Bureau of Standards for the member industrial companies in an effort to unify the nuclear decay data and maintain equivalent millicurie values for the major radiopharmaceutical manufacturers.

Since January 1978, intercomparison studies have been completed on standards of gallium-67, based on the decay scheme issued by the Nuclear Data Group of the Oak Ridge National Laboratories as part of the Evaluated Nuclear Structure Data File (ENSDF) program. This nuclear decay data can be found in NCRP Report 58.

A new issuance of the gallium 67 standard will be available in July 1980, from the National Bureau of Standards, based on the decay data shown above.

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Kinetics of Thyroidal Uptake of Pertechnetate

In recent articles, Hays (1–3) has used a model with two intrathyroidal compartments to describe thyroidal uptake of pertechnetate in the normal and diseased gland. A two-compartment model was introduced because a single-compartment model (4) could not produce a computed uptake curve that adequately fitted experimental data. The two compartments consist of a fast ("follicular cell") compartment, which equilibrates instantaneously with plasma, and a slower ("colloid") compartment. A plasma tracer curve, based on venous blood samples, was used in the computational procedures. No consideration appears to have been

TABLE 1. "INSTANTANEOUS" UPTAKE BEFORE AND DURING TREATMENT OF THYROTOXICOSIS WITH ANTITHYROID DRUGS

Months on treatment	0	1	2	4	6
Patient	"Instantaneous" uptake \pm s.e. (% dose)				
M. C.	0.6 \pm 0.9	5.7 \pm 0.6	10.4 \pm 1.1	11.8 \pm 1.0	11.5 \pm 1.3
M. A.	-0.2 \pm 0.6	2.0 \pm 0.6	3.4 \pm 0.3	4.5 \pm 1.6	6.4 \pm 0.7
D. W.	9.8 \pm 1.8	9.7 \pm 0.6	16.8 \pm 1.1	20.5 \pm 1.2	23.9 \pm 1.3
A. McG.	2.9 \pm 0.7	13.1 \pm 0.9	11.8 \pm 1.2	14.2 \pm 0.8	9.0 \pm 0.5
D. K.	1.2 \pm 0.3	1.3 \pm 0.5	3.2 \pm 0.3	7.1 \pm 0.3	5.4 \pm 0.4
Mean \pm s.d.	2.9 \pm 4.0	6.4 \pm 5.0	9.1 \pm 5.8	11.6 \pm 6.3	11.2 \pm 7.5

given to the effect of arteriovenous differences in plasma tracer concentration (5,6). It appears also that, in trials with the single-compartment model, the computed uptake curve was constrained to pass through the point (0,0).

Similar difficulties in describing Tc-99m uptake kinetics in thyrotoxic subjects have been found by Gray (6) and Hilditch (7). In both cases estimated tracer levels in arterial plasma were used in the analysis, these being derived from observed venous levels, which are lower at early times after i.v. administration of the tracer (5,6). It was still not possible, however, to obtain satisfactory fits to the observed uptake data using a single-compartment model (4). The problem in fitting the experimental data arose because of very rapid uptake of the tracer within the first minute after its administration. Satisfactory fits could not be obtained with the boundary condition that the computed uptake curve should pass through the point (0,0).

The purpose of this letter is to outline a method (8) of overcoming these difficulties—one that has similarities to that adopted by Hays (1-3). It entails the use of a model with a single intrathyroidal compartment (4), but freedom is given to the value at time zero of the computed uptake curve. No attempt is made to fit observed uptake values at times less than 45 sec.

An arterial tracer curve, approximated from observations in venous plasma, is used in the analysis. The curve, consisting of three exponential components, is obtained by increasing the best-fit curve through the observed venous concentrations by factors of 2.13, 1.77, 1.32, 1.23, and 1.14 at minutes 1, 2, 6, 10, and 20, re-

spectively (8). Implicit in this method is the hypothesis that there is "instantaneous" uptake that cannot be explained by the approximated arterial tracer curve and by the parameter values determined by the analysis. There could well be high-clearance uptake into a fast cell compartment, as Hays' work suggests, or immediate uptake due to the passage of an initial bolus of tracer through the gland. Furthermore, there could be a significant early reduction in the amount of tracer available for thyroid uptake because of plasma binding (9-11). If this binding takes place rapidly, the clearance of tracer into the gland will diminish rapidly until plasma binding reaches saturation. This would have the effect of producing an initial uptake too large to be explained by the final unidirectional clearance determined by the analysis.

Figure 1 displays the better fit obtained when freedom is given to the initial value of the computed uptake curve. The uptake data were obtained from a thyrotoxic subject under treatment with the antithyroid drug, propylthiouracil. Thyroid uptake was measured using an uptake counter, with appropriate correction for extrathyroidal activity made by performing a separate study with gland uptake blocked by 300 mg i.v. sodium perchlorate. A dose of 250 μ Ci [99mTc]pertechnetate in 2.5 ml isotonic saline was administered rapidly into the antecubital vein. The "instantaneous" uptake in this study was 16.8% of dose. Table 1 shows further values of "instantaneous" uptake in studies during the first 6 mo of treatment with antithyroid drugs in this subject and four other thyrotoxic subjects. All subjects except D. W. (who received propylthiouracil alone) received carbimazole and triiodothyronine. As can be seen, "instantaneous" uptake ranged from 0-23.9% of dose, with a significant increase with time on antithyroid drugs ($P < 0.02$ by the paired-t test). Except in subject D. W. (where there was an increase in unidirectional clearance), all other parameters of the compartmental analysis remained virtually unchanged.

While the symptoms of thyrotoxicosis—for example increased cardiac output (12)—are reduced by the action of antithyroid drugs, the present evidence suggests that, if triiodothyronine is also given, blood flow to the gland remains unchanged over the first few months of treatment. The observed increase in "instantaneous" uptake with time, during the initial stages of treatment, is consistent with the idea that this parameter may reflect the fraction of cardiac output passing through the thyroid gland. As treatment takes effect and cardiac output is reduced, a larger portion of the injected bolus of tracer is presented to the gland and "instantaneous" uptake increases. A more complete study of "instantaneous" uptake, including comparative studies of iodide and pertechnetate, is being published elsewhere (8).

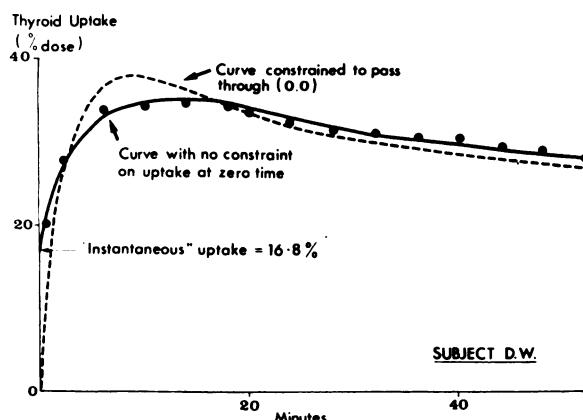


FIG. 1. Improved fit to thyrotoxic subject on propylthiouracil given i.v. dose of [99mTc]pertechnetate. Analysis based on single-compartment model of thyroid.

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Gallium-67 Accumulation in the Stomach in Patients with Postoperative Gastritis

Reflux of bile and duodenal contents into the stomach is common in patients after Billroth II gastroenterostomy, and is occa-

sionally seen in other gastric diseases. It is frequently difficult to distinguish between gastritis due to bile reflux and other causes of abdominal pain. Clinical findings and endoscopy are often ambiguous; endoscopic observation of bile reflex is not necessarily proof of gastritis. We have recently observed gastric uptake of gallium-67 in two patients with histologically confirmed bile-reflux gastritis.

Case 1. Following a vagotomy, antrectomy, and Billroth II gastroenterostomy for peptic ulcer a year before this admission, a 49-year-old man had symptoms of gastric dumping, watery stools, and nonspecific upper abdominal pain. A cholecystectomy was performed 2 mo before admission for chronic cholecystitis and cholelithiasis, but following this surgery he continued to have abdominal pain and fever. A gallium-67 study showed intense activity in the gallbladder region, a focus of activity to the left of the liver, later identified as the stomach, and weak uptake in the region of the splenic flexure. He was treated with appropriate antibiotics with gradual resolution of fever, and then was admitted to our hospital for re-evaluation of his abdominal pain. Repeat gallium study (Fig. 1) showed no activity in the gallbladder region and diminished activity near the splenic flexure. These changes were attributed to resolving infection. The undiminished, marked accumulation of Ga-67 in the stomach appeared unrelated to the resolving febrile process. Bile gastritis was then considered as a cause of the persistent abdominal pain. Gastroscopy showed severe diffuse erythema and friability of the gastric mucosa, and biopsy revealed focal acute and chronic gastritis.

Case 2. A 60-year-old man, who had had a vagotomy, antrectomy, and Billroth II gastroenterostomy 16 yr before this admission, subsequently experienced intermittent episodes of epigastric pain, bilious vomiting, and occasional hematemesis. Upper and lower gastrointestinal radiocontrast studies were normal. Radioisotope studies showed evidence of both bile reflux and gastric accumulation of gallium (Fig. 2). Gastroscopy with biopsy of the stomach at several sites showed visible and histologic changes interpreted as foci of moderately severe gastritis.

A probable cause of gallium accumulation in these patients is inflammatory disease in the gastric wall, similar to the gallium accumulation observed in other inflammatory diseases (1-4). Another possibility is a change in gastric mucosal function or permeability resulting in abnormal excretion of the gallium into the gastric lumen. Excretion into the gastric lumen has been reported as an unexplained finding in a single patient at 18 hr after

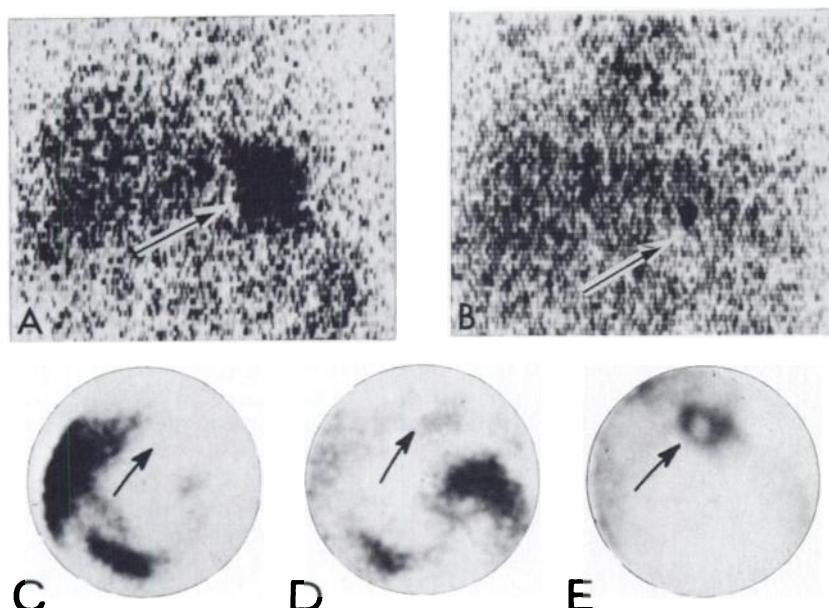


FIG. 1. Scintigrams of stomach region in Patient 1. Anterior view (A) shows abnormal left upper quadrant accumulation (arrows) in stomach 3 days after i.v. injection of 3 mCi Ga-67 citrate. Posterior view (B), imaged at same time, shows same accumulation faintly. Abnormal radioactivity remained after patient was given a drink of water. Slight persisting radioactivity is also noted in region of splenic flexure. Anterior rose bengal ($I-131$) scintigram (C) was imaged just before a meal, 40 min after tracer injection (150 μCi). Another image of same region (D), 5 min after meal, shows reflux of hepatobiliary marker into stomach. Anterior stomach image (E) was obtained 20 min after injection of 2 mCi of $Tc-99m$ as pertechnetate; it confirms stomach location.