

**RADIOCHEMISTRY
AND RADIOPHARMACEUTICALS**

**Indium-111-Labeled Autologous Platelets for
Location of Vascular Thrombi in Humans**

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Twenty-two patients suspected of having either venous or arterial thrombi were studied with In-111-labeled autologous platelets. Whole-body scans were performed 3, 24, and 48 hr following i.v. injection. Twelve patients studied with saline-washed platelets had unsatisfactory 15-min recovery and biologic half-time. When the labeling was carried out in plasma, these values compared favorably with normal values reported for Cr-51-labeled autologous platelets. Of ten patients studied using platelets labeled in plasma, three had normal scans, six had abnormal scans, and one had an equivocal scan. All six abnormal scans were confirmed with corresponding positive findings in either the venogram, arteriogram, or lung scan.

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Chromium-51 as the chromate has provided a useful label for the study of platelet kinetics and sequestration in humans (1-3). Several important limitations have prevented this technique from achieving widespread clinical use. The disintegration of Cr-51 results in only 9%, 320-keV gamma photons, which are neither sufficiently abundant nor of optimum energy for imaging with the gamma camera. Chromium-51 is usually not carrier free and the stable chromium ions, if present in high enough concentration, may have a toxic effect (4) and also may affect the distribution of label between old and young platelets (5). Furthermore the low labeling efficiency severely limits the total amount that can be bound to human platelets. These factors have limited the in vivo study of human platelets to the measurement of lifespan, turnover rates, and gross organ countings—studies that are possible with low-activity blood samples and flat field scintillation probes.

Indium-111 has several advantages as a label for tracing biologic processes that must be followed by scanning over a period of 1-8 days (6). The physical decay of In-111 provides two useful gamma pho-

tons at 171.2 and 245.3 keV (7), in high abundance (183%), these, together with the 2.8-day half-life, furnish a high photon/rad ratio for studies carried out over a 1-wk period (8).

Recently a lipophilic chelate of indium, In-111 with oxine (9), has been described as an efficient agent for the labeling of platelets (10), as well as of polymorphs (12-15) and lymphocytes (16). Studies in dogs (10,11) and rabbits (17) have shown this method to give recoveries and survival times similar to Cr-51-labeled platelets with the added advantage of gamma-camera imaging. The present study was undertaken to evaluate the usefulness of autologous platelets labeled with In-111 oxine for locating vascular thrombi in humans (18).

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METHOD

Twenty-two patients suspected of having either arterial or venous clots were studied. A modification (17) of the method of Thakur et al. (10) was used. The main steps included purification of the $^{111}\text{InCl}_3$, formation and extraction of the oxine chelate, and separation and labeling of the platelets. The purification was carried out on an anion-exchange column. Approximately 3 mCi of $^{111}\text{InCl}_3$ in 1.5 ml were further acidified to approximately 2.0 M HCl and applied to a 1×28 cm 200–400 mesh anion exchange resin column equilibrated with 2.0 M HCl. This was washed with five bed volumes of 2.0 M HCl and the activity was then eluted with 0.2 M HCl and evaporated to dryness. This procedure removes traces of iron, cadmium, zinc, lead, copper, and chelates shown to be present by atomic absorption analysis and thin layer chromatography, with no loss of In-111 activity. We have found that removal of these contaminants increase by 30% the amount of In-111 chelated by 100 μg of oxine. This procedure also removes potentially toxic metal ions. The $^{111}\text{InCl}_3$ was brought into solution with 1 ml of 0.3 M acetate buffer, pH 5.5, and 50–150 μg^* (50–150 μl) of oxine (8-hydroxy quinoline) in absolute ethanol were added. This solution was mixed well on a vortex mixer and incubated 15 min at room temperature. The resultant chelate was extracted in one volume in each of two extractions of either methylene chloride or chloroform and evaporated to dryness with a gentle stream of nitrogen. Purification of the $^{111}\text{InCl}_3$ has eliminated precipitates previously seen with both of these solvents. The complex was then redissolved in 50 μl of absolute ethanol, followed by the dropwise addition, with mixing, of 150 μl of 0.9% NaCl to reduce the ethanol concentration to 25%. Approximately 85% of the original activity was recovered as In-111 oxine in the faintly yellow-colored labeling solution.

The platelet separation and labeling were carried out as follows. Two 50-ml syringes each containing 7.5 ml of NIH ACD solution A† were used to obtain 85 ml of venous blood. After gentle mixing by inverting the syringes three or four times, the citrated blood was transferred to two 50-ml sterile siliconized polypropylene centrifuge tubes. All subsequent procedures were carried out in siliconized‡ plastic at room temperature ($22^\circ\text{C} \pm 2^\circ$). The tubes were capped and centrifuged at 220 g for 15 min. The platelet-rich plasma (PRP) was removed and respun, if necessary, at 150 g for 5 min to remove contaminating red blood cells (RBCs) and centrifuged at 1000 g for 5–15 min (inspected at 5-min intervals for a platelet “button”) and all but 2 ml of the platelet-poor plasma (PPP) were removed and

saved. The platelet buttons were resuspended by gentle repeated suction in the 2-ml plasma layer and acidified with 200 μl of ACD solution to pH 6.5–6.7. The In-111-oxine complex was added dropwise to the platelet suspensions with gentle agitation, and mixtures incubated at room temperature for 30 min. The incubation mixtures were pooled and diluted with a total of 5–10 ml of PPP, gently mixed, and centrifuged at 1000 g for 5–15 min. The radioactive PPP was removed, and the platelet button gently layered with 2 ml of PPP to remove any remaining unbound radioactivity. The labeled platelets were gently resuspended in 5–10 ml of PPP and inspected microscopically in a hemocytometer for aggregates and platelet purity. If necessary, any contaminating RBCs were removed with a final spin at 150 g for 5 min. The radioactivity in the labeled platelets, and the total radioactive PPP were measured in a dose calibrator. A standard was prepared by dilution of 100 μl of the dose in 100 ml of 0.2 M HCl, and mixing well. Note: for the first 12 patients the platelet labeling was carried out in ~ 7.0 ml 0.9% saline and not in plasma (10). The time from obtaining the blood until the reinjection of labeled platelets was 2–3 hr.

From 3×10^8 to 3×10^9 (av. 8.5×10^8) autologous platelets were labeled with 200–500 μCi (av. 300 μCi) In-111. These platelets were injected in 5–10 ml plasma through a peripheral vein, with

TABLE 1. FIFTEEN-MINUTE RECOVERY AND HALF-TIME WITH TWO LABELING METHODS

Patient	Method A		Platelet biologic $t_{1/2}$ days
	% dose in circulating platelets @ 15 min	% dose in plasma @ 15 min	
FL	1.5	18.1	
OP	0.7	6.1	
HD	0.0	11.3	
JB	14.3	2.4	Uninterpret- able
CT	2.7	6.5	
CN	12.6	34.8	
HJ	7.3	15.3	
HA	4.0	8.5	
$\bar{X} \pm \text{SD}$	5.4 ± 5.5	12.9 ± 10.2	
	Method B		
DN	43.9	14.3	4.6 (.99)
EB	37.6	13.8	4.4 (.99)
JT	67.9	4.0	4.2 (.99)
KS	59.2	6.9	4.6 (.99)
$\bar{X} \pm \text{SD}$	52.2 ± 13.9	9.8 ± 5.1	4.5 ± 0.2
Significance or difference between two means A&B	P < 0.001 significant	P < 0.4 > 0.2 not significant	

* Correlation coefficient.

care taken not to withdraw blood into the syringe. The volume injected was determined by weighing the syringe. A 15-min postinjection blood sample was taken from the opposite arm to determine the fraction of labeled platelets remaining in the circulation ("recovery"). The activity remaining in the syringe was measured in the dose calibrator. The calculation of "recovery," and of cell and plasma activity, was done using the formulae listed in the addendum. Blood samples were obtained daily for 10 days for platelet lifespan determination; the points were plotted on a linear scale and the disappearance calculated using the method of least squares. Whole-body scans were obtained at 3, 24, and 48 hr using a scintillation camera with a moving bed. Spot views were taken where clinically indicated, and of any abnormal areas on the whole-body scan. The results of the scans were compared with arteriography or venography where available. Twelve patients were studied with saline-washed platelets (Method

A) and ten were studied with platelets labeled in plasma (Method B).

RESULTS

The recovery of labeled platelets circulating at 15 min, and the 15-min plasma activity, is shown in Table 1. Recoveries obtained with the saline method were low ($5.4\% \pm 5.5\%$), and correlated with high liver uptake and no visible blood-pool activity on the corresponding whole-body scan. The recovery of platelets labeled in plasma averaged $52.2\% \pm 13.9\%$, significantly higher ($p < 0.001$) than the saline method. The labeling efficiency however, was significantly higher when the platelets were labeled in saline (average, $64\% \pm 13\%$) than in plasma (average, $34\% \pm 15\%$). This is due to the high affinity of plasma transferrin for In-111 activity. Transferrin labeling is minimized by keeping the plasma volume in the labeling mixture no more than 2 ml and by acidifying with ACD solution dur-

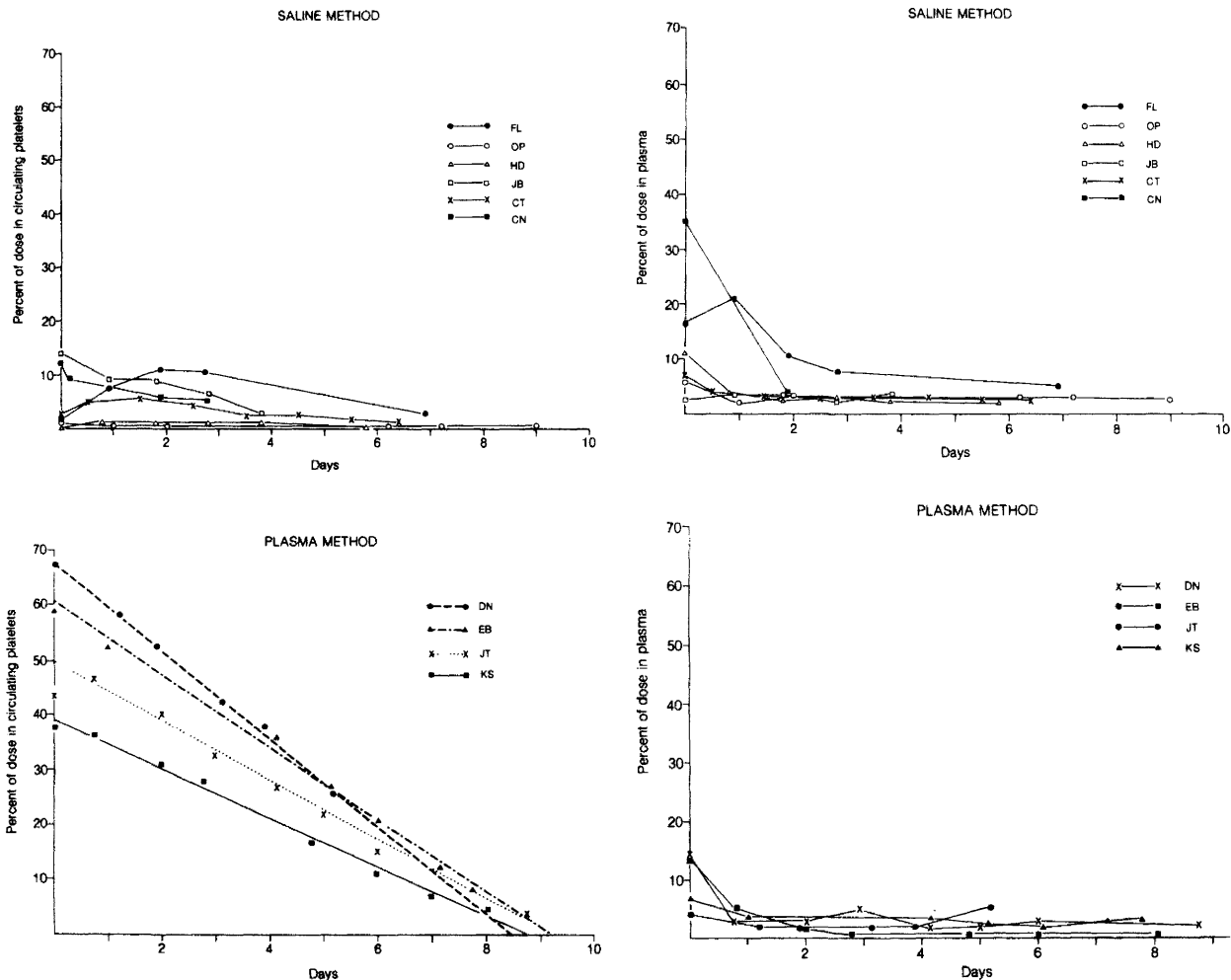


FIG. 1. In-111 platelet blood disappearance curves and plasma levels: comparison of the two methods (see text). Top two curves, saline method bottom two curves, plasma method; left, % injected dose in circulating platelets; and right, % injected dose in plasma.

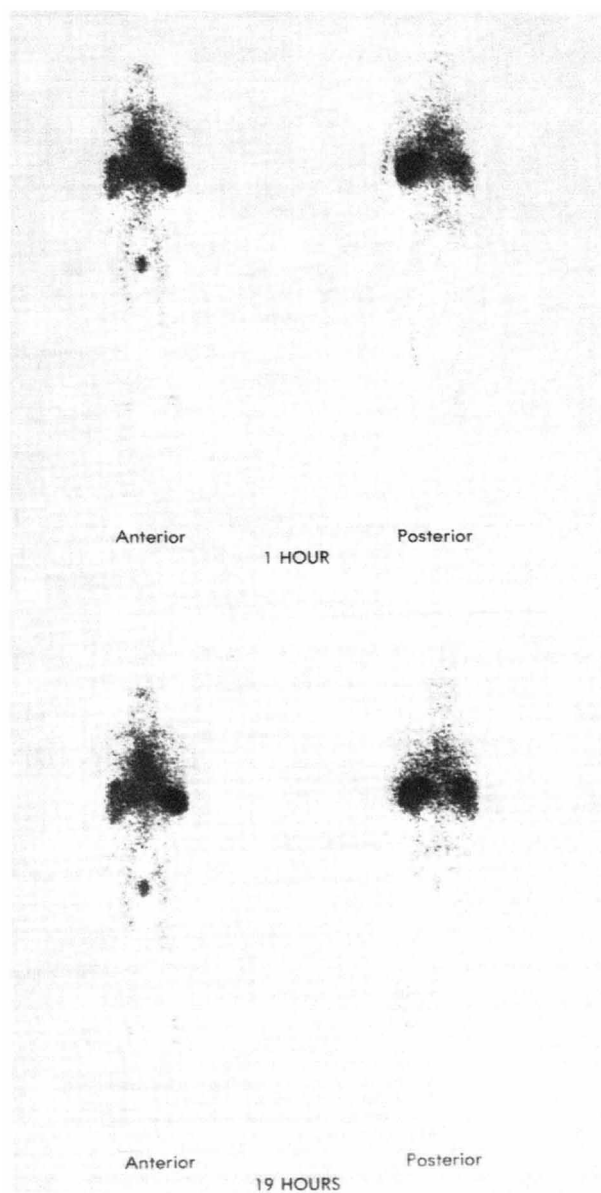


FIG. 2. Normal In-111 platelet whole-body scans at 1 and 19 hr after injection of 2×10^9 platelets tagged (in plasma) with 300 μ Ci In-111. Note: spleen activity predominant, with well-defined blood-pool activity (liver approximately equal to blood) and genital activity.

ing the labeling procedure. That the low recovery with Method A was not due to free In-111 is shown by the fact that the 15-min plasma levels were also low, and not significantly different between the two methods (Table 1). The blood curves showed rapid removal of saline-labeled platelets from the circulation, and it was not possible to obtain a reproducible half-time value (Fig. 1). In contrast, a smooth, linear disappearance of platelets labeled in plasma was found when blood-platelet activity was plotted linearly as a function of time (Fig. 1). The average half-time (Method B) was 4.5 ± 0.2 days (aver-

age lifespan 9 days), with a correlation coefficient, r , of 0.99 for each curve. Figure 2 shows normal whole-body scan at 1 and 19 hr after injection of 2×10^9 autologous platelets tagged with 300 μ Ci In-111 in plasma. The cardiac blood pool, major vessels, and genitalia are easily seen; there is much less liver activity (approximately equal to blood) than splenic activity; and no bone marrow is visible.

In the first group of 12 patients studied with platelets prepared by the saline method, only one showed an abnormal accumulation of activity in the vascular tree, and this was at the site of previous catheterization of the femoral arteries (Fig. 3). This patient also had the highest level of circulating platelets in his group—note the easily visible cardiac and large-vessel blood pool seen on the whole-body scan. The other patients in this group had no visible blood-pool activity, and all of the platelets were concen-

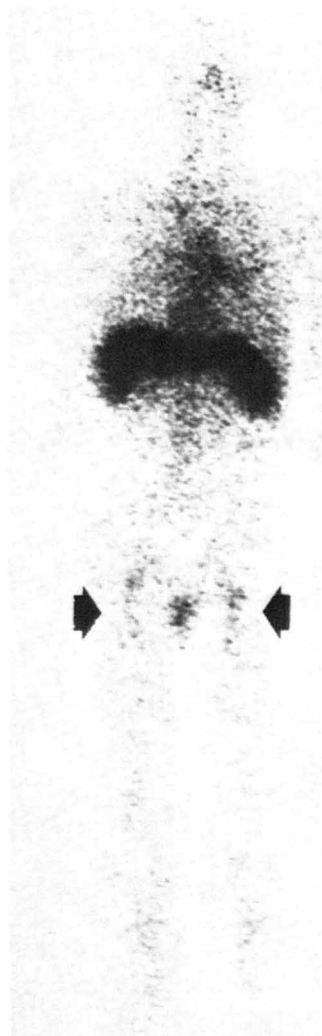


FIG. 3. (Patient 7, Table 2). Anterior whole-body scan of patient with left carotid atherosclerosis and occlusion. Note uptake of platelets bilaterally at the site of recent femoral-artery catheterizations (arrows). No uptake over carotids.

TABLE 2. CORRELATION OF SCAN WITH CLINICAL AND LABORATORY FINDINGS

Patient No.	Age	Sex	Clinical diagnosis	Laboratory findings	¹¹¹ In-platelet scan findings
7	67	M	Carotid atherosclerosis and TIA's	Br. scan L CVA arteriogram L carotid occlusion.	Bilateral accumulation of platelets in femoral vessels. No accumulation over carotids (see Fig. 3).
13	27	M	Back pain, lumbar disc L4-5. Otherwise normal	Recent back surgery with rod fixation	Normal activity in spleen, blood pool, and penis. Region of interest spleen cf: phantom; 45% platelets in spleen, 47% circulating at 15 hr.
14	54	M	Alcoholism, anxiety. Possible carotid stenosis	X-Ray: carotid calcifications	Normal spleen, blood pool and genital activity (see Fig. 2).
15	55	M	Amaurosis fugax R eye R carotid endarterectomy	Angio: R. carotid obstruction	Focal platelet accumulation R carotid maximum @ 18 hr. Also accum. of platelets R cephalic anticubital veins.
16	51	M	Swollen erythematous R leg: R/O thrombophlebitis	L.V. angio: cardiomyopathy pulm. angio: normal	Normal.
17	45	M	Coumadin toxicity, hematoma R sciatic nerve w/paralysis and foot drop	Venogram: R DVT calf veins	Accumulation of platelets R calf 22 & 48 hr (see Fig. 4). Repeat scan 1 mo (after anticoag.): normal.
19	70	M	Adeno Ca lung, R lobectomy on heparin	Venogram R VDT calf veins. Lung scan: pulm. emb.	Progressive accumulation platelets R calf; also R femoral and iliac veins (see Fig. 5).
20	42	M	Paraplegia, R chest pain. On coumadin	Lung scan: P.E. post apical seg. R.U.L.	Focal accumulation of platelets at the apex of lung perfusion defect in R.U.L.
21	52	F	Pain and swelling L leg: thrombophlebitis on coumadin	Venogram: DVT L calf, no evidence of thrombosis above popliteal vein.	Focal platelet accumulation R calf; also prominent focus of activity R femoral vessels @ 12 and 36 hr.
22	65	M	R chest pain R/O pulm. emboli. On heparin	Lung scan: P.E. lat. seg. R.L.L.	Equivocal abnormal activity @ 12 hr R lower thorax. Note 24 and 48 hr views not obtained due to critical illness.

trated in the liver, spleen, and bone marrow. The other nine patients summarized in Table 2 were all studied with platelets prepared by the plasma method. For the patients listed in Table 2 three of the scans were normal, six abnormal, and one equivocal. One patient (No. 18, not listed) had a kinetic study only, with no scan. This patient, a 59-year-old male, had chronic lymphatic leukemia in remission, but with a low platelet count of 50,000. The measured blood disappearance was abnormally fast: $t_{1/2} = 2.4$ days.

Arterial accumulation of platelets was seen in two patients (Nos. 7 and 15), one in the recently catheterized femoral arteries, and one in a recently endarterectomized carotid artery 1 wk after surgery—clinically, both fairly acute lesions.

Three patients with deep-vein thrombosis in the legs, documented by venography, had accumulation of platelets in the corresponding areas of the scan (Fig. 4). In addition, two of these patients had obvious areas of accumulation proximally in the iliac veins (Fig. 5). Four patients studied were already on anticoagulant therapy at the time of injection of

platelets (Table 2). Patient No. 20—a paraplegic with recent pulmonary embolism documented by lung perfusion scan—had a focal accumulation of platelets at the apex of the perfusion defect. The equivocal scan (Patient 22) was a suboptimal study, since the 24- and 48-hr views were not obtained, owing to the patient's critical condition.

DISCUSSION

This study demonstrates that the In-111-oxine method provides a reliable means for obtaining viable In-111 labeled human platelets. These platelets had a well-defined linear disappearance from the blood (Fig. 1), and the similarity of these disappearance curves and half-times to those published for chromium-labeled human platelets (2,3,19) is evidence that In-111-oxine provides at least as good a label for human platelets as does Cr-51.

Our findings confirm in humans the observation of Scheffel et al. in rabbits (17), that it is necessary for platelet viability to retain some of the plasma during the incubation of In-111-oxine with the platelets. The damaging effect of saline washes is not

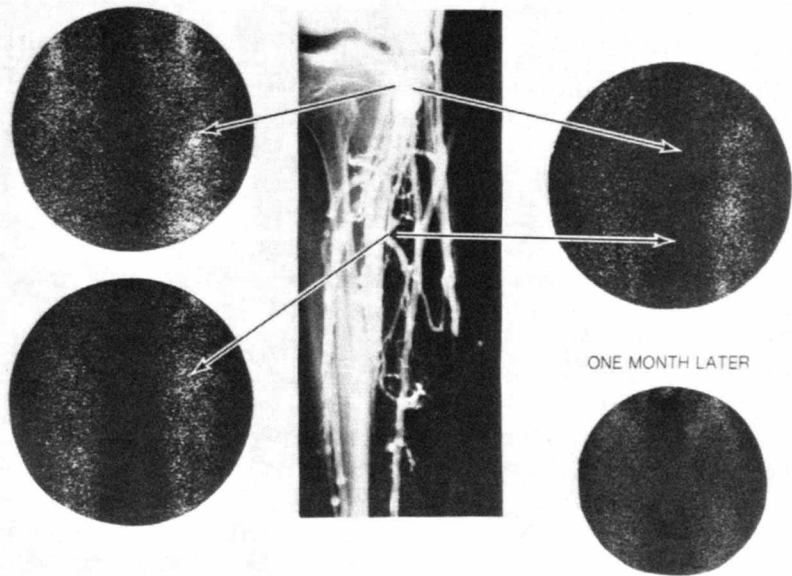


FIG. 4. (Patient 17, Table 2). Spot views over right calf and popliteal area show accumulation of platelets at 22 hr (upper & lower left) and 48 hr (upper right) corresponding to site of venous clot by venogram. Repeat study following clinical recovery, 1 mo later (lower right) shows no abnormality.

nearly as pronounced with dog platelets (10). In other experiments, using saline-washed dog platelets labeled with In-111-oxine we have noted that the blood disappearance is curvilinear, suggesting that at least some damage was incurred during labeling. The linearity of the human blood-platelet disappearance is consistent with the hypothesis that normally the predominant mechanism for removal of platelets from the blood involves aging and senescence. It also suggests that elution of the In-111 label from the platelets is not a major factor, and this is supported by the lack of significant plasma activity at any time. The appearance of large amounts of liver activity 1 hr after administration of platelets labeled in saline suggests that the damaged platelets are re-

moved intact and very rapidly by the reticuloendothelial system of this organ, at a rate similar to that of labeled colloidal material. Because little free In-111 activity was released into the circulation, the significant bone-marrow activity seen on the scans performed with saline-labeled platelets was probably due to sequestration of damaged platelets. Unlike In-111-labeled leukocytes, normal platelets labeled with In-111-oxine in plasma do not concentrate in the bone marrow. If care is taken to remove contaminating red blood cells, the platelet fraction represents a single cell population and may be used for kinetic studies. The physical half-life and decay characteristics of In-111 are well matched to the biologic half-time of the human platelet, and provide

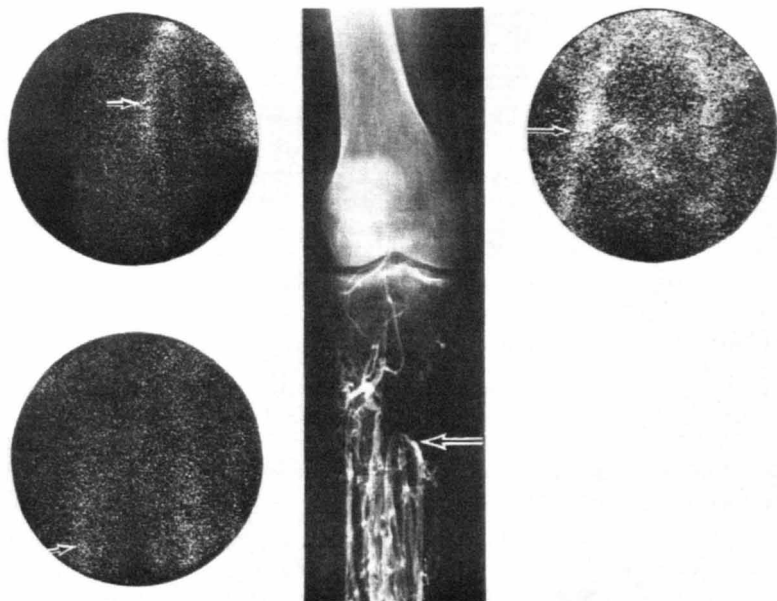


FIG. 5. (Patient 19, Table 2). Spot scintiphoto shows accumulation of In-111-labeled platelets over right calf (lower left), corresponding to D.V.T. on venogram. Also accumulation of platelets over right iliac and femoral vessels at 24 hr (upper left) and 48 hr (right).

a maximum number of useful photons with minimum radiation exposure.

No excretion of In-111 activity was seen in the urine or feces. This aided the visualization of venous clots in the femoral and iliac vessels in two patients. These areas are difficult or impossible to examine with the I-125 fibrinogen uptake test (20-23), plethysmography (24), Doppler ultrasound (25), or venography (26). In comparing the pattern of platelet activity with the venogram in Patients 17, 19, and 21, we noted that accumulation of platelets occurred to the greatest degree at the proximal end of the clot. In two of these patients, even more proximal pelvis clots were detected where the veins were not visualized on the venogram. This finding is in accord with the idea that these venous clots are actively propagating centrally. It is also in agreement with the hypothesis that in chronic thrombophlebitis, platelets will not accumulate on the older inactive clot. Further observations are under way in humans to determine more clearly the relationship between the age of the clot and uptake of platelets in thrombophlebitis (27). The optimum time for visualizing venous clots was 24 hr after injection, although some uptake was noted in all cases at 4-6 hr.

Indium-111-labeled platelets were seen to accumulate in areas of recent arterial trauma in two patients (Nos. 7 and 15). We have not had an opportunity to study acute arterial thrombosis such as stroke, myocardial infarction or acute obstructive peripheral-artery disease, but the results in our two patients suggest that there is a good possibility of obtaining visualization of these arterial processes with In-111-labeled platelets. A chronic lesion in the carotid artery in Patient 7 did not accumulate platelets.

The positive visualization of a recent pulmonary embolus (Patient 20) suggests that the method will have an important place in the study of this disease. If further observations show that this is a consistent finding, the test could prove to be highly specific (28).

The clinical importance of clot location is reflected by the large number of radiopharmaceuticals that have been investigated in an effort to find an optimum scanning agent. These include I-123-labeled fibrinogen (29-31), I-123-labeled plasminogen (32), In-111-labeled fibrinogen (33), highly iodinated fibrinogen (which is removed more rapidly from the circulation (34), Tc-99m oxine-labeled autologous platelets (35), and Tc-99m macroaggregated albumin (36,37). Indium-111-labeled platelets have biologic and physical characteristics that compare very favorably with all these agents, and larger

TABLE 3. DOSIMETRY

Critical organs	% distribution*	Dose: Rads from 500 μ Ci
Blood	43	0.506
Whole body	43	0.174
Spleen	45	12.3
Liver	5	0.318

* Organ distribution measured in patient #13 (Table 2) using a phantom standard.

clinical trials are planned to better define their relative usefulness.

The mechanism of labeling and the intracellular location of In-111 have been studied in human neutrophils (39). In-111-oxine complex diffuses rapidly across the cell membrane and then dissociates. Some of the oxine leaves the cell, and the In-111 binds to intracellular ligands. After short periods of incubation, the label is distributed to four soluble components, but with longer incubation more radioactivity becomes associated with particulate material. The In-protein complexes within the cell are isolated from the plasma transferrin by the cell wall, so that binding of In-111 by transferrin does not occur.

The patient's radiation dose from 500 μ Ci In-111 platelets was calculated using $t_{1/2}$ physical = 2.81 days, $t_{1/2}$ biologic = 4.5 days and $t_{1/2}$ effective = 1.73 days. As can be seen from Table 3, the limiting factor is the radiation dose to the spleen.

We have used this technique to study catheter thrombogenicity in dogs and are extending our observations to humans undergoing cardiac catheterization. In these studies the platelets are labeled before the procedure, and platelet accumulation is monitored at intervals over the catheter, the lungs, and distal arterial tree.

CONCLUSION

The In-111-oxine method provides a simple, reliable way to label human platelets with a stable radioactive marker, without altering their viability.

These In-111 labeled platelets may be used to study platelet kinetics and platelet distribution by whole-body scanning techniques.

We have visualized venous and arterial accumulation of platelets in thrombophlebitis, arterial trauma, and recent pulmonary embolism.

This method provides a rational approach to the study of the role of the platelet in hemostasis and thrombosis in vivo, as well as the effect of the various antiplatelet drugs on these processes.

ADDENDUM

1. Net cpm from platelets, per ml w.b. = net cpm/ml_{w.b.} — net cpm/ml_{pl} (1 — H × 0.97* × 0.91†).
2. % activity in platelets =
$$\frac{\text{Net cpm/ml w.b. in platelets}}{\text{Net cpm/ml in w.b.}} \times 100.$$
3. % dose in circulating platelets =
$$\frac{\text{Net cpm/ml w.b. in platelets} \times \text{TBV}^\ddagger (\text{ml}) \times 100}{\text{Net cpm injected}}$$

- * 0.97 = correction factor for plasma trapping in micro-hematocrit tube
 † 0.91 = correction factor for ratio of total-body Hct to peripheral Hct.
 ‡ TBV = total blood volume, from Tulane tables of ht. and wt.
 w.b. = whole blood
 Hct. = hematocrit
 pl = plasma

FOOTNOTES

- * The smaller amounts may be used with column purified indium.
 † National Institutes of Health acid citrate dextrose Solution A.
 ‡ "Siliclad;" Clay Adams, Div. of Becton Dickinson & Co., Parsippany, N.J.

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3rd ANNUAL WESTERN REGIONAL MEETING THE SOCIETY OF NUCLEAR MEDICINE

October 13-15, 1978

Vancouver Hotel

Vancouver, B.C., Canada

ANNOUNCEMENT AND CALL FOR ABSTRACTS FOR SCIENTIFIC PROGRAM

The Scientific Program Committee welcomes the submission of abstracts of original contributions in nuclear medicine from members and nonmembers of the Society of Nuclear Medicine for the 3rd Annual Western Regional Meeting. Physicians, scientists, and technologists—members and nonmembers—are invited to participate. The program will be structured to permit the presentation of papers from all areas of interest in the specialty of nuclear medicine. Abstracts submitted by technologists are encouraged and will be presented at the scientific program. Abstracts for the scientific program will be printed in the program booklet and will be available to all registrants at the meeting.

Guidelines for Submitting Abstracts

The abstracts will be printed from camera-ready copy provided by the authors. Therefore, only abstracts prepared on the official abstracts form will be considered. These abstract forms will be available from the Western Regional Chapters SNM office (listed below). Abstract forms will only be sent to the Pacific Northwest, Southern California, Northern California, and Hawaii Chapters in a regular mailing. All other requests will be sent on an individual basis.

All participants will be required to register and pay the appropriate fee.

Please send the original abstract form, supporting data, and six copies to:

Jean Lynch, Administrative Coordinator
3rd Western Regional Meeting
P.O. Box 40279
San Francisco, CA 94140

Deadline for abstract submission: Postmark midnight, July 7, 1978.

THE 3RD ANNUAL WESTERN REGIONAL MEETING WILL HAVE COMMERCIAL EXHIBITS AND ALL INTERESTED COMPANIES ARE INVITED. Please contact the Western Regional SNM office (address above). Phone: (415) 647-1668 or 647-0722.