

# Study of Factors that May Condition Scintigraphic Detection of Venous Thrombi and Pulmonary Emboli with Indium-111-Labeled Platelets

Kenneth M. Moser, Roger G. Spragg, Frank Bender, Ronald Konopka, Michael T. Hartman, and Peter Fedullo

*University of California, San Diego School of Medicine, San Diego, California*

**Incorporation of indium-111-labeled platelets (In-111-P) into venous thrombi and pulmonary emboli may permit rapid detection of these thromboemboli by gamma imaging. In a series of dogs in which femoral-vein thromboses and/or pulmonary embolism were induced experimentally by stasis and small amounts of thrombin, we addressed several questions pertinent to the sensitivity, specificity, and potential applicability of this approach. We found that when In-111-P were injected intravenously before thrombus induction or embolus release, femoral-thrombus images were consistently detectable within 15 min, whereas control femoral-vein images were unremarkable. Pulmonary emboli were also promptly imaged, and such In-111-P images agreed well with defects on Tc-99m MAA perfusion scans. When thrombi were aged in vivo for up to 10 hr after formation, they could still be imaged within 20–90 min after In-111-P injection. Administration of heparin, as an initial bolus followed by constant infusion, blocked platelet deposition on femoral-vein thrombi as assessed by both thrombus-to-blood ratios and failure to image. Injection of protamine at 6 hr, however, resulted in prompt thrombus imaging.**

**These data indicate that this approach may well have applicability to the detection of thromboemboli in humans, since imaging remains possible in canine thrombi aged in vivo for 10 hr so long as heparin therapy has not been instituted. The dose of heparin required to inhibit imaging is not known.**

**However, if these data prove comparable in humans, they suggest that imaging of thromboemboli could be achieved so promptly that only modest delay in the institution of heparin therapy would be required.**

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Many questions regarding the diagnosis, natural history, and management of venous thromboembolism are unanswered (1,2). A noninvasive technique that would allow rapid detection of venous thrombi and pulmonary emboli would clarify the situation. Recent publications suggest that intravenous injection of indium-111-labeled platelets (In-111-P), followed by imaging with a gamma camera, may provide such a

technique (3–6). Unfortunately, many factors that may condition the sensitivity and specificity of this approach cannot be controlled in patients with venous thromboembolism. For example, the age of such thromboemboli is rarely known, and heparin therapy may intervene. Accordingly in an experimental canine model in which venous thrombi were induced without trauma to the venous intima, we have addressed several questions: (a) can such venous thrombi (VT) and pulmonary emboli (PE) be visualized readily by the gamma camera when they are formed *after* intravenous injection of In-111-P; (b) can such VT and PE be visualized when In-111-P are injected after the thrombi or emboli have been aged in

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For reprints contact: Kenneth M. Moser, MD, Pulmonary Div., Dept. of Medicine, Univ. of California, San Diego School of Medicine, San Diego, CA 92103.

situ; and (c) does heparin administration interfere with visualization by In-111-P?

#### MATERIALS AND METHODS

**Platelet preparation and labeling.** Platelets were isolated by one of two methods.

1. Forty-three cc of venous blood were drawn into a clean, dry, plastic syringe containing 7 cc ACD solution (NIH Formula A). The syringe was inverted five times to ensure complete mixing. The blood was then transferred to a round-bottomed plastic tube and centrifuged for 45 sec at 7000 g. The platelet-poor plasma was removed, an equal volume of 0.9% NaCl added, and the platelets and red blood cells resuspended by gentle suctioning with a 10-ml plastic pipette. Next a second centrifugation (20 sec at 700 g) was carried out and the sample handled as described above. Finally a third spin (15 min at 200 g) was carried out. The platelet-rich suspension was then drawn off.

2. Blood was drawn as described above but was spun initially at 200 g for 15 min. This resulted in a platelet-rich plasma layer, which was removed and spun at 1400 g for 10 min. The platelet-poor plasma was removed and the platelet button resuspended in 10 cc 0.9% NaCl by gentle suctioning.

Both methods provided adequate preparations, but with the second method platelet counts showed an average of  $\sim 10^9$  platelets in the total final 10 cc of suspension, against  $10^8$  by the first method. The second method was used in all studies except Protocol 1 (see Table 1).

**Preparation of In-111-oxine compound.** The In-111-oxine complex was formed by a modification of Thakur's method (3). Three mCi of  $^{111}\text{InCl}_3$  solution in a volume of 1.5 cc was mixed with 1.5 ml of distilled

water. The pH was adjusted to 5.5 by addition of 0.3 M sodium acetate, and 0.2 ml of an oxine-ethanol solution (1 mg oxine per ml 95% ethanol) was added and the solution mixed.

After 15 min, the solution was removed and placed in a clean 16 × 150 mm Pyrex glass culture tube that contained 3 ml of dichloromethane. Following vortex mixing for 1 min, the dichloromethane layer was removed and placed in a second glass test tube. The In-111-oxine dichloromethane solution was evaporated to dryness in a hot-water bath under an air stream. The dry In-111-oxine complex could be stored at room temperature for up to 72 hr. The complex was resuspended in 0.1 ml of dimethyl sulfoxide (DMSO), and 0.1 ml 0.9% NaCl was added just before use.

**Labeling of platelets.** The In-111-oxine complex was added dropwise to the platelet suspension and allowed to incubate for 30 min at room temperature. Comparing centrifuged and uncentrifuged samples, labeling efficiency approximated 70% in several preparations.

**Production of experimental venous thrombi.** In dogs anesthetized with pentobarbital, VT were produced in the femoral vein using modified thermodilution Swan-Ganz catheters. Two new orifices were created in the catheter. The first was just proximal to the balloon; the second just distal to the original proximal Swan port. A 22-gauge, blunt-tip needle was inserted into the newly created proximal orifice and was cemented into place. Solutions injected through the "blunt needle" orifice would now emerge just proximal to the balloon.

To induce VT, the modified catheter was introduced though a venous cutdown just above the paw and the tip advanced to the femoral triangle. The location of the catheter tip was easily determined by palpation of the inflated balloon. When in position, the balloon was inflated with 1 cc of air. Two minutes later, 10 units of

TABLE 1. PROTOCOLS USED IN THIS STUDY

**Protocol 1: (10 dogs)**

In-111-P injected → Venous thrombus formed  
and imaged → venous thrombi released  
as emboli → lungs (emboli) imaged

**Protocol 2: (13 dogs)**

Venous thrombus formed → In-111-P  
injected 1 hr (5 dogs), 2 hr (2 dogs), 4 hr (2 dogs), 8 hr (2 dogs), and 10 hr (2 dogs) after thrombus formation

**Protocol 3: (10 dogs)**

Venous thrombi formed → aged 2 hr in situ then released to lungs → In-111-P injected immediately after embolization (4 dogs);  
2 hr after embolization (6 dogs)

**Protocol 4: (12 dogs)**

Venous thrombi formed → heparin given (bolus of 300 units/kg followed by 90 units/kg/hr for 6 hr (6 dogs)  
or  
saline infusion given (6 dogs).

**Protocol 5: (8 dogs)**

Identical to protocol 3 except that: (a) in 4 dogs, In-111-P injected after 2 hr of heparin infusion; (b) in 4 dogs, In-111-P injected after 4 hr of heparin infusion; protamine sulfate was given (i.v.) at 6 hr to return partial thromboplastin time to normal

topical thrombin were injected through the blunt-needle orifice and flushed in with 2 cc normal saline. Thirty minutes later, the balloon was deflated. In preliminary trials this method consistently (> 95%) produced extensive, large femoral VT. All studies used a scintillation camera equipped with a medium-energy diverging collimator. Appropriate settings were used for In-111 (174 keV) and Tc-99m (140 keV). In addition to recording images on Polaroid film, the camera was interfaced to a computer system.

**Experimental protocols.** (Table 1): Protocol No.1 (ten dogs) involved the injection of 9.5 cc In-111-P (~ 10<sup>8</sup> platelets, 2 mCi) followed by the formation of VT, release of eight of these VT to form pulmonary emboli (by gentle "milking" of the leg), and attempts to image both VT and PE. Thus, these thromboemboli incorporated In-111-P.

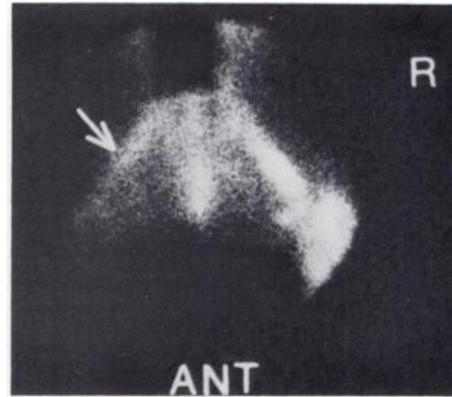
Protocol No. 2 involved the formation of nonradioactive VT in 13 animals. In each dog, a thrombus was induced in one femoral vein; the other femoral vein was treated identically except that only saline was injected after balloon inflation. In five of these dogs, In-111-P were injected through a foreleg vein at 1 hr after thrombus formation, and sequential gamma images of both femoral regions obtained. In four instances, time-activity curves were generated with the computer system using cursors placed over the femoral regions of both legs. In two dogs each, In-111-P were injected at 2, 4, 8, and 10 hr after thrombus formation, and sequential images were obtained.

Protocol No.3, in ten dogs, femoral VT were induced as before, aging them in place for 2 hr, then releasing them by milking. In four dogs, In-111-P were injected intravenously immediately after embolization; in six, In-111-P were injected 2 hr after embolization.

Protocol No.4 (12 dogs) consisted of studying VT in six pairs of dogs (e.g., Dog 1 and Dog 2). Each pair was studied concurrently. In Dog 1, VT formation was followed by In-111-P injection, using labeled platelets derived from Dog 2. In Dog 2, VT formation was followed by intravenous heparin\* administration in a dose of 300 units/kg as a bolus, followed by infusion of 90 units/kg/hr by a constant-infusion pump for 6 hr. In-111-P was injected into Dog 2 15 min after heparin infusion was begun. Sequential scintiphotos were acquired for 6 hr of heparin (Dog 2) or saline (Dog 1) infusion. Partial thromboplastin times (7) were performed before heparin infusion and hourly thereafter.

Protocol 5 (eight dogs). This was identical to Protocol 4 except that: in four dogs, In-111-P were injected after 2 hr of heparin infusion, and in four others after 4 hr of heparin infusion. At 6 hr protamine sulfate was given intravenously in a dose sufficient to return the partial thromboplastin time to normal.

In dogs in which emboli were studied, anterior, posterior, and right and left lateral lung scans were obtained



**FIG. 1.** Representative scintiphoto of femoral veins taken 15 min after thrombus induction in right femoral vein. In-111-labeled platelets had been injected before thrombus formation. Thrombus image is evident on right side (distal activity is due to edema). On left (arrow), only "background" activity is present in femoral vein.

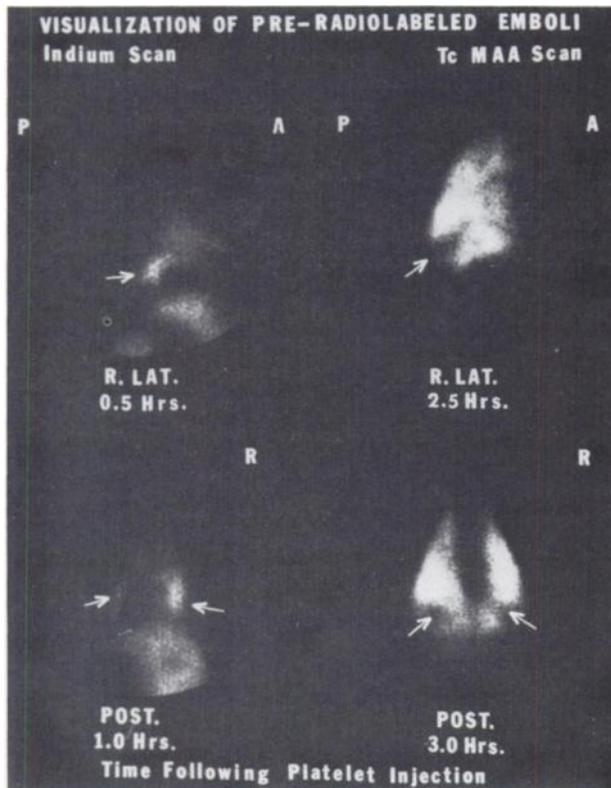
with the In-111 setting; then Tc-99m-labeled microspheres were injected and similar scans done. In all dogs, a blood sample was obtained just before sacrifice and a large dose of intravenous heparin (1000 units/kg) given those that had not received heparin. Autopsy was then performed and thrombi recovered from the veins and the pulmonary arterial system. These thrombi were minced and counted in a well counter, as were the pre-mortem blood samples. Thrombi and emboli ranged in volume from 3.1–6.8 ml; this variation reflects the variable length of thrombus formed behind the obstructing balloon.

## RESULTS

**Protocol 1.** In the ten dogs in which In-111-P were injected before venous thrombus formation, the thrombi were readily detectable by gamma imaging within 15 min after thrombus formation. The contralateral femoral vein did *not* image despite the same manipulation and period of balloon occlusion (Fig. 1). Optical microscopy of the intima of control veins disclosed no abnormality.

When these In-111-P-containing venous thrombi were embolized in eight dogs, they were visualized by lung scanning (Fig. 2) in all. In the first two dogs studied, however, the thoracic anatomy of the dog, with its relatively large heart and small lung fields as seen in anterior and posterior views, tended to obscure centrally-lying In-111-P emboli. Hyperinflation of the lungs during imaging substantially improved our ability to image emboli in the next six dogs. In these animals there was excellent agreement between the In-111-P embolic images and the zones of hypoperfusion on the Tc-99m perfusion scans (Fig. 2).

**In-111-P injected following thrombus formation (Protocol 2).** Of these 13 dogs, In-111-P were injected

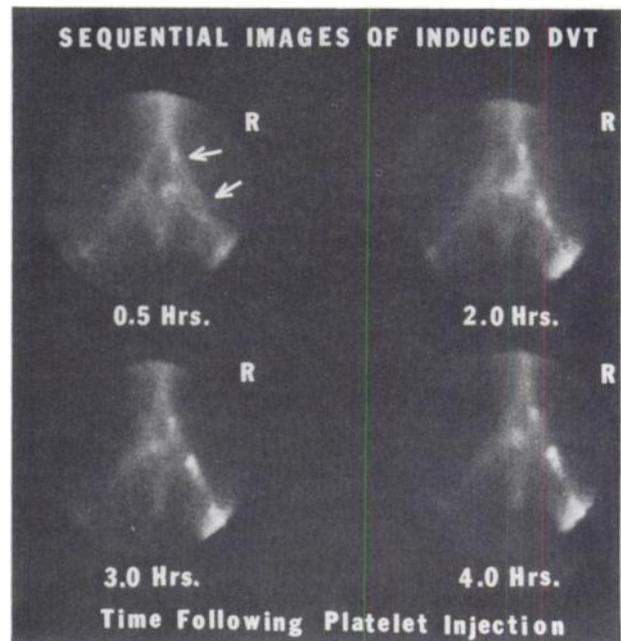


**FIG. 2.** Lung scans in dogs in which In-111-labeled platelets were injected, then femoral thrombus formed and aged for 0.5 hr, then released. Upper left: In-111 image immediately after thrombus release. Lower left: In-111 image 30 min later. These images were unchanged over next 3 hr. On right are corresponding perfusion scans taken 2 hr after initial In-111 images. Agreement between In-111 emboli and Tc-99m perfusion defects (arrows) was consistently excellent.

one hour after thrombus induction in five (that is, 30 min after balloon release). A positive image was detectable 20–90 min after In-111-P injection (average = 39 min) in each (Fig. 3). Examination of the time-activity curves (Fig. 4) showed a rapid initial increase over the site of the induced VT. Neither a positive image nor an increasing activity curve was seen over the control leg. Dissection, in each dog, confirmed thrombus as the origin of the positive images. Thrombus-to-blood activity ratios ranged from 5.5–18. Furthermore, when these thrombi were embolized, the leg images became “cold” (Fig. 5), indicating the absence of In-111-P accumulation on the venous intima per se.

In the eight dogs in which venous thrombi were “aged” for 2, 4, 8, and 10 hr before injection of In-111-P, the thrombi could be imaged from 10–60 min (average: 36 min) after In-111-P injection. Thus “age” did not influence the time at which the image became detectable.

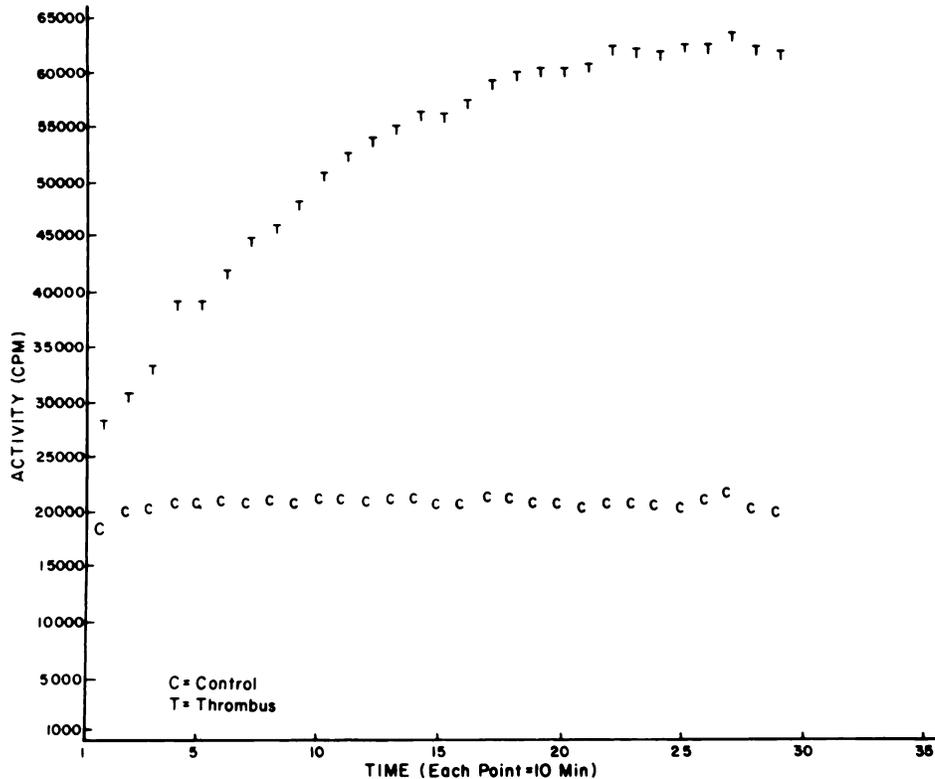
**In-111-P injected following embolization (Protocol 3).** In the ten dogs in which VT were aged in situ for 2 hr then embolized, In-111-P were injected *immediately* after embolization in four. Positive imaging of emboli



**FIG. 3.** Sequential images in dog in which In-111-labeled platelets were injected 1 hr after thrombus induction. Thrombus image was consistently detectable 20–90 min after platelet injection. No image was detectable in contralateral femoral vein. In this dog, thrombus was detectable at 25 min (arrows) and its activity then increased.

was successful in one dog but equivocal in three, despite abnormal Tc-99m perfusion scans in all. In the next six dogs, in which In-111-P were injected *two hours* after embolization, readily detectable images of these emboli were seen in all six dogs within 90 min after In-111-P injection, in regions that were positive by perfusion scan (Fig. 6). Autopsy of these animals disclosed that, in all three of the “equivocal” In-111-P dogs, large emboli totally occluded a major central pulmonary artery (right, left, and/or lobar). In the other dogs, the emboli did not appear to occlude the involved vessel(s) totally. Thrombus-to-blood radioactivity ratios ranged from 2 to 4 in the equivocally visualized thrombi and 4 to 12 in those visualized. Unfortunately, the emboli were not examined microscopically or by autoradiography, nor were counts from proximal parts of the emboli segregated from those obtained from distal portions.

**Effect of heparin administration (Protocol 4).** In the six dogs treated with continuous infusion of heparin, In-111-P injected 15 min after the initial bolus failed to provide a positive image of the venous thrombus during the 6-hr period of observation, and time-activity curves over the femoral thrombi were identical to those obtained over the control leg (Fig. 7). In all of the control dogs, which received the same batch of In-111-P but only a saline infusion without heparin, imageable thrombi were obtained within 60 min after In-111-P injection. Autopsy disclosed extensive venous thrombi in all dogs. Thrombus-to-whole-blood ratios ranged from 0.44 to 0.72 in heparin-treated dogs, and from 6 to 10 in control dogs.



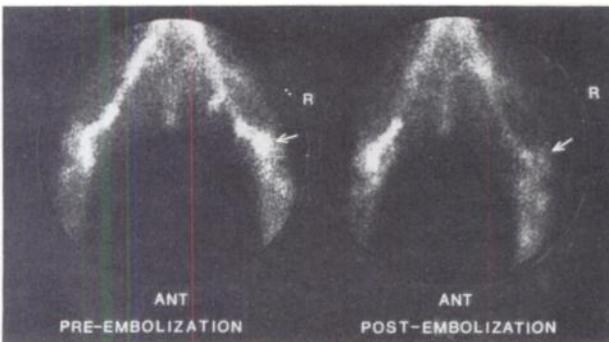
**FIG. 4.** Time-activity curves over femoral area with thrombus (T) and control femoral area in contralateral leg (C). Total duration of observations was 300 min. Increasing activity over thrombus area is evident to about 200 min. Activity over control area shows no significant change from 10–300 min.

Hourly partial thromboplastin times in treated dogs were consistently > 90 sec (the maximum time allowed for the test) against a control averaging 12 sec.

**Protocol 5.** In dogs in which In-111-P were injected after 2 or 4 hr of heparin infusion, the results were identical: thrombus imaging failed to occur. When protamine sulfate was given at 6 hr, thrombi previously not imaged became visible within 40 min, and thrombus-to-blood ratios ranged from 6 to 12.

#### DISCUSSION

Other investigators have reported the successful

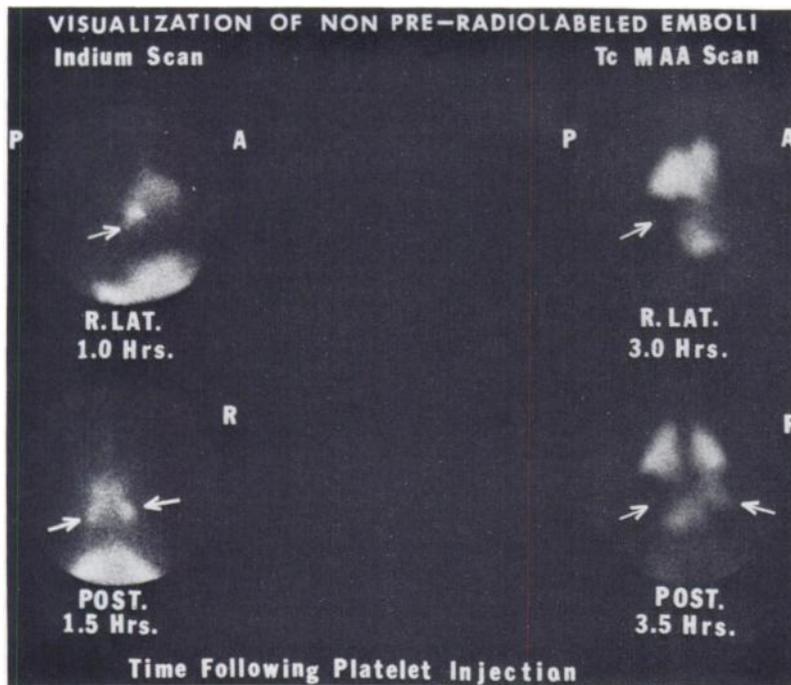


**FIG. 5.** Dog in which bilateral venous thrombi were labeled with In-111 platelets injected *after* formation. Thrombus on right (arrow) was embolized by gentle “milking” of leg.

positive imaging of VT and PE following injection of In-111-P in both animals and man (3–6). Our experience confirms these findings and provides new information regarding the timing of observations and the effect of heparin administration. It is clear that the images of VT and PE obtained in our animal model are derived from the thromboemboli, not intimal injury. Our model does not involve the induction of venous intimal injury, which others have used (3–6). We have not imaged platelet deposition in the many control veins studied, nor has platelet deposition been observed on the intima of these control veins by light microscopy, even though these veins were subjected to balloon occlusion. Also, after embolization of imageable femoral thrombi, the femoral image consistently disappeared.

Our initial series of observations were useful in demonstrating that VT formed *after* injection of In-111-P—thereby incorporating In-111-P at the time of formation—are readily visualized within minutes after thrombus formation. This is not only reassuring (since at this time “background” radiation in circulating blood is substantial) but also may have relevance to the potential use of In-111-P as a “monitoring” approach in patients at high risk of VT, much in the fashion that I-125 fibrinogen is now used (9).

Furthermore, when embolized, such thrombi are readily detected in the lungs by the gamma camera. The



**FIG. 6.** Images in dogs in which emboli were released and In-111-labeled platelets were injected 2 hr after release. In this dog, pulmonary emboli were readily detectable at 1 hr after platelet injection (left), remained imageable throughout the next 4 hr of study, and corresponded to zones of perfusion deficit (right).

initial difficulty we experienced with centrally lying emboli is not likely to occur in humans because of their more favorable thoracic configuration. Should it occur, however, gated imaging at end-inspiration, or during breathhold at total lung capacity, is likely to be helpful.

It is also evident that VT formed before injection of In-111-P incorporate sufficient In-111-P to become detectable within 20–90 min. This applies to thrombi studied from 1–10 hr after formation. These are useful observations because “aging” of a thrombus in vivo may alter it in a fashion that militates against platelet accretion upon it. In our model this does not occur for at least 10 hr. How “old” a thrombus can be, and still be detected, is not known. Extending such studies in the dog is difficult because spontaneous thrombolysis begins to supervene (8). Presumably, the technique will be useful as long as thrombin remains available on the thrombus surface. If one is justified in extrapolating from I-125 fibrinogen data in man, this period may last 3–5 days in the absence of heparin therapy (9,10). In-111-P imaging of thrombi thus will be subject to the same “biologic” limitation as I-125 fibrinogen; that is, only “active” thrombi will be detectable. In humans, in whom thrombus “age” is never known with precision, we can only accumulate data relating duration of symptoms, venographic results, and I-125 fibrinogen or impedance phlebographic data to clarify this issue.

Our data also disclose that fresh experimental emboli released before In-111-P injection also became imageable promptly. The “oldest” emboli studied were 4 hr old: aged 2 hr before release and 2 hr after release. Several factors were found to condition the imaging

success of emboli. Very “fresh” emboli have had no opportunity to undergo fibrinolytic dissolution (8). Therefore, these femoral-vein-sized emboli tend to lodge in, and completely occlude, major central pulmonary arteries. This permits access of circulating In-111-P only to the most proximal (exposed) portion of the embolus, that is, the portion lying directly within the large “heart pool” of the dog. This made clear identification of the thrombus image difficult when In-111-P were injected immediately after embolization. However, after a 2-hr postembolic wait, some dissolution of the embolus is known to occur (8). It appears likely that this permits access of In-111-P to a more extensive thrombus surface. This would explain our consistent success in imaging when we waited 2 hr after embolic release to inject In-111-P. In other instances in which In-111 images were negative—despite the finding of emboli in small vessels at autopsy and despite positive Tc-99m scans—it also appeared that total occlusion was the problem. Thus, exact correlation perfusion scan and indium scan does not exist in the experimental animal and should not be anticipated in humans. The perfusion scan will continue to depict blood-flow distribution; the indium scan will depict platelet incorporation into “active” and “accessible” emboli.

The preliminary results with heparin are of interest in several regards. We have previously shown that VT in dogs treated with lower doses of heparin soon after clot formation fail to develop “lines of Zahn”—those layers representing accretion of fibrin and platelets on an active thrombus (8). The present report, using positive imaging, time-activity curves, and thrombus-to-blood ratios, demonstrates (with high-dose, constant-infusion hepa-

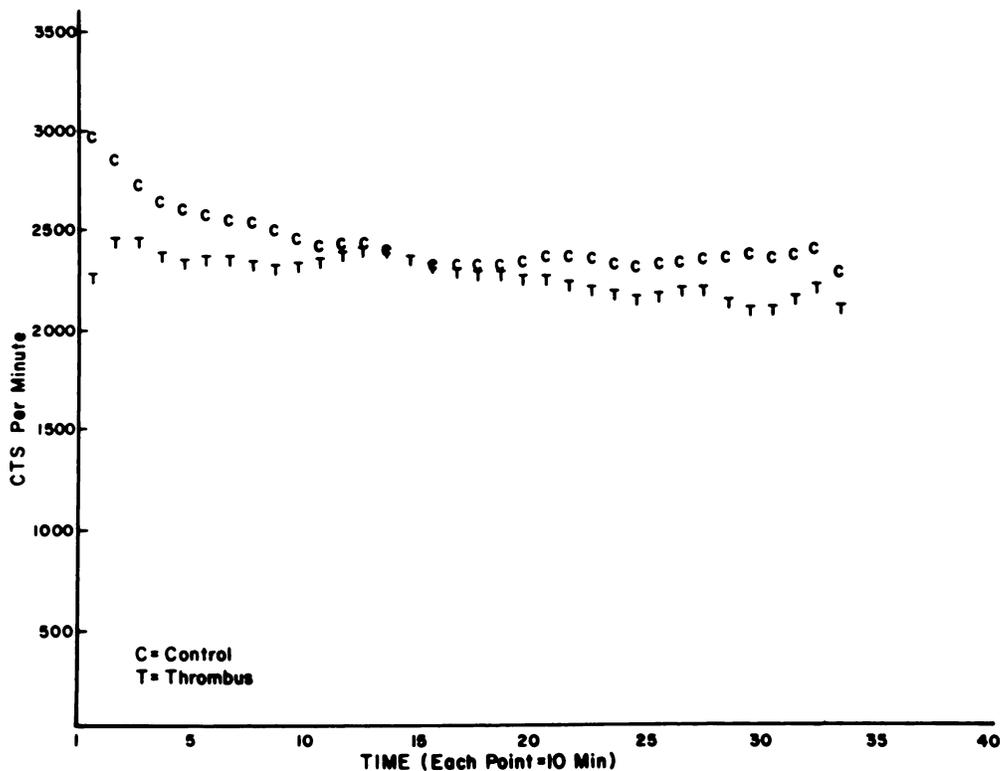


FIG. 7. Time-activity plots over thrombus (T) and control femoral veins (C) in dogs given heparin before injection of In-111 platelets. No increase in activity was noted over either region during 6 hr of observation.

rin) the same phenomenon, whether In-111-P are injected after 15 min, 2 hr, or 4 hr of heparin infusion. At what heparin infusion rate (or partial thromboplastin time) platelet accretion may be inhibited in the dog is the object of continuing study. From the results of protamine injection, however, it is clear that In-111-P remain capable of thrombus attachment when heparin is inhibited, as measured by return of partial thromboplastin time to normal. We view this as a promising model for studying the efficacy of anticoagulant-antithrombotic regimens. Further, administration of heparin before In-111-P injection in man may compromise the diagnostic value of the technique, a fact to be borne in mind in future investigations.

Definition of the ultimate utility of this approach to human thromboembolic disease clearly requires further extensive and careful study in both animals and man. The available data (5) suggest that extension to man is at least feasible. Furthermore, in animal models, as our data indicate, the technique offers a valuable means of answering questions regarding the diagnosis, natural history, and treatment of experimental thromboembolism, and this can serve to enhance the design and understanding of future observations in humans.

#### FOOTNOTE

\* USP units; aqueous sodium heparin.

#### ACKNOWLEDGMENT

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**ANNUAL SPRING MEETING  
Pacific Northwest Chapter  
Society of Nuclear Medicine**

**March 27-29, 1981**

**Alderbrook Resort  
ANNOUNCEMENT**

**Union, Washington**

Drs. Raymond Marty, Program Chairman and Michael Graham, Program Co-Chairman announce the following plans for the Pacific Northwest Chapter Spring Meeting.

Clinical Aspects of single photon emission tomography.  
Practical aspects and applications of the 400T system.

John Keyes, M.D.  
Dave Williams, M.D., James Ritchie, M.D., James Caldwell, M.D., and Glen Hamilton, M.D.  
Thomas Davis, M.D.

Combined Imaging Modalities in the evaluation of the Abdomen.  
Nuclear Medicine, Ultrasound, CAT scans and conventional radiography in the evaluation of renal function.

Tom Rudd, M.D.  
John Denney, M.D.

Evaluation of the gallbladder and biliary tree by various imaging modalities.  
General overview of the various imaging modalities and their appropriateness and cost effectiveness.

Wil Nelp, M.D.

There will also be a Technologist sponsored program.

Application for AMA category I credit for physicians will be on file.

There will be a Chapter General Business Meeting on Saturday, March 28, 1981 at the scheduled lunch.

For further information and hotel and registration cards, please contact: Jean Parker, Administrator, Pacific Northwest Chapter, SNM, P.O. Box 40279, San Francisco, CA 94140.

**NORTHERN CALIFORNIA CHAPTER  
SOCIETY OF NUCLEAR MEDICINE  
MIDWINTER MEETING**

**January 17-18, 1981**

**Doubletree Inn**

**Monterey, California**

The Northern California Chapter will hold its Annual Midwinter Meeting in Monterey, California, January 17 and 18, 1981. This will be a weekend meeting with invited lectures presented on Saturday and Sunday mornings. There will be a reception and dinner on Saturday evening with Dr. Fred Bonte as the invited speaker.

Saturday morning will include a minisymposium on monoclonal antibodies and their implications for nuclear medicine. Invited speakers are Professor Henry Kaplan of Stanford University and Dr. Steven Larson from the University of Washington, Seattle. An additional portion of the program will be devoted to new techniques in cardiology including applications of dynamic computerized tomography as well as those using nuclear medicine techniques. The invited speakers for this portion of the program are Drs. Robert Herfkens and John Verba. The Technologist Section is planning a program for Saturday afternoon. Plans are also underway for a tennis tournament and other social functions.

For further information please contact Jean Parker, P.O. Box 40279, San Francisco, CA, 94140 or call (415) 647-1668 or 647-5909.

Dr. Howard Parker is General Program Chairman and Dr. Paul Weber is Scientific Program Chairman. This is an approved program for Category I CMA Continuing Medical Education Credits.