piratory symptoms (*1*). Seventy of these patients were homosexual males, the other patient was a female with Von Willebrand's disease. There were no i.v. drug abusers represented in their study.

Intravenous drug abusers comprise ~17% of AIDS cases in the United States (*2*), although they have accounted for <2% of AIDS victims here in Canada (*3*). Intravenous drug abusers are at increased risk for a variety of disorders, including pulmonary talc granulomatosis (*4*). To date there has been only occasional reference to pulmonary talc granulomatosis (PTG) in the literature on AIDS-related pulmonary disease.

We recently reported a case of PTG that clinically simulated *Pneumocystis carinii* pneumonia (*5*). The patient was a 32-yr-old HIV-positive bisexual male intravenous drug abuser who presented with respiratory symptoms. Routine chest radiology showed some nonspecific changes in the lower regions of both lungs. However, ^{67}Ga scintigraphy revealed marked bilateral diffuse pulmonary uptake. Transbronchial biopsy and bronchoalveolar lavage resulted in a definitive diagnosis of pulmonary talc granulomatosis. Thus pulmonary talc granulomatosis should be added to the list of pulmonary lesions that give rise to a positive ^{67}Ga lung scan in patients with or at risk for AIDS.

References

1. Kramer EL, Sanger JJ, Garay SM, et al. Gallium-67 scans of the chest in patients with acquired immunodeficiency syndrome. *J Nucl Med* 1987; 28:1107–1114.
2. Fauci AS, Macher AM, Longo DL, et al. NIH Conference. Acquired immunodeficiency syndrome: epidemiologic, clinical, immunologic and therapeutic considerations. *Ann Intern Med* 1984; 100:92–106.
3. Elmslie K, Nault P. Epidemiologic reports: AIDS surveillance in Canada. *Can Med Assoc J* 1986; 135:780.
4. Arnett EN, Battle WE, Russo JV, et al. Intravenous injection of talc-containing drugs intended for oral use. A cause of pulmonary granulomatosis and pulmonary hypertension. *Am J Med* 1976; 60:711–718.
5. O'Connor RN, Brunham RC, Bowden DH. Pulmonary talc granulomatosis mimicking pneumocystis carinii pneumonia. *Can Med Assoc J* 1988; 128:53–54.

R.N. O'Connor
R.C. Brunham
D.H. Bowden
Health Sciences Centre
Winnipeg, Manitoba
Canada

Prognostic Value and Pathophysiologic Significance of the Rim Sign in Cholescintigraphy

TO THE EDITOR: It was with great interest that we read the article by Meekin et al. in the *JNM* (*1*). All of their 27 patients had been fasting at least 2 hr but <48 hr prior to the study. It was mentioned that none of them received cholecystokinin. However, we would like to know whether some or all of them received low dose morphine (*2*) and/or a fatty meal (*3*). Furthermore, for a quicker diagnosis, ultrasonography may provide a safer alternative to scintigraphy, because pericholecystic edema is visualized as hypoechoic areas and triple-layered thick-walled gallbladder is characteristic in acute cholecystitis.

References

1. Meekin GK, Ziessman HA, Klappenbach RS. Prognostic value and pathophysiologic significance of the rim sign in cholescintigraphy. *J Nucl Med* 1987; 28:1679–1682.
2. Kim EE, Nguyen M, Pjura G, et al. Use of morphine in cholescintigraphy for obstructive cholecystitis [Abstract]. *J Nucl Med* 1985; 26:P79.
3. Willson SA. The fatty meal biliary tree diagnosis. *Semin Ultrasound, CT and MR.* 1987; 8:114–117.

M.A. Taher
Institute of Nuclear Medicine
Bangladesh Atomic Energy Commission
Postgraduate Hospital, Shahbag, Dhaka
Bangladesh

REPLY: In reply to Dr. Taher's question, no patient in our study received morphine or a fatty meal prior to the study. Pericholecystic fluid, usually a sign of gallbladder perforation, is seen in only 10–25% of cases of acute cholecystitis with ultrasonography and may also be seen with pancreatitis, peptic ulcer, liver abcess, peritonitis, or hemorrhage from a rupture of a hepatic adenoma or an ectopic gestation (*1,2*). Marchal et al. described continuous or localized anechoicities within a thickened, irregular wall as a sign of acute cholecystitis and ascribed it to a zone of edema (*3*). One study found anechoic areas in the wall of 71% of patients with acute cholecystitis (*4*). However, gallbladder thickening with or without anechoicities has been seen in a variety of conditions unassociated with intrinsic gallbladder disease. The majority of conditions with associated wall thickening have a common physiological basis for this finding, i.e., increased transudation of fluid into the extravascular space secondary to decreased plasma oncotic pressure or increased portal or systemic venous pressure. These conditions include hypoproteinemia, cirrhosis, congestive heart failure, renal failure, focal obstruction of gallbladder lymphatic drainage, e.g. by a porta hepatis mass (*5*). In a comparative study of 100 patients with pathologically proven acute cholecystitis, realtime ultrasonography had a sensitivity of 24% using strict criteria (wall edema and/or pericholecystic fluid), 86% using liberal and less specific criteria (stones, thick wall, nonshadowing echo and/or Murphy's sign), compared to 97% for cholescintigraphy (*6*). Another study comparing real time ultrasonography with cholescintigraphy found similar sensitivities for confirmed acute cholecystitis (97%), but scintigraphy demonstrated better specificity (93% vs. 64%) (*7*). In this study, echogenic foci with acoustical shadowing as well as wall edema, and/or pericholecystic fluid was used as diagnostic criteria. Therefore, we do not agree with Dr. Taher's suggestion that ultrasonography might be preferable to cholescintigraphy for the routine diagnosis of acute cholecystitis. However, in the setting of prolonged fasting or intercurrent illness, where cholescintigraphy is still sensitive, but less specific than desired, gallbladder sonography may be a reasonable alternative (*8*).

Clinically, the rim sign is an ancillary finding for the diagnosis of acute cholecystitis in the setting of non-visualization of the gallbladder. It appears to be an indicator of patients presenting at a later stage of the pathological spectrum of acute cholecystitis and, therefore, at increased risk for complications. If the sign holds up as specific for acute cholecystitis, it may also be helpful in decreasing the length of the

study in those cases of chronic cholecystitis that might otherwise require 2-4 hr for delayed filling of the gallbladder (*9*).

References

1. Madrazo BL, Francis I, Hricak H, et al. Sonographic findings in perforation of the gallbladder. *Am J Roentgenol* 1982; 139:141–496.
2. Nyberg DA, Laing FC. Ultrasonographic findings in peptic ulcer disease and pancreatitis that simulate primary gallbladder disease. *J Ultrasound Med* 1983; 2:303–307.
3. Marchal GJF, Casaer M, Baert AL, et al. Gallbladder wall sonolucency in acute cholecystitis. *Radiology* 1979; 133:429–433.
4. Raghavendra BN, Feiner HD, Subramanyam BR, et al. Acute cholecystitis: sonographic-pathological analysis. *Am J Roentgenol* 1981; 137:327–332.
5. Durrell CA, Vincent LM, Mittelstaedt CA. Gallbladder ultrasonography in clinical context. *Semin Ultrasound, CT and MR* 1984; 5:315–332.
6. Fink-Bennett D, Freitas JE, Ripley SD, Bree RL. The sensitivity of hepatobiliary imaging and real time ultrasonography in the detection of acute cholecystitis. *Arch Surg* 1985; 120:904–906.
7. Samuels BI, Freitas JE, Bree RL, Schwab RE, Heller ST. A comparison of radionuclide hepatobiliary imaging and real-time ultrasound for the detection of acute cholecystitis. *Radiology* 1983; 147:207–210.
8. Samuels BI, Freitas JE, Gross MD. A pragmatic review of gallbladder imaging. *Semin Ultrasound, CT and MR,* 1985; 6:156–171.
9. Bushnell DL, Perlman SB, Wilson MA et al. The rim sign: association with acute cholecystitis. *J Nucl Med* 1986; 27:353–356.

Harvey A. Ziessman
Gregory K. Meekin
Georgetown University Hospital
Washington, DC

Correction: Figure Corrections

Due to a printer's error, Figures 1 and 2 on pp. 1284 and 1285 of "Thallium-201/Technetium-99m-RP-30A Disparity in the Course of Myocardial Infarction After Attempted Reperfusion" by Tatum et al. (J Nucl Med 1988;29:1283–1286) are shown incorrectly. The correct orientation is shown below.

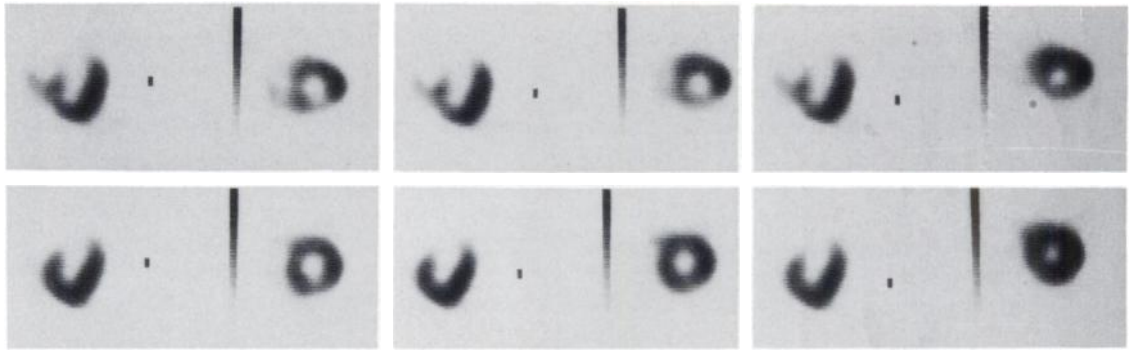

FIGURE 1
Long axis (left) and corresponding short axis (right) tomographic images of the heart immediately following stress (top) and on redistribution (bottom) delayed 4 hr following injection of ^{201}Tl. Stress images demonstrate a small anterior wall perfusion defect seen on the most proximal (basilar) short axis slice. Redistribution images demonstrate an extensive region of "reverse redistribution" involving the entire antero-septal region.

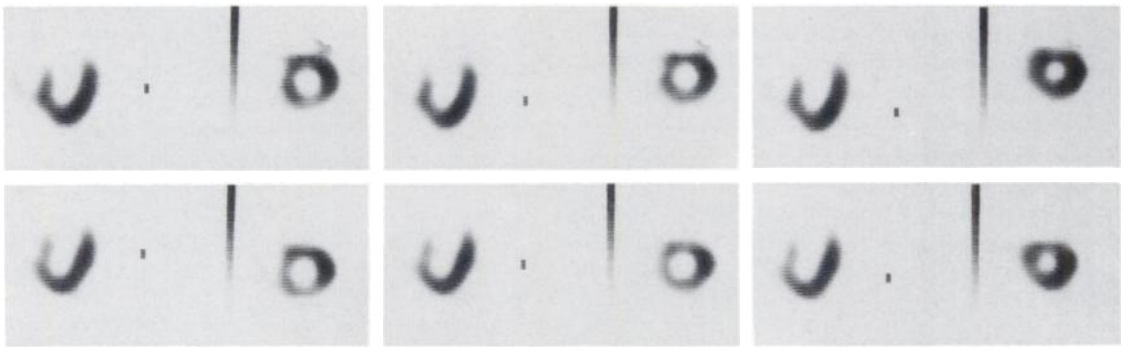

FIGURE 2
Long axis (left) and corresponding short axis (right) tomographic images of the heart obtained following ^{99m}Tc-RP-30A administration at stress (top) and rest images obtained following ^{99m}Tc-RP-30A injection at rest (bottom). Stress images demonstrate an extensive antero-septal perfusion defect. Rest images continue to demonstrate a small proximal anterior wall defect however there is significant improvement in the antero-septal region.