certain class, the true frequency of that class could still be as high as 28%.

The conclusions we should draw are obvious. First, a technique that shows normal visualization in as many as five of nine cases with proven gallbladder disease cannot be expected to show transient nonvisualization in acute pancreatitis. Second, even if the technique had been 100% accurate and all the figures correct, elementary statistics show that transient nonvisualization could still occur as often as in one of four cases with acute pancreatitis.

According to Ali et al. we "assert that in such cases (of pancreatitis) normal gallbladders frequently fail to visualize." These were certainly not our words. Our figures did not allow us to express an opinion as to the frequency of transient nonvisualization in acute pancreatitis. Our aim was to draw attention to the fact that transient nonvisualization can occur, a phenomenon that is documented by images presented in our paper (*2*). It is hardly a reason for great wonder and excitement. Temporary failure of gallbladder visualization at cholecystography in acute pancreatitis was first described some 40 years ago (*5, 6*). A prospective comparative study of different diagnostic procedures to evaluate gallbladder function in acute pancreatitis is at present being performed in our hospital. It is only under the strict and standardized conditions of a prospective study, as opposed to a retrospective "review of results," that problems of this nature can be solved.

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Reply

We are dismayed that Drs. van der Linden et al. feel we have taken them "to task for having observed transient nonvisualization" of the gallbladder in patients with acute pancreatitis (*1*); certainly, that was not our intent. On the assumption that a free and honest exchange of ideas regarding important clinical problems benefits the entire medical community and its patients, we have reported conclusions regarding cholescintigraphy in pancreatitis that differ from theirs (*2*). We regret that they have found our dissenting opinion offensive.

Van der Linden et al. state that we have misrepresented their position regarding the frequency of nonvisualization of normal gallbladders in patients with acute pancreatitis. In the discussion section of their paper (*1*) they make the following series of statements: "In five of seven patients with acute pancreatitis, the gallbladder failed to visualize by Tc-99m HIDA scintigraphy performed during the attack"; "Thus, in all five patients the scintigrams gave false information"; "The reliability of Tc-99m HIDA scintigraphy in acute pancreatitis is disputed"; and, "scintigraphy may be even less reliable in acute pancreatitis than is oral cholecystography." We have taken these remarks to mean that nonvisualization of normal gallbladders during cholescintigraphy of patients with acute pancreatitis is frequent enough to render the test unreliable for diagnosis of acute cholecystitis when pancreatitis is present. If this were not the intended meaning, we apologize for our misunderstanding but we do not see an alternative interpretation.

Van der Linden et al. point out that we failed to identify chronic cholecystitis in five of our patients. They take the fact that the gallbladder visualized in these five cases to indicate that our technique is faulty. This conclusion, of course, is completely without justification. Certainly they must realize that cholescintigraphy is not useful in the evaluation of gallbladder disease in general. On the contrary, the unreliability of cholescintigraphy in the identification of chronic cholecystitis has been well documented (*3-5*). It is irrelevant to this discussion in any case, since we have addressed ourselves to the detection of acute cholecystitis in the presence of pancreatitis, not the detection of chronic cholecystitis. We are pleased at the comment in their letter that our "technique appears capable of differentiating . . . . . acute . . . . . cholecystitis" in our patients with acute pancreatitis, since that is the most important conclusion of our paper (*2*). We were nonplussed, however, by their assertion that a diagnosis of acute cholecystitis can be established by monitoring a patient's temperature, given the obvious fact that fever is present in many diseases that are not related to the gallbladder. The utility of cholescintigraphy in the diagnosis of acute cholecystitis is well documented in the literature (*4-9*), and surgeons at our institution have found the procedure very helpful in their daily practice.

Van der Linden et al. are surprised that in our series some patients who had normal cholescintigrams later underwent cholecystectomy. They interpret this to mean that "Ali et al. tend to overlook cases of cholecystitis" and that "the surgeons eventually chose to ignore Ali et al.'s assertion that the cholescintigram was normal." We regard these remarks as ill-considered. Acute cholecystitis is not the only indication for cholecystectomy. The patients with normal cholescintigrams in our series, who underwent elective cholecystectomy after acute pancreatitis had subsided, were operated on because they had cholelithiasis demonstrated by oral cholecystography or ultrasonography. Cholecystectomy was indicated because cholelithiasis is a common cause of acute pancreatitis. The patients were spared undesirable emergency cholecystectomy during an acute episode of pancreatitis precisely because the surgeons at our institution have confidence in the cholescintigram and in our interpretation of the study. They recognized that visualization of the gallbladder indicates the absence of acute (not chronic) cholecystitis, and were willing to treat their patients conservatively until acute pancreatitis subsided (*3,4,6,8,9*). In contrast, four of five acutely ill patients with nonvisualization of the gallbladder on delayed imaging (as long as 4 hr after tracer administration) (*4,7*) underwent immediate cholecystectomy, and all had acute cholecystitis as well as pancreatitis. With regard to the suggestion of van der Linden et al. that we may have missed the acute phase of cholecystitis in patients with normal cholescintigrams, we can only point out that the sensitivity of cholescintigraphy for acute cholecystitis is well established (*3,4,7,9*) and note that all patients in this group did extremely well with conservative management.

Van der Linden et al. have commented that our series was not large enough to determine precisely the incidence of the phe-

nomenon of "transient nonvisualization" of the gallbladder, as defined by Edlund et al. We agree. Moreover, it would have been impossible for us to demonstrate this phenomenon, even if we had wanted to do so, since most patients with nonvisualizing gallbladders (4 of 5) underwent immediate cholecystectomy and thus could not be re-examined for normal gallbladder filling at a later date. It is possible that the two patients in our series whose gallbladders were not visualized until 90 min after injection might have had visualization within 1 hr at a second examination after their acute pancreatitis had subsided, but we did not repeat the studies. Thus we cannot (and did not) rigorously challenge the data of Edlund et al., since our study was quite different from theirs. Rather, we wished to dispute the validity of their conclusion that "scintigraphy may be even less reliable in acute pancreatitis than is oral cholecystography" (*1*).

Our contention is that cholescintigraphy is reliable in the detection of acute cholecystitis, even in the presence of acute pancreatitis, provided that delayed views are taken when the gallbladder is not visualized within 1 hr, as described by Weissmann et al. (*7*). (In fact, we suggested in our paper that Edlund et al. might have observed gallbladder filling in one or more of their five patients with nonvisualization had they not limited their examination time to 1 hr.) If they feel we are bold to draw such a conclusion from a series of 21 patients and other data in the literature, they should recall that there were only seven patients in their own series.

We did not include images in our paper since they would have added nothing to the report. Our methods of performing and interpreting the cholescintigrams are widely used, and they were well described and referenced in our paper. Van der Linden et al. call into question our use of subjective interpretations of images by an experienced observer to determine whether tests were positive or negative. That is the usual way in which cholescintigrams (and most other diagnostic images) are interpreted by practitioners the world over. Thus we fail to understand their objection. Furthermore, we fail to understand what relevance oral cholecystography would have had to our conclusions.

We understand the pitfalls of retrospective clinical studies, and we wish Drs. van der Linden et al. well in their "prospective comparative study" of gallbladder function. We hope they will examine their patients for longer than 1 hr in cases in which the gallbladder is not visualized within that time. We also hope they will take notice of the rapidly accumulating evidence that patients who do not eat for a prolonged period (e.g., those on total parenteral nutrition) may exhibit nonvisualization of the gallbladder on that basis alone and that factor will be controlled in the interpretation of their data (*10,11*). In any event, pending the outcome of their study or reports of others that might be forthcoming, we must continue to believe, "on the basis of data available to us, that cholescintigraphy is as useful for detecting acute cholecystitis in patients with acute pancreatitis as it is in patients without the latter disease" (*2*).

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Re: Thyroid Activity on Technetium-99m Macroaggregated Albumin Lung Scans

Perfusion lung scanning using technetium-99m macroaggregated albumin is one of the most common nuclear medicine studies performed. Extrapulmonary activity is rare in these studies unless associated with right-to-left intracardiac or intrapulmonary shunts, or with poor quality control. When seen in the thyroid, extrapulmonary activity suggests thyroid disease as a possible cause, and could lead to an unsuspected diagnosis. We report two cases of thyroid activity seen on perfusion lung scans in patients with Graves' disease and Hashimoto's thyroiditis.

Case 1. A 30-yr-old female presented with left lower pleuritic chest pain. The physical examination was unremarkable except for a friction rub heard in the region of the left lower lobe. A chest radiograph was normal. Ventilation/perfusion lung scans were performed because of the possibility of pulmonary embolism. The result was normal, excluding pulmonary embolism, but during the perfusion image, performed 10 min after intravenous injection of 4 mCi of Tc-99m macroaggregated albumin, notable thyroid activity was observed (Fig. 1, top). Uptake in salivary glands, stomach, brain, and kidney was sought, but not found.

Because of the appreciable uptake of activity in the thyroid, the patient was evaluated for thyroid disease. Serum thyroxine was 2.8 μg/dl; thyroid-stimulating hormone (TSH) was greater than 40 μIU/ml. Thyroid uptake and scan with 200 μCi of iodine-123 revealed elevated uptake: 28% at 4 hr, 41% at 6 hr, and 55% at 24 hr. The thyroid images showed no evidence of anatomic abnormality. Antithyroid antimicrosomal antibodies were positive, with a titre of 1: 6400. The diagnosis of Hashimoto's thyroiditis was made, and the patient was appropriately treated.

Case 2. A 72-yr-old woman had weight loss, palpitations, and dyspnea for approximately 3 wk. She had a history of heart disease, for which she was on digoxin and propanolol. Physical examination revealed a pulse of 110/min, a blood pressure of 140/70 mm Hg, and normal temperature and respiratory rate. The examination