and metastases will cause comparable defects in a colloid scan. However, liver scan results combined with other diagnostic procedures may be used effectively to obtain a correct diagnosis, as in the differentiation of focal nodular hyperplasia from hemangioma (J). In fatty infiltration the density values of TCT fall, which results in a smaller contrast difference between the liver parenchyma and metastases. Circumscribed solid tumors may thus appear iso- or even hyperdense when compared with normal liver tissue. In these patients SPECT appears to be the diagnostic modality of choice, even being superior to TCT.

We do agree with Buell that the combination of ultrasound and SPECT should be assessed to determine whether a major information gain can be obtained when results of the two procedures are combined.

In summary, we feel that SPECT should be used in the following situations: 1. In patients with fatty infiltration of the liver; 2. when the results of TCT or ultrasound are equivocal and when the suspected lesion has a diameter above 1.5 cm; 3. for follow-up of known hepatic metastases, where SPECT has the advantage over ultrasound in obtaining reproducible, standard cross sections; and 4. in combination with ultrasound, especially when TCT is not available.

LUDWIG STRAUSS
FRANK BOSTEL
JOHN H. CLORIUS
Klinikum Mannheim
Mannheim, West-Germany

REFERENCES

Re: Uptake of Tc-99m MAA by the Liver During a Lung Scan
In patients with iliac vein or inferior vena cava occlusion the uptake of lung imaging agents by the liver following injection in the lower extremities has been documented previously (1,2). In contrast to the recent report by Marcus and colleagues (3) where there appeared to be uniform distribution in the liver, the earlier cases show preferential uptake in the left lobe, suggesting shunting through the umbilical vein.

E. P. WRAIGHT
Addenbrookes Hospital
Cambridge, England

REFERENCES

Re: Tc-99m IDA Cholescintigraphy in Acute Pancreatitis
In the October issue of the Journal, Ali et al. present a retrospective review of the results they obtained from Tc-99m IDA cholescintigraphy in acute pancreatitis (J). Declaring cholescintigraphy to be "useful for detecting acute cholecystitis in patients with acute pancreatitis" they take us to task for having observed transient nonvisualization in such patients (2).

First, let us have a look at the design of their study. In their files Ali et al. came across 21 patients with symptoms and signs suggestive of acute pancreatitis. They analyzed the "interpretations of the cholescintigrams by an experienced observer" but do not present any images. In none of the 21 patients was the examination repeated or cholecystography performed, but the findings at operation are given in nine patients who were subjected to surgery.

Now for a look at the results in the 21 patients. Visualization occurred in 16 patients. Five were operated on and "all five were found to have... chronic cholecystitis." Nonvisualization occurred in five patients. Four were operated on and "all were found to have acute cholecystitis." Thus, five out of the nine cases with proven gallbladder disease showed normal visualization.

Judging by these figures, the technique of Ali et al. does not appear to be very helpful in excluding gallbladder disease. Admittedly, the technique appears capable of differentiating between the acute and the chronic stage of cholecystitis but most surgeons prefer to get such information from a glance at the temperature chart.

Where did their technique go wrong? Again, since this retrospective study does not present any images, we have to look at the figures, and these clearly suggest that Ali et al. tend to overlook cases of cholecystitis. No less than five of their 16 patients with normal visualization were later cholecystectomized. Why were these patients operated on? Not because they had acute pancreatitis. Pancreatitis per se is not an indication for surgery. We must assume that the surgeons eventually chose to ignore Ali et al.'s assertions that the cholescintigram was normal. When first told that visualization was normal, the surgeons of course abstained from operation. Why ask for a scintigram if you intend to operate anyway? Thus, the operation was delayed. When they finally operated, "all five (patients) were found to have... chronic cholecystitis." In view of the delay it is not surprising that the disease had reached its "chronic" stage. Given time, any acute cholecystitis will subside and become "chronic" (3).

As for the 11 nonoperated patients with normal visualization, no one can be certain how many had cholecystitis and how many had not. For the sake of the argument let us assume that Ali et al. are correct when they claim that all 11 patients had normal gallbladders. It is this claim that leads them to conclude that cholescintigraphy is "... as useful... in patients with acute pancreatitis as it is in patients without..." They did not have one single case of nonvisualization in a sample of 11 patients with acute pancreatitis and gallbladders presumed to be normal. But, what about chance? From a table of 95% confidence limits (4) we learn that if a sample of 11 patients does not contain one single case of a
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.

W. VAN DER LINDEN
V. KEMPI
G. EDLUND
Depts. of Surgery and Radiophysics
Ostersund sjukhus
S 831 83 Ostersund, Sweden

REFERENCES
4. MAINLAND D, HERRERA L, SUTCLIFFE MI: Tables for use with Binomial Samples. New York, 1956, p 79

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 cholecintigraphy 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 cholecintigraphy 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 cholecintigraphy is not useful in the evaluation of gallbladder disease in general. On the contrary, the unreliability of cholecintigraphy 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 cholecintigraphy 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 cholecintigrams 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 cholecintigram was normal.” We regard these remarks as ill-considered. Acute cholecystitis is not the only indication for cholecystectomy. The patients with normal cholecintigrams 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 cholecintigram 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 cholecintigrams, we can only point out that the sensitivity of cholecintigraphy 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-