Hepatobiliary Radiopharmaceuticals

Dr. Ronai (*1*) may be premature in dismissing hepatobiliary radiopharmaceuticals from a role in the diagnosis of acute cholecystitis. We have reported the utility of previously administered (i.v.) cholecystokinin (CCK) to facilitate the interpretation of such studies in patients with suspected acute cholecystitis (*2,3*). In 39 patients, we administered 75 units of CCK intravenously 30 min before a hepatobiliary radiopharmaceutical. Ten patients with acute cholecystitis failed to show gallbladder accumulation of radioactivity, reflecting the obstruction of the cystic duct that initiated this disease. All other patients showed gallbladder accumulation, indicating that the cystic duct was patent at the time of the study. Within the limitations, and subject to the precautions that are described in our report, we feel that the procedure is helpful in assessing patients with suspected acute cholecystitis.

Hepatobiliary radiopharmaceuticals, used with CCK or a synthetic analog of CCK, deserve further evaluation and comparison with alternative or complementary diagnostic procedures, some of which Dr. Ronai mentioned in his illuminating editorial.

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REFERENCES

1. RONAI M: Hepatobiliary radiopharmaceuticals: Defining their clinical role will be a galling experience. *J Nucl Med* 18: 488–490, 1977

2. EIKMAN EA, CAMERON JL, COLMAN M, et al: Radioactive tracer techniques in the diagnosis of acute cholecystitis. *J Nucl Med* 14: 393, 1973 (Abst)

3. EIKMAN EA, CAMERON JL, COLMAN M, et al: A test for patency of the cystic duct in acute cholecystitis. *Ann Int Med* 82: 318, 1975

Reply

Thank you for the opportunity to reply to Dr. Eikman's letter. I appreciate his comments and am, of course, familiar with his pioneering work on the use of hepatobiliary radiopharmaceuticals in the diagnosis of acute cholecystitis (see Reference 10 in my editorial).

I am puzzled by the letter, however, because what I thought I said is the reverse of what Dr. Eikman thinks I said. Quoting from the editorial (*1*): "A less hazardous alternative to the IVCC [intravenous (contrast) cholangiogram] is certainly needed and the intravenous (radionuclide) cholangiogram (IVRC) using diethyl IDA may perhaps fill this role. One of the major current uses of the IVCC is the demonstration of patency of the cystic duct in patients with suspected acute cholecystitis. If the contrast medium enters the gallbladder, the cystic duct is patient and acute cholecystitis is most unlikely." Also: "These considerations suggest that diethyl IDA may prove useful in excluding cystic duct obstruction."

I must therefore plead innocent to Dr. Eikman's charge that I am "dismissing hepatobiliary radiopharmaceuticals from a role in the diagnosis of acute cholecystitis." The reverse is in fact the case.

I agree entirely that some maneuver is necessary to avoid false positives due to failure of uptake of radiotracer by the occasional normal gallbladder, and I said as much in the editorial. Dr. Eikman's work with cholecystokinin suggests that these false positives can be eliminated by emptying the gallbladder immediately before the study. On the other hand, the work of Stadalnik et al (see Reference 7 in the editorial) implies the reverse, namely, that false positives occur when the gallbladder is emptied (with a fatty meal) and not when contraction of the gallbladder is avoided (with a fat-free diet). This paradox is at present unresolved; hence, I qualified my prediction that diethyl IDA would be useful in the diagnosis of acute cholecystitis by saying: "Protocols for clinical trials of diethyl IDA should take these observations (i.e., the work of Drs. Eikman and Stadalnik) into account."

Accordingly, sir, I believe that Dr. Eikman and I are in substantial agreement, and I must conclude that Dr. Eikman misunderstood or misread the editorial. It follows, then, that I must not have made my point sufficiently clear—a capital crime among editorial writers. If I am spared the guillotine, I promise to be more explicit next time.

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REFERENCE

1. RONAI PM: Hepatobiliary radiopharmaceuticals: Defining their clinical role will be a galling experience. *J Nucl Med* 18: 488–490, 1977

Tc-99m-Labeled Leukocytes

Recently Bagwe and Sharma (*1*) reported that a high percentage (22–40%) of Tc-99m-labeled autologous rat leukocytes accumulated in bone cortex following intravenous administration of radiolabeled cells. In order to show that this was not due to the accumulation of a cell-free complex, they injected a cell-free mixture of pertechnetate, dextran, dextrose-phosphate buffer, and stannous chloride. Bone uptake decreased to the range of 4–14%, suggesting that much of the accumulated radioactivity indeed was cell-associated. They observed further that if totally non-viable Tc-99m-labeled leukocytes were injected, bone accumulation decreased to 6–11%, indicating that uptake was at least partially dependent upon cell viability.

In our previous studies with Tc-99m as a label for lymphocytes in the mouse (*2–5*) and rabbit (*6*), we failed to consider the possibility of significant bone uptake and were intrigued by Bagwe and Sharma's observations. We therefore decided to duplicate their experiments as closely as possible, employing both the labeling procedure that they described and one that we have used (*3*). The present brief report summarizes the results of these experiments.

Leukocytes from male Fisher rats were isolated from 10 ml of blood obtained by cardiac puncture using the