# Sincalide-Augmented Quantitative Hepatobiliary Scintigraphy (QHBS): Definition of Normal Parameters and Preliminary Relationship Between QHBS and Sphincter of Oddi (SO) Manometry in Patients Suspected of Having SO Dysfunction

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Sphincter of Oddi (SO) dysfunction presents with vague abdominal pain and/or abnormal liver function tests, and is presumably due to SO stenosis or spasm. Clinical, laboratory, and imaging methods of diagnosis have been less than ideal. Initially, we determined normal quantitative hepatobiliary scintigraphy (QHBS) parameters both pre- and post-sincalide administration. Thirty-one "normals" were analyzed, and post-sincalide common bile duct (CBD) dynamics could be satisfactorily determined in 29 (94%) subjects. Normal values at sincalide-augmented QHBS are reported. Next, 10 patients suspected of having SO dysfunction were studied prospectively using SO manometry and QHBS. The two tests were in agreement in seven cases (4: normal CBD dynamics, 3: abnormal). In one case of advanced SO stenosis, QHBS was abnormal, but SO manometry could not be performed. In the two remaining cases, SO manometry and QHBS gave discordant results. Of greatest importance, no significant correlation existed between the quantitative parameters of these two tests. Sincalide-augmented QHBS is possible and may, in the future, be of value in the diagnosis of SO dysfunction and/or partial CBD obstruction.

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Chronic right upper quadrant (RUQ) pain in the face of a normal diagnostic evaluation is a relatively common condition. After gallbladder disease has been excluded as a cause, sphincter of Oddi (SO) dysfunction must be considered in the differential diagnosis (*1–2*). This disorder has been proposed to occur secondary to SO stenosis (due to stone passage or post-inflammatory fibrosis) or spasm (with sphincteric spasm due to a paradoxical response to cholecystokinin (CCK)) (*1–5*). SO dysfunction is a difficult and, as of yet, uncertain diagnosis. Methods used to date for diagnosis have included SO manometry (*1–5*), fatty meal-augmented ultrasonography (*6–8*), and hepatobiliary scintigraphy (*8–11*). To date, these modalities have been applied almost exclusively in patients following cholecystectomy (the post-cholecystectomy syndrome). If SO dysfunction truly exists, it should be possible to diagnose this disorder prior to cholecystectomy. An imaging method of screening patients for SO dysfunction prior to a potentially unnecessary cholecystectomy would be of value. Such a method is also needed to test the physiologic significance of aberrations in SO manometry. As preliminary work toward this goal, we investigated sincalide-augmented, quantitative hepatobiliary scintigraphy (QHBS) in "normals." Our goals were: (1) to determine if common bile duct (CBD) dynamics could be adequately evaluated before and after stimulation of gallbladder emptying with sincalide, and if so, (2) to establish normal parameters with this test for future investigations of suspected SO dysfunction. Our third goal of this preliminary investigation was to define the relationship, if any exists, between the quantitative parameter at QHBS and SO manometry. From a scientific viewpoint, it would be of value to study this relationship in both normals and abnormals. SO manometry is, however, invasive, and not without signifi-

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cant complications, and as such, this goal was designed to compare these two techniques in patients clinically suspected as having SO dysfunction.

## MATERIALS AND METHODS

Sincalide-augmented QHBS was performed in 31 subjects, all of whom were screened for evidence of biliary disease. All subjects were asymptomatic in regard to biliary disease, and screening laboratory tests and ultrasonography were normal. There were 20 males and 11 females (mean age: 43 yr; range: 25–64 yr). Ten additional asymptomatic patients (status-post cholecystectomy) were also studied. Informed consent was obtained from each subject.

Following the bolus injection of 6–8 mCi of $^{99m}$Tc-di-isopropyl-iminodiacetic acid (DISIDA), anterior sequential images of the upper abdomen were performed using either a standard (23 patients) or a large field-of-view (8 patients) gamma camera, equipped with a low energy, all-purpose collimator. Continuous computer acquisition was used at a rate of one frame/min for 90 min. At 60 min after radiotracer administration, each subject was administered 1.5 $\mu$g of sincalide (the eight amino acid, active portion of CCK) as a slow i.v. push injection over 1–2 min (this injection was given to the post-cholecystectomy patients as well.) The importance of remaining motionless during the examination was stressed with each patient.

Regions of interest (ROIs) were placed about the following sites: gallbladder, common bile duct (CBD), liver (using a square ROI over an area of the right lobe, avoiding the major bile ducts), and the entire hepatobiliary region (liver and CBD). (see Figure 1). Two patients were excluded due to failure to adequately identify the CBD. Careful attention was paid to avoid overlap of duodenum with the CBD ROI, and placement was checked by visual inspection of the dynamic study in "movie-mode" with ROIs in place to exclude overlap or significant patient motion. Time-activity curves were generated for each of these ROIs. Time-to-peak activity and half-time of wash-out were calculated for each time-activity curve.

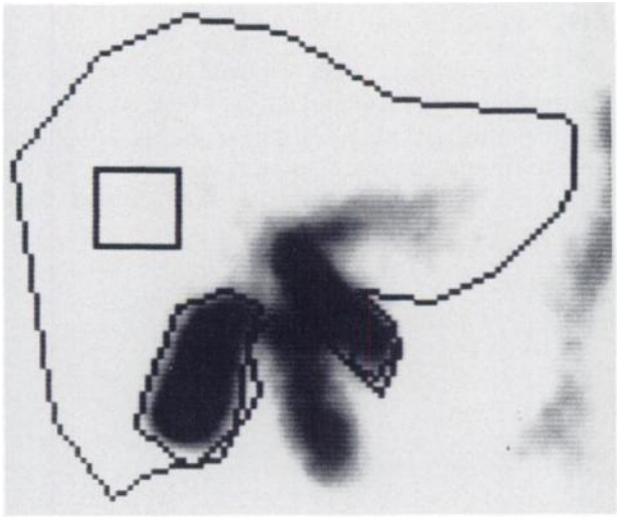


**FIGURE 1**
Illustration of placement of ROIs: hepatobiliary, hepatic, gallbladder, and common bile duct.

**TABLE 1**
Normal QHBS Parameters

| Time to Peak | Mean (min) | Upper limit mean ± 2 s.d. (min) |
|---|---|---|
| Spontaneous | | |
| Liver | 12 | 19 |
| CBD | 24 | 39 |
| Post-Sincalide | | |
| CBD | 4* | 10 |
| Half-time of Wash-out | | |
| Spontaneous | | |
| Liver | 29 | 44 |
| CBD | 33 | 63 |
| Post-Sincalide | | |
| CBD | 10 | 24 |

Post/Pre-sincalide peak CBD ratio: mean = 0.47; range = 0–1.2; mean + 2 s.d. = 1.23.

Gallbladder ejection fraction: mean = 41% and range = 7–80%

* Excludes the seven patients with no post-CCK peak (n = 22).

These parameters were calculated for both pre-sincalide and post-sincalide time periods. Also, the post/pre-sincalide peak CBD ratio was calculated. If no peak in CBD activity occurred after sincalide injection, this ratio was given the value of zero. Gallbladder ejection fractions in response to sincalide also were calculated. All resultant data were analyzed by correlative and linear regression analysis.

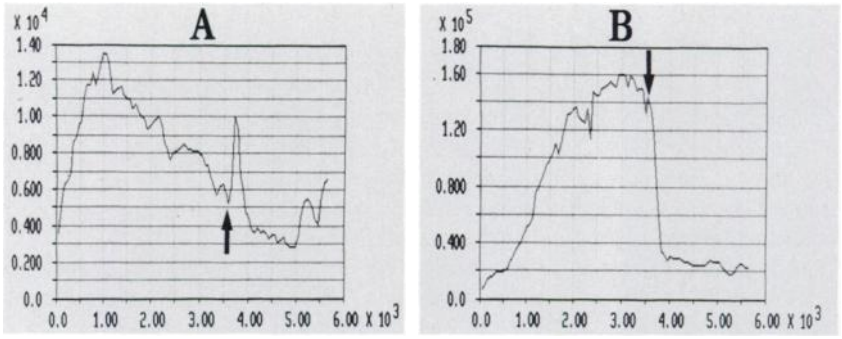
A prospective study of sincalide-augmented QHBS and SO manometry was performed in ten patients with chronic RUQ pain, suspected of SO dysfunction (primarily based on their otherwise uneventful diagnostic evaluations). There were nine females and one male (mean age: 42 yr; range 25–59 yr). Four of these patients had previous cholecystectomies. Informed consent was obtained in each patient. QHBS parameters greater than 2 standard deviations (s.d.s) above the derived normals were considered abnormal. SO manometry was performed within 3 days of QHBS. Standard SO manometric technique was used, as described in the appendix.

Correlative and linear regression analysis was performed between the quantitative parameters of QHBS and SO manometry. If SO manometry was abnormal, the typical course of action was endoscopic sphincterotomy. However, for the purposes of this preliminary investigation, the results of SO manometry were not taken as proof of disease. Instead, clinical course over the next 12 mo, especially in those patients with attempted therapy by sphincterotomy, was used as the main indicator of the significance of the findings at QHBS and SO manometry.

## RESULTS

Of the 31 normal subjects, 29 (94%) had adequate scintigraphic visualization of the CBD for proper quantitation. The other ROIs were easily produced on all scans. Table 1 shows the mean values and upper limits of normal (arbitrarily defined as mean ±2 s.d.s) of the quantitative results in the "normal" population. A typ-

**FIGURE 2**
Common bile duct (CBD) curve (A) shows prompt peak activity occurring at 17 min, spontaneous half-time of washout of 37 min, and prompt "spike" in activity through the CBD in response to sincalide administration (arrow marks time of sincalide injection). The post/pre-sincalide peak CBD ratio is 0.74 (normal). Curve B is the gallbladder time-activity curve, showing marked gallbladder emptying in response to sincalide (85% emptying). (X-axis in all graphs is shown in seconds, y-axis indicates activity)




ical CBD time-activity curve is shown in Figure 2. A clear "spike" in activity is seen in the CBD in response to sincalide. The CBD time-activity curves of seven patients showed no "spike" in activity following sincalide. Since the gallbladder clearly contracted in these cases and CBD activity continued to decline during this period, this pattern was considered a normal variant (see Figure 3). The post/pre-sincalide peak CBD ratio was under 1.0 in 28 of 29 normals, obviously meaning that the spontaneous CBD activity almost always peaked at a higher level than the CBD activity after stimulation of gallbladder contraction. Of interest in the data presented in Table 1 is the fact that gallbladder ejection fraction was relatively variable (mean: 41%; range: 7%–85%). However, there was no significant correlation between gallbladder ejection fraction and any of the CBD quantitative parameters, and as such, the rate of CBD emptying after sincalide administration was not affected by degree of gallbladder contraction.

No significant differences were found between quantitative parameters in the post-cholecystectomy patients versus the normal individuals with gallbladders. No "spike" occurred in CBD activity after sincalide injection in the post-cholecystectomy group, and no change in the rate of CBD wash-out was seen after sincalide administration. Time to peak liver activity was 12 ± 4 min (mean ± 2 s.d.) and 30 ± 14 min for the CBD. Half-time of spontaneous liver washout was 27 ± 16 min, and the half-time of spontaneous CBD washout was 28 ± 23 min.

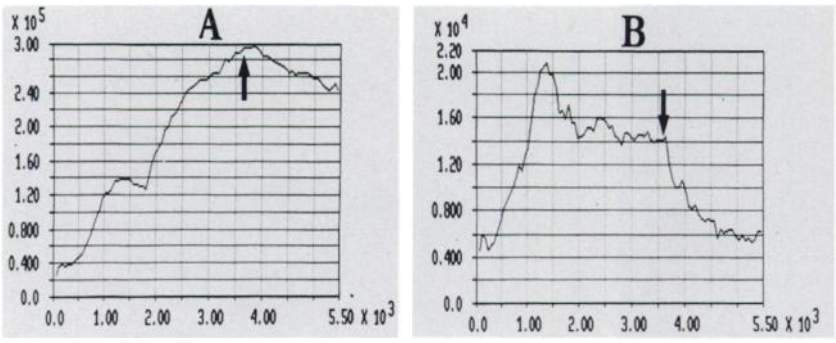
In our evaluation of patients with suspected SO dysfunction, QHBS and SO manometry were normal in four patients. An etiology for these patients' symptoms was not elucidated. One patient had normal QHBS and abnormal manometry and had continued symptoms after sphincterotomy. One patient had abnormal QHBS and normal manometry and did not undergo therapy (symptoms were present at a 12-mo follow-up). Three patients were positive on both tests. One of these patients had an ampullary carcinoma as the cause of her symptoms (see Figure 4). The other two patients had relief of symptoms following sphincterotomy. The final patient had positive QHBS, but unsuccessful manometry, and sphincterotomy caused relief of symptoms.

Table 2 shows the quantitative data for the individual patients. No statistically significant correlation was found between SO manometric parameters and those of QHBS. Figure 5 shows an abnormal CBD time-activity curve from a patient with an intact gallbladder and both QHBS and manometry compatible with SO dysfunction. This curve shows abnormal delays in both spontaneous CBD peak activity and post-sincalide CBD peak, as well as elevation of the post/pre-sincalide peak CBD ratio.

## DISCUSSION

This investigation has shown that QHBS can analyze the dynamics of CBD emptying in response to sincalide. Krishnamurthy et al. (*12*) previously studied biliary kinetics using a similar approach in normals and patients with cholelithiasis, using somewhat different tech-

**FIGURE 3**
Gallbladder time-activity curve (A) showing only 20% emptying after sincalide administration. CBD curve (B) shows abrupt increase in rate of emptying after sincalide. No post-sincalide "spike" in CBD activity occurred. (arrow marks time of sincalide injection). Since no "spike" occurred, the post/pre-sincalide peak CBD ratio is 0 (normal).



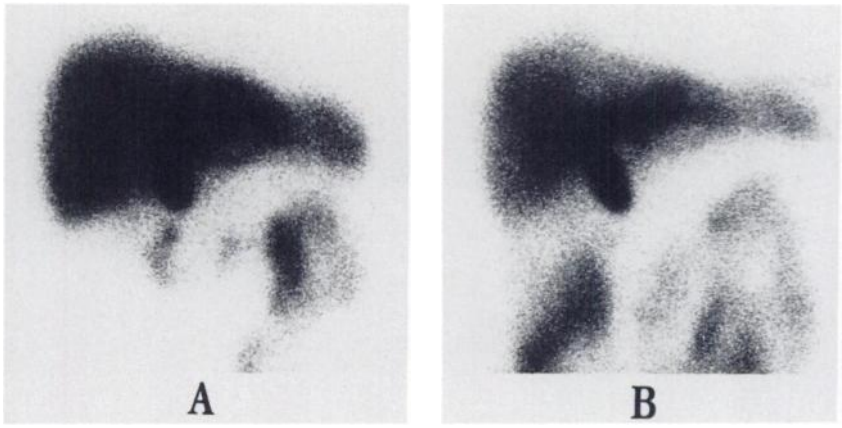



**FIGURE 4**
Sixty-minute (A) and 120-min (B) images from hepatobiliary scan showing normal biliary-bowel transit, but marked prominence of the CBD. The gallbladder has been previously removed. Peak CBD activity occurred at 47 min, with a half-time of wash-out of 82 min (both values abnormal). SO manometry showed a normal basal pressure, but marked increase in frequency of contractions. Ampullary carcinoma was the cause of this pattern.

nical and quantitative parameters. They emphasized the analysis of gallbladder ejection parameters and not the indices of CBD dynamics. In our current study, quantitation of CBD dynamics was possible in 94% of volunteers, in spite of the fact that 74% of the studies were performed on an older standard field of view gamma camera. We have found that identification of the CBD is easier when studies are performed on a modern, large field-of-view gamma camera, with greater spatial resolution. Most patients exhibited a clear-cut "spike" in CBD activity in response to sincalide, with both a rapid rise and fall in activity.

Several interesting findings occurred during definition of these normal parameters. We would have predicted that gallbladder ejection fraction or rate of contraction would have a major effect on CBD dynamics after sincalide. This, however, was not the case, and no significant correlation was found. This finding simply shows that emptying of the CBD is not simply a passive response to gallbladder contracture and SO relaxation. Other factors, such as peristaltic activity of the bowel may play a role in CBD dynamics as well. Simply stated, CBD dynamics are a complex process.

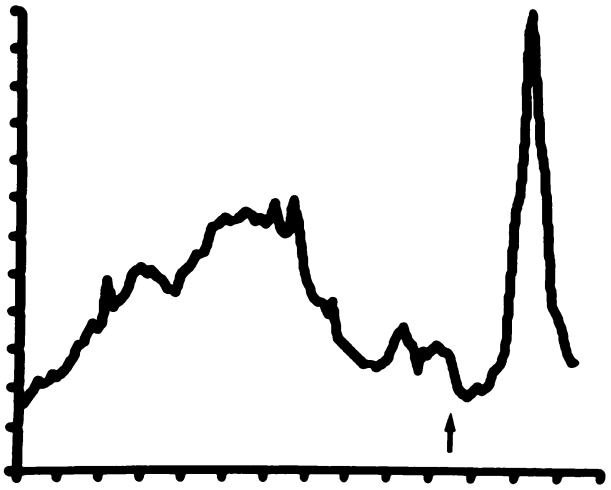


**FIGURE 5**
CBD time-activity curve showing spontaneous peak activity at 46 min (abnormal) with normal spontaneous CBD washout (half time of 10 min). Post-sincalide time to peak is abnormal (13 min) and the post/pre-sincalide peak CBD ratio is 1.71, an elevated value (arrow marks time of sincalide injection). Basal sphincter pressure was 35 mm of Hg, and symptoms were relieved following sphincterotomy. (Each division on the x-axis is ≈ 6 min.)

In our "normal" population, thirteen individuals had gallbladder ejection fractions below 35%, which many investigators have considered abnormal (*13–14*). All these patients were asymptomatic, and as shown by Davis et al. (*15*) and Mesgarzadeh et al. (*16*), low gallbladder ejection fractions can occur in "normals" in response to a single dose of sincalide. Interestingly, the dynamics of the CBD activity in response to sincalide could be analyzed in all these patients with low gallbladder ejection fractions, and their quantitative parameters were not significantly different than those individuals with gallbladder ejection fractions above 35% (see Figure 6). This finding suggests that sincalide-augmented CBD dynamics can be studied in patients with decreased gallbladder ejection fractions, even in the face of potential chronic cholecystitis. However, until further work substantiates this opinion, we recommend that post-sincalide CBD dynamics be viewed with caution in patients with diminished gallbladder ejection fraction (<35%).

The CBD dynamics in patients (status: post-cholecystectomy) were no different from the "normal" individuals. No "spike" occurred after sincalide, since there was no gallbladder to contract. Also, no alteration in CBD wash-out occurred. A paradoxical response to sincalide, as proposed in SO spasm, would presumably delay CBD wash-out, but no such effect was seen in these asymptomatic post-cholecystectomy patients.

SO dysfunction is a difficult clinical diagnosis. SO spasm represents a clinical syndrome, without definitive pathologic findings. Ultrasonography (both with or without fatty meal challenge) (*6–8*), QHBS (*8–11*), and SO manometry (*1–5*) have all been used for possible diagnosis. However, appropriate strategies for diagnos-

**TABLE 2**
QHBS and SO Manometric Findings in Patients Suspected of Having Biliary Dyskinesia

| | Patient No. | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Quantitative Parameters | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
| Hepatic peak | 10 | 10 | 5 | 10 | 9 | 12 | 7 | 3 | **21** | **20** |
| Hepatic $T_{½}$ | 26 | 40 | 41 | 28 | 34 | 31 | 40 | 10 | 38 | **44** |
| Spontaneous CBD peak | 25 | 35 | **46** | **55** | 14 | 27 | 35 | 13 | **59** | **47** |
| Spontaneous CBD $T_{½}$ | 56 | 58 | 10 | 25 | 24 | 28 | 46 | 58 | **295** | **82** |
| Post-sincalide CBD peak | 2 | 5 | **13** | 3 | 7 | 0 | — | — | — | — |
| Post-sincalide CBD $T_{½}$ | 15 | 10 | 3 | 1 | 8 | **33** | — | — | — | — |
| Hepatobiliary peak | 10 | 17 | 60 | 15 | 37 | 60 | 26 | 6 | 28 | 20 |
| GB ejection fraction | 56% | 28% | 62% | 72% | 60% | 24% | — | — | — | — |
| Post/Pre-sincalide peak CBD ratio | 0.83 | 0 | **1.71** | 0.33 | 0.73 | 0 | 0 | — | † | 0.73 |
| Basal SO pressure | 22 | **78** | **35** | 20 | 20 | 28 | 20 | 10 | n.a. | 25 |
| Frequency of contractions | 4.5 | 5 | 4 | 5 | 5 | **8** | 4 | — | n.a. | **10** |
| % retrograde contractions | (All patients had less than 50% retrograde contractions) | | | | | | | | | |
| Paradoxical Sincalide response | (No patient had a paradoxical response to sincalide) | | | | | | | | | |
| Sphincterotomy | No | **Yes** | **Yes** | No | No | **Yes** | No | No | **Yes** | * |
| Relief of symptoms | No | No | Yes | No | No | Yes | No | No | Yes | * |

Positive findings are listed in boldface.
* Patient was found to have ampullary carcinoma.
n.a. = manometry was technically unobtainable.
† No pre-sincalide peak occurred prior to sincalide injection.

tic evaluation of potential SO dysfunction have not been adequately established.

Non-quantitative HBS has been used for the diagnosis of SO stenosis in the "post-cholecystectomy syndrome" (*9–10*). These non-quantitative methods analyzed the "prominence" of the CBD as well as a qualitative evaluation of its rate of emptying. Sensitivities of 80%–90% were reported for the detection of SO stenosis by non-quantitative methods alone, but the patients reported by Zeman et al. (*9*) all had CBD dilatation and probably represented an advanced phase of the spectrum of disease.

Quantitative methods at hepatobiliary scintigraphy have been previously applied to this difficult population as well. Using clinical presentation, instead of SO manometry or blinded results of therapy, for categorization, Shaffer et al. (*11*) studied post-cholecystectomy patients versus normal "controls". They reported the detection of eight of nine patients using QHBS, but could not differentiate SO dysfunction from cholestasis. Darweesh et al. (*8*) studied QHBS in a similar group of patients (32 post-cholecystectomy "controls", 28 patients with suspected partial CBD obstruction). These authors used endoscopic retrograde cholangio-pancreatography (ECRP) and SO manometry as the "gold standard" for disease. They reported a 67% sensitivity and 85% specificity for partial CBD obstruction with QHBS, with hepatic washout at 45 min as the best indicator of dysfunction. The authors also studied fatty-meal sonography (FMS) in this population. They found QHBS and FMS to be complementary, with FMS having a higher specificity.

If a fatty meal (basically a more prolonged CCK response) is needed to detect these abnormalities at ultrasonography, a similar physiologic response should be of benefit at QHBS. We chose sincalide challenge over fatty meal because of its more predictable time of onset of effect, allowing a shorter imaging duration. Sincalide-augmented QHBS should theoretically be more sensitive for partial CBD obstruction of any etiology than standard QHBS. Sincalide-augmented QHBS has not been previously applied to a population of

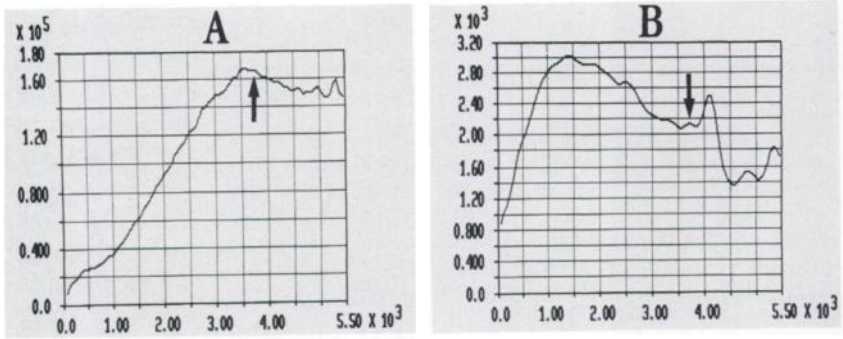



**FIGURE 6**
Gallbladder time-activity curve (A) shows only 12% emptying after sincalide. In spite of this limited gallbladder contraction, a clear "spike" in CBD activity (curve B) is seen. The post/pre-sincalide peak CBD ratio is 0.83 (normal). (Arrow marks time of sincalide injection)

patients suspected of SO dysfunction prior to cholecystectomy. Our current work clearly shows that this technique is possible and potentially adds very valuable physiologic data concerning CBD function.

A number of studies of SO dysfunction have already used SO manometry as the "gold standard" for diagnosis (*1–5*). This is a premature award to this technique. SO manometry has a number of limitations. It is technically difficult to perform and interpret, it has a significant incidence of post-procedural pancreatitis, and it requires a very cooperative patient (*5*). Artifacts occur from patient movement, respiration, retching, and duodenal activity. Overall success rate is only about 70%–80%. A well-controlled, blinded study of SO manometry, FMS, and QHBS has never been performed, using relief of symptoms by sphincterotomy or drug therapy (calcium channel blockers) as the proof of disease. Abnormalities of the esophagus have been overdiagnosed in the past at manometry (*17–18*). A similar problem is clearly possible with SO dysfunction and SO manometry. Care must be maintained to prevent an unquestioned acceptance of SO manometry.

Obviously, our reported patient population is small (n = 10). We feel, however, that this preliminary information is very important. We initially hypothesized that a relationship between gallbladder ejection fraction, post-sincalide CBD dynamics, and SO pressures would exist. However, no significant correlation was found between any of these parameters, simply emphasizing the fact that biliary dynamics are a complex function. The results of this preliminary study are not intended to support the use of QHBS for the diagnosis of SO dysfunction. Instead, we hope to emphasize that little is truly known about this syndrome, that SO manometry and QHBS results are complex and no simple relationship exists between the physiologic parameters that each measures, and that much greater clinical investigation is needed. In this small population, we have found that QHBS can identify atypical biliary dynamics in cases where SO manometry is either normal or cannot be performed, and also, that QHBS may be normal in the face of abnormal SO manometry. Both methods, as well as FMS, have a potential role in the evaluation of SO dysfunction.

A prospective, double-blind study of sphincterotomy versus "sham" procedure in patients with suspected SO dysfunction was recently reported by Geenen et al. (*19*). This study showed that SO manometry could be used to select patients who will improve with sphincterotomy. Patients with elevated sphincter pressures were almost uniformly improved after sphincterotomy, and improvement persisted for 4 yr after treatment. Though extremely encouraging, these results have yet to be replicated. Because of the complexity of SO manometry, its fairly high failure rate, and its invasive nature, a noninvasive substitute would be of value. In the study by Geenen et al. (*19*), 208 of 289 patients with possible SO dysfunction were excluded from further biliary evaluation after ECRP showed no evidence of biliary abnormality and biochemical parameters were normal. Obviously, a large number of patients with chronic abdominal pain remain an enigma. A controlled, double-blinded trial of placebo versus sphincterotomy or drug therapy for the treatment of potential SO dysfunction, using therapeutic relief of symptoms as the "gold standard" for the presence of disease and with careful prospective analysis of the roles of sincalide-augmented QHBS, FMS and SO manometry is still needed.

Sincalide-augmented QHBS has potential use in the evaluation of other etiologies of partial CBD obstruction. The study of CBD kinetics after gallbladder contracture will potentially improve the ability of QHBS to separate partial CBD obstruction from cholestasis. Much greater clinical experience with sincalide-augmented QHBS is needed.

## APPENDIX

SO manometry was performed by the following technique: Manometrics were performed using a 1.7-mm outer diameter, 0.8 mm inner diameter triple lumen catheter connected to a low-compliance pneumohydraulic capillary pump, perfused at 0.25 ml/min. Baseline duodenal pressures were recorded using a standard ECRP catheter (1.7 mm outer diameter, 1.0 mm inner diameter). The manometric catheter was passed into the papilla of Vater until the distal tip was localized. Position of the catheter was documented radiographically without injection of contrast material. The catheter was then withdrawn across the SO using the marked rings on the catheter for reference. All measurements were averaged from a 2-min recording period and expressed relative to intraduodenal pressure which was defined as zero. The following parameters were measured: Basal SO pressure (normal: <35 mm of Hg), peak amplitude of phasic contractions (normal: <350 mm of Hg), frequency of contractions (normal: ≤6 per min), and direction of propagation of contractions (normal: <50% retrograde). The entire procedure was repeated in every patient following the injection of sincalide (0.02 μg/kg i.v.).

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# Editorial
# Quantitative Hepatobiliary Scintigraphy

Suspected partial obstructive sphincter-of-Oddi (SO) dysfunction due to stenosis or dyskinesia is a common diagnostic consideration in patients with unexplained biliary-like upper abdominal pain. Generally, most of these patients have symptoms after a cholecystectomy (postcholecystectomy syndrome). Potentially useful diagnostic tests emphasized during the past decade include SO manometry, fatty-meal sonography, and quantitative hepatobiliary scintigraphy (QHBS).

In this issue, Drane and coworkers describe a study that evaluates QHBS in 31 patients judged to be normal and in 10 patients with suspected SO dysfunction. In their study, most of the controls had an intact gallbladder although the exact percentage is not given. In several previous studies that evaluated quantitative or semiquantitative hepatobiliary scintigraphy in patients with suspected partial common bile duct obstruction, investigation was limited to patients with a cholecystectomy because of the concern that a normal gallbladder might act as a reservoir that obscured the findings of distal common bile duct obstruction (*1–4*). Therefore, the precise utility of the pooled control values given by Drane et al. is not clear. Evidence is not provided to show that normal values for noncholecystectomized and cholecystectomized subjects are comparable.

Another innovative approach by Drane et al. is the use of sincalide in an attempt to develop an augmented QHBS stress test that might unmask abnormalities not shown by standard methods. Regrettably, however, the authors used a small bolus dose of sincalide (1.5 μg i.v.) that would provide only a short-lived stimulus. Because of their short half-life, bolus doses of CCK or sincalide have a biologic effect for only several minutes whereas a sustained effect of 30 min or more is needed for maximal emptying of the gallbladder or to maximize the enterohepatic cycling of bile acids and hepatic bile flow. Thus, the design of this study does not allow any conclusions about the utility of sincalide augmentation for QHBS. One of the rationales for the use of CCK or CCK-like agents during QHBS is the notion that an important type of SO dyskinetic is paradoxical contraction of the SO in response to CCK. This variant of SO dyskinesia, however, is uncommon among SO dyskinesia patients. The most common variant of SO dyskinesia is sphincter spasm

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