

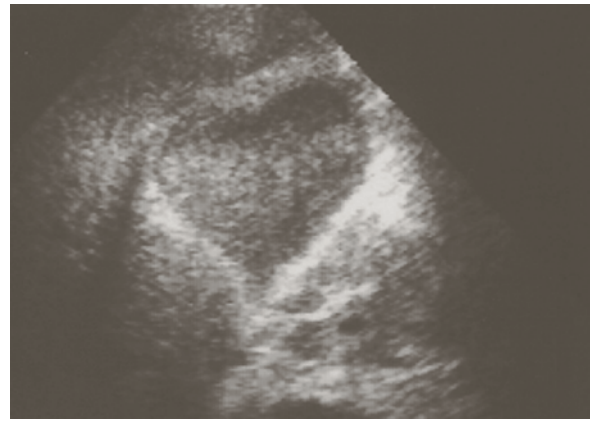
The gallbladder has been assessed using ultrasound since 1951 (see Fig. page viii). Acute acalculous cholecystitis is a traditional complication and a classic target for general ultrasound in the intensive care unit. Time has not modified the opinion we expressed in our previous editions, using histological examination as the “gold standard”. First, this disorder seems to remain exceptional in the medical ICU and affects mostly surgical patients. Second, if ultrasound can accurately describe data, the very interpretation of these data remains subtle. In fact, the gallbladder can show a wide variety of patterns, from the normal to the pathological, in passing even picturesque (Figs. 8.1 and 8.2). A strictly normal gallbladder in the ICU is an infrequent finding (see Fig. 4.9 page 30). The variations in volume, wall thickness, content, shape and surroundings create infinite combinations. Some are variants of the normal, some are pathological but do not require emergency procedures, and others need prompt surgery.

It is timely to describe our mix of experience. Comparing systematic observations from our medical ICU and a surgical ICU with major vascular surgery, we found one case of acute acalculous cholecystitis every 500 days of physician presence for medical patients and 23 days for surgical patients. This means a frequency 20-times lower for medical patients.

A 5-MHz microconvex probe is perfect for this investigation.

### Classical Signs of Acute Acalculous Cholecystitis

Acute acalculous cholecystitis is found in 5–15% of acute cholecystitis and 47% of postoperative cholecystitis [1].



**Fig. 8.1** A picturesque gallbladder. Elegance is not forbidden in an organ as critical as the gallbladder. A simple folding at the hepatic aspect is enough to confer this discrete charm



**Fig. 8.2** In another gallbladder, a very irregular sludge seems to represent a crouched coyote (asymptomatic patient)

The diagnosis is suggested by infectious syndrome and local signs in an exposed patient [2]. Histology alone provides definite diagnosis, a mandatory sign being

wall infiltration by neutrophils. Classically associated ultrasound patterns:

- Size: enlarged gallbladder, with a long axis caliper over 90 mm and a short axis over 50 mm.
- Wall: thickening greater than 3 mm.
- Content: sludge (echoic, compact, dependent sediment).
- Surroundings: perivesicular fluid collection.
- Murphy's sign: pain due to the pressure of the gallbladder. Ultrasound precisely locates the gallbladder, making Murphy's sign more accurate.

Sensitivity of ultrasound is weak (67%) for some [3], high (90–95%) for others [4,5]. When distension, thickening and sludge are combined, sensitivity falls and specificity climbs [2].

### Our Observations of Acute Acalculous Cholecystitis

Acute acalculous cholecystitis seems to be specific to the surgical ICU. It may complicate major trauma or major vascular surgery such as aorta surgery. Although ultrasound can localize the gallbladder and accurately delineate the phenomena described above, we suspect that these signs, taken one after another or even together, should be interpreted. Our observations of

histologically proven acute acalculous cholecystitis have led to the following observations (Fig. 8.3).

**Size:** On average, the gallbladder measured 103 mm on the long axis (range, 65–150 mm) and 40 mm on the short axis (range, 29–55 mm).

**Wall:** The wall was always moderately thickened, measuring on average 4.6 mm (minimum observed, 3.0 mm; maximum, 6.2 mm).

**Content:** Sludge was present in 90% of cases.

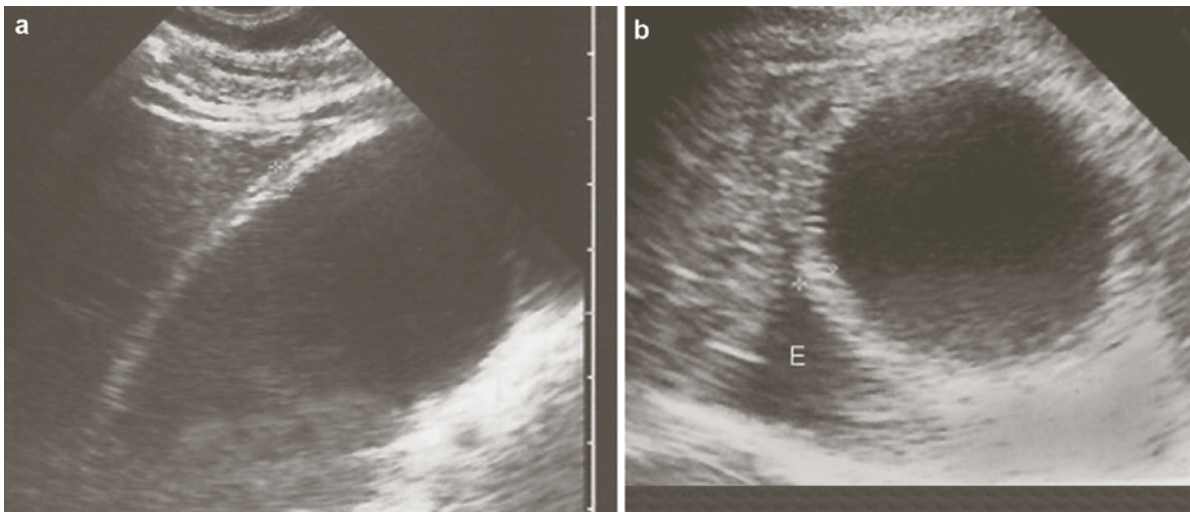
**Surrounding:** We observed selective effusion in 12% of cases.

**Murphy's sign:** We observed a genuine Murphy sign in 8% of cases.

The problem begins when we also consider a disorder encountered in our histology reports: chronic subacute cholecystitis. This frequent disorder will raise serious diagnostic problems.

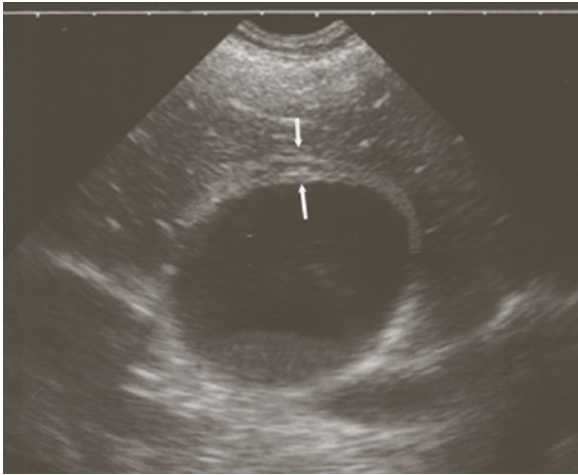
### Chronic Subacute Cholecystitis

This is a histological definition. In fact, neither ultrasound nor even perioperative findings can distinguish it from acute acalculous cholecystitis (Fig. 8.4). Nearly half of our patients operated on for suspicion of acute acalculous cholecystitis had chronic subacute cholecystitis. This disorder does not seem to require surgery.



**Fig. 8.3** Acute acalculous cholecystitis, with histological proof. A homogeneous thickening of the wall (4 mm), a caliper of 30 mm, and dependent sludge are depicted. There was no pain in this

sedated patient, but this gallbladder is suspect because the patient developed fever after major aortic surgery. (a) Axial scan. (b) Short-axis scan, in which a moderate peritoneal effusion is visible (E)



**Fig. 8.4** Cholecystitis. This gallbladder has a homogeneous 5.5-mm thickened wall, a pattern not really different from Fig. 8.3. Sludge is also discretely present. Pathological examination confirmed the diagnosis of chronic subacute cholecystitis

**Table 8.1** Acute acalculous versus chronic subacute cholecystitis

	Acute acalculous cholecystitis	Chronic subacute cholecystitis
Wall thickening	4.6 mm (3.0–6.2)	4.5 mm (3.0–7)
Long axis	103 mm (65–150)	105 mm (84–160)
Short axis	40 mm (29–55)	37 mm (23–56)
Sludge	90%	66%
Localized perivesicular effusion	12%	0
Murphy's ultrasound sign	8%	10%

Extreme values are *in parentheses*

In our observations, the average long axis was 105 mm (range, 84–160 mm), average caliper 37 mm (range, 23–56 mm), average wall thickness 4.5 mm (range, 3.0–7.0 mm), sludge was present in 66% of cases, Murphy's sign in 10%, and localized effusion was never present.

These data are quite similar to those seen in acute acalculous cholecystitis (Table 8.1). One consequence is that this disorder is diagnosed, with subsequent surgery, with the same frequency as acute acalculous cholecystitis. This means useless surgery, which is increased operative risk, but above all, an initial problem that remains undiagnosed. A perioperative pattern is sometimes misleading, and many gallbladders deemed acute or even

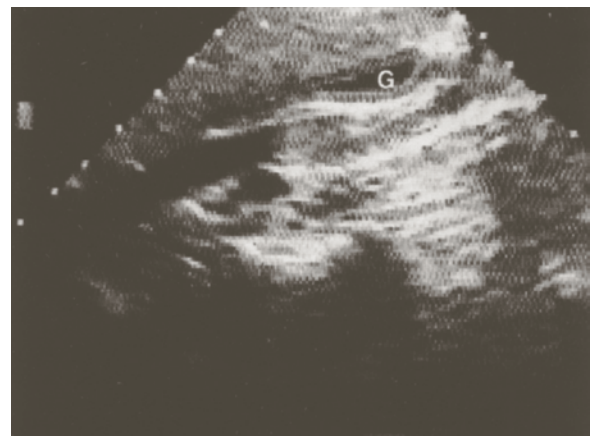
gangrenous become simple chronic subacute cholecystitis once under the microscope.

## Common Gallbladder Patterns Seen in the Intensive Care Unit

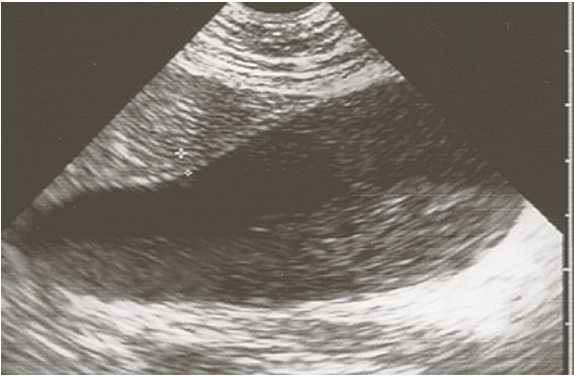
In our critically ill patients with no superimposed clinical problem, the majority of their gallbladders are enlarged and contain sludge. Wall thickening is frequent; the major form of this thickening will be dealt with in a later section. Peritoneal effusion is routine in critically ill patients. All these changes are routine and of little relevance, even when integrated in a suggestive context. Let us examine them in detail.

### Volume

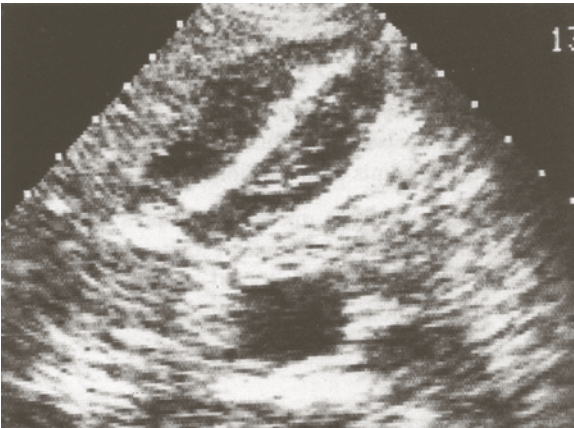
Volume can vary between complete vacuity to distension. Detecting an empty gallbladder requires experience. One should first identify a portal structure, then the right portal branch, which leads to the fossa vesicae felleae, which always leads to the gallbladder space (Fig. 8.5). An empty gallbladder is, in principle, functional, since it is able to contract. It may also be perforated. A distended gallbladder (long axis >90 mm, short axis >50 or 40 mm) is the rule in patients under parenteral feeding and morphines (Fig. 8.6). The lumen can be virtual and the wall thickened (Fig. 8.7). Among other patterns, one can see septate contents, variations



**Fig. 8.5** Empty gallbladder. This discrete image should be recognized to avoid erroneous diagnoses



**Fig. 8.6** Enlarged gallbladder. The volume is  $100 \times 40$  mm, the wall 3.6 mm (slightly thickened), the content 40% sludge. This is frequent in the ICU. However, this gallbladder did not provoke symptoms. This female patient admitted for ARDS (aspiration pneumonia) fully recovered



**Fig. 8.7** The lumen of this gallbladder is virtual, reduced to an echogenic stripe, and an extremely thickened wall, to 12 mm. Laparotomy and pathology revealed simple gallbladder edema in this patient in septic shock with major lung injury

in length, complete calcifications of the wall, or tumors. Images of these anomalies are accessible in abdominal ultrasound textbooks [6, 7].

### Wall Thickening

The normal wall measures between 1.5 and 3 mm. With modern units (i.e., since 1992), the resolution precision allows us to consider 3 mm as a cutoff. The measurement is easy when the wall is outlined between a peritoneal effusion and the bile (Fig. 8.8), but difficult when the wall continues with an isoechoic hepatic



**Fig. 8.8** The wall of this gallbladder is perfectly outlined between bile (G) and ascites. This wall is perfectly fine. This figure easily invalidates the traditional idea that ascites causes gallbladder wall thickening

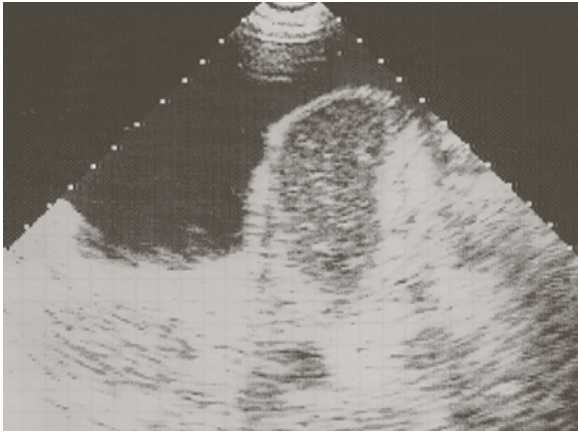
parenchyma, with superimposed edema, which makes any precise measurement illusory.

We routinely find a thickened wall (Fig. 8.6). It can be split, with two echoic layers surrounding an hypoechoic layer. A striated pattern is described as a sign of acute acalculous cholecystitis [8], but the follow-up of our patients does not support this impression.

Traditionally, a thickened wall is nearly equivalent to acute acalculous cholecystitis. Experience shows that this sign has very low specificity. The classic list of causes includes ascites, hepatitis, hypoalbuminemia, and cardiac failure, a rather vague term [9]. Observation shows that, in the case of ascites and in spite of the traditional widespread belief to the contrary, the wall can be perfectly thin (see Fig. 8.8). We regularly observe thin walls in gallbladders surrounded by massive volumes of ascites, proving that peritoneal effusion is not in itself a cause explaining wall thickening. Cardiac failure is an overly vague notion. In contrast, acute right heart failure should certainly be considered a prominent cause. We even speak of “cardiac gallbladder” (see next section).

### Sludge

Sludge is nearly always present in the critically ill patient, since the gallbladder does not work in a physiological way. The pattern can vary greatly, although



**Fig. 8.9** Complete sludge. This gallbladder, floating within massive peritoneal effusion, contains a totally echoic lumen. The patient was asymptomatic

we could not attribute a particular value to each. Sludge can be homogeneous (Fig. 8.6) or heterogeneous, containing hyperechoic dots (microlithiasis possibly may be included in the mass). The interface between the sludge and the anechoic nondependent bile can be regular (Fig. 8.6) or ragged (Fig. 8.2). Sludge can be discrete or massive: in some cases, 100% sludge yields a pattern isoechoic to the liver – a hepatization of the gallbladder, so to speak (Fig. 8.9). Solid knowledge of anatomy is then required to recognize the gallbladder. The sludge may be tumor-shaped. Sludge usually appears during a prolonged stay, but may be present at admission. Eventually, it may completely vanish.

### **Peripheral Peritoneal Effusion**

Peritoneal effusion is frequent in the critically ill patient. Localized effusion in acute cholecystitis is a rare finding.

### **Murphy's Sign in Ultrasound**

Murphy's sign is rarely contributive since critically ill patients are sedated or, if not, they are in shock or encephalopathic. Pain is either absent or diffuse over the entire body.

## **A Distinctive Feature: Major Wall Thickening of the Cardiac Gallbladder**

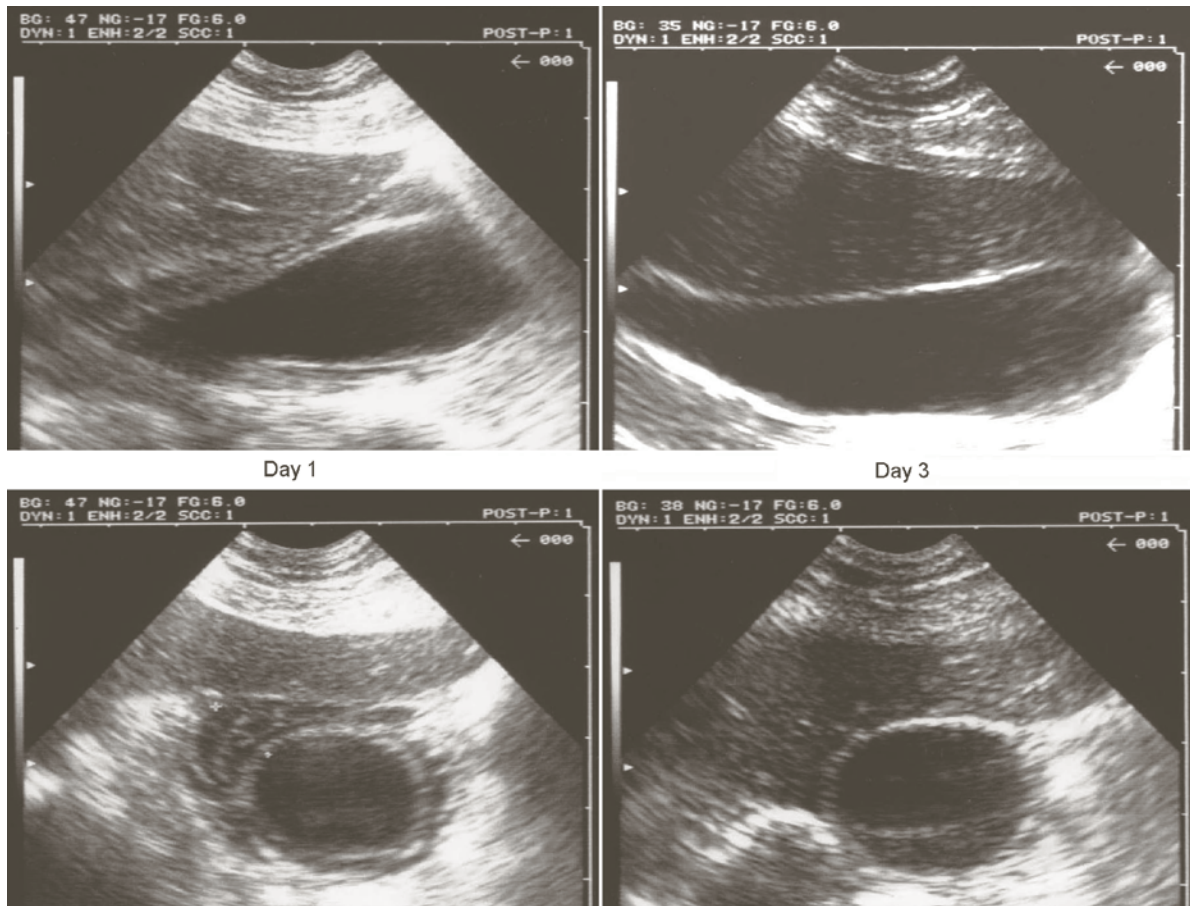
We regularly observe gallbladders with the remarkable feature of major wall thickening, more than 7 mm, up to 18 mm (Fig. 8.10). This pattern always occurs in patients with right heart failure, such as acute asthma, pneumonia, adult respiratory distress syndrome, pulmonary embolism, acute tricuspid regurgitation, and exacerbation of chronic obstructive pulmonary disease, in the most severe forms. This population is more often seen in medical ICUs, hence possibly a higher rate of cases observed here. There is no local sign in these sedated patients. The gallbladder cavity itself is often small, possibly because the walls enlarge to the detriment of the cavity. Time allowing, one can observe the complete regression of this major thickening (Fig. 8.11). A dozen observations among a large number benefited from histologic examination, using laparotomy, for instance. All of these observations were the result of wall edema, sometimes chronic subacute cholecystitis, but never up to now acute acalculous cholecystitis.

We suggest labeling this frequent observation of overly thickened wall the “cardiac gallbladder,” with analogy to cardiac liver or cor pulmonale. It can be assumed that the cardiac gallbladder:

- Is above all the manifestation of congestive phenomena that is observable at the gallbladder wall, which is an accessible area, as retinal vessels are a



**Fig. 8.10** Cardiac gallbladder. The wall of this gallbladder is extremely enlarged, up to 20 mm. The lumen is narrow (max. 12 mm), probably because of the space taken by the walls. This patient has acute right heart failure. Pathology confirmed simple wall edema



**Fig. 8.11** Evolution of a “cardiac gallbladder” in a patient with acute respiratory failure. *Left:* Sections of the gallbladder on admission. *Right:* Same sections 3 days after. Maybe an intermediate analysis would prove an even shorter delay for “recovery”

privileged site to assess general circulatory function.

- Is frequent.
- Can be occult, because this is a transitory feature.

Conversely, an ultrasound examination performed at the climax of the wall thickening can lead to an erroneous diagnosis of acute acalculous cholecystitis, and result in a number of unnecessary laparotomies.

There is a clinical relevance to the recognition of a cardiac gallbladder. Data suggest that the detection of thickening over 7 mm in a patient admitted in a medical ICU with symptoms that may evoke acute acalculous cholecystitis should incite the physician to search for *another cause* to explain the present symptoms (fever, pain). A laparotomy is at risk of being useless if the real cause is not recognized. Frequently, the

gallbladder is removed, and the patient comes from the operating theater with no more fever: this is may simply be the postoperative hypothermia. When the fever recurs again, 1 day later, it is usually interpreted as a new problem (and, usually, the pneumonia that was not visible previously has a radiologic appearance, making the diagnosis of pneumonia easier, among examples).

### How to Improve the Diagnosis of Acute Acalculous Cholecystitis

We believe that ultrasound is an excellent method for localizing and measuring the gallbladder, but not for distinguishing the surgical emergency from insignificant variants of the normal.

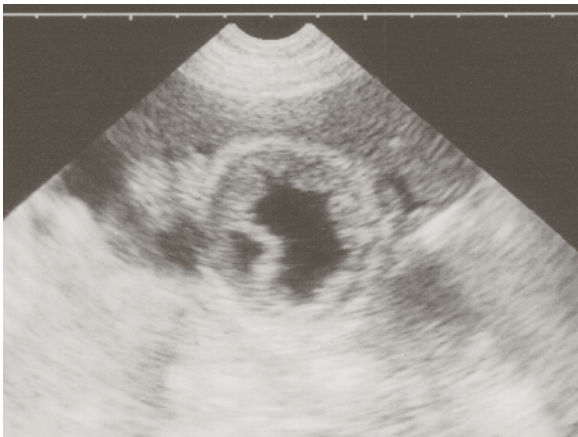
## Patient Background and Current Situation

It seems wise to evoke acute acalculous cholecystitis only in well-defined patients. Major vascular (aorta) surgery occurred in half of our cases, a major trauma in a quarter of cases. As for chronic subacute cholecystitis, major vascular surgery occurred in only 16% of cases, trauma in 33%. Most patients with cardiac gallbladder have an acute right heart failure in the setting of ARDS or multiple organ failure, which is less often severe asthma.

## Considering Certain Ultrasound Signs

We recall that a wall thickening greater than 7 mm in a medical ICU patient suspected of having acute acalculous cholecystitis should prompt a search for another cause explaining the symptoms.

A subtle study showing parietal ulcerations would be valuable, but our investigations are at a standstill. We sometimes see shreds detached from the mucosa (Fig. 8.12), but with pathology ruling out the diagnosis of acute acalculous cholecystitis. Detachment of the mucosa with shreds floating in the lumen is described in the literature as a sign of gangrenous cholecystitis [10].



**Fig. 8.12** The gallbladder of this patient admitted for exacerbation of chronic respiratory disease had an unsettling pattern: a scalloped wall with possible debris detached from the left aspect. Pathology authenticated a simple chronic subacute cholecystitis

Intramural gas should be observed in emphysematous cholecystitis. We did not have the privilege of observing this sign, which is probably rare. Mural gas should give hyperechoic punctiform images, which should not be confused with cholesterol calculi contained in the Rokitansky-Aschoff sinuses, which are part of the picturesque setting of gallbladder adenomyomatosis, although this is of little interest to us here.

**Perforation of the wall.** A thin wall is described in the preperforative cases, but we are still awaiting our first case. Facing this potential rarity, we use to make comprehensive scan of the wall. If the wall thickening is homogeneous, a preperforative state is unlikely.

**Technical note:** a small gallbladder may be normal, or (in theory) the consequence of a perforation.

## Doppler

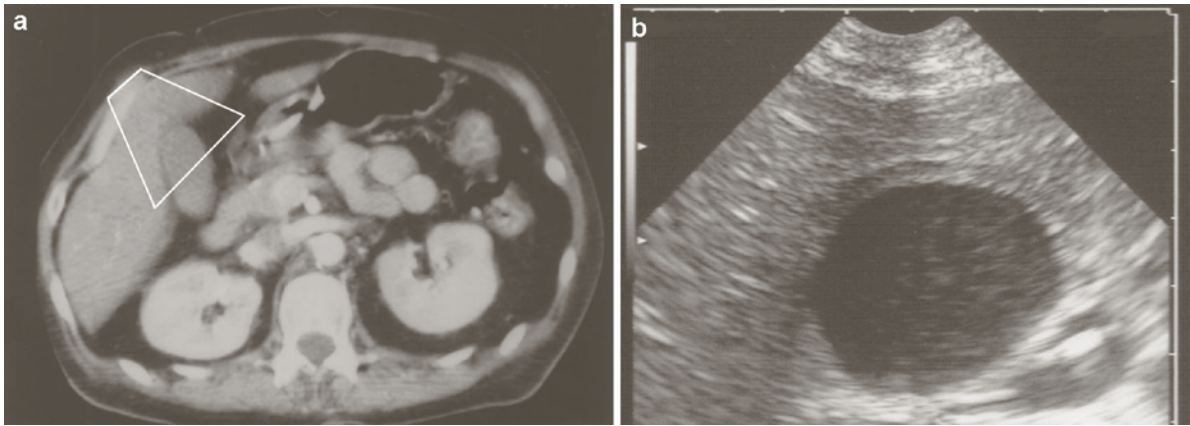
If the Doppler could accurately distinguish between ischemic and edematous wall, it would then be potentially of interest. We await the proof and, above all, the benefit of Doppler. In the supposition of an interest, the degree of emergency disease could give time to use the DIAFORA (Doppler intermittently asked from outside: rare applications) logistics.

## CT

CT does not contribute a great deal, since a careful ultrasound is almost always able to analyze the gallbladder. This is the opportunity to see that ultrasound focal resolution appears superior to that of CT (see Fig. 8.13). The measurement of wall thickening is more accurate using ultrasound [11]. This potential is found at many areas (see Figs 19.1 and 19.2 page 184).

## Dynamic Cerulein Test and Scintigraphy

Dynamic cerulein tests and scintigraphy are of little value [10]. We fear that cerulein, or any other way able to make the gallbladder contract (like simple fatty food), may be harmful in a critically ill patient.



**Fig. 8.13** It is easy to objectify ultrasound's superiority (b) over CT (a) for focal spatial resolution. The area scanned by ultrasound is roughly the one circled in white in the left image. The

gallbladder wall, difficult to measure on CT, is sharply measurable on ultrasound

### Ultrasound-Guided Aspiration of Gallbladder Bile

In our experience, this procedure is simple. A 21-gauge needle is sufficient. The gallbladder should be punctured throughout a nonvascularized area of the liver (the hole will be recovered by the liver). Bile leakage cases described in the literature result from transperitoneal approaches. The dependent bile is aspirated, since the nondependent area may yield false negatives. Since pathological bile is viscous, aspiration must be done patiently. The amount of aspirated bile should be sufficient to diminish the possible hyperpressure and thus limit the (low) risk of leakage. Conversely, if percutaneous drainage is envisaged, the volume of the gallbladder should not be decreased too much. When the needle is withdrawn, manual compression is applied at the point of puncture. If strong compression is not applied, for fear of bile leakage, hemoperitoneum or subcapsular hematoma of the liver can result in patients with impaired hemostasis. Control at 1 and 12 h will search for perivesicular effusion. The vesicular bile of a critically ill patient is usually dark brown or green brown and mildly sticky. The aspirated sludge appears black, like tar.

The risk of vesicular tap is possible though rare. It should be compared with the risk of allowing angiocholitis or cholecystitis to develop, which can be clinically difficult to detect. Of 25 procedures performed as described, we have encountered no complications.

This technique is simple and seems safe. Is it relevant? For some, it is [12], when it provides proof of

infection at the bedside, which should be present in 66% of the cases [13]. Other studies [14] question the sensitivity of this procedure, which is almost always performed on patients under antibiotic therapy. For some, leukocytes found in the gallbladder bile should indicate cholecystitis [14]. The most important limit is that acute acalculous cholecystitis appears more as an ischemic than an infectious process [15]. For paucity of cases at the current time, we lack experience to say whether this procedure is contributive or not.

In our practice, when there is a clear infectious history and clinical suspicion of cholecystitis, in patients



**Fig. 8.14** Acute purulent cholecystitis. Dependent areas (lower part of the image) are not typical of sludge, since they are rather echoic, nor do they evoke calculi, since there is no posterior shadow. Images of membranes seeming to detach from the wall are visible at the upper part of the image. An ultrasound-guided tap immediately confirmed the diagnosis (frank pus) and the patient was promptly sent to the operating room



admitted for shock, especially when there are unusual echoes within the gallbladder (Fig. 8.14), the puncture is envisaged, sometimes withdrawing pure pus, prompting the patient to the operating room.

## Other Pathological Patterns of the Gallbladder

### *Cholecystectomy Space*

Infection of the cholecystectomy space is frequently suspected (Fig. 8.15). Ultrasound-guided aspiration appears to be an accessible procedure and can distinguish pus collection from old sterile blood.

### *Calculous Acute Cholecystitis*

This disorder is rarely of interest to the intensivist. The stone gives a dependent hyperechoic, round image with frank posterior shadow (see Fig. 1.6 page 7). Gallstones are frequently observed. Obviously, the smaller the stones, the more they are able to move and cause trouble. The association of gallstones, thickened wall and Murphy's sign on ultrasound has a positive predictive value of 95%, and the absence of these three signs has a negative predictive value of 98% [16]. Acute calculous cholecystitis rarely raises diagnostic problems.



**Fig. 8.15** Gallbladder space hematoma. Heterogeneous echoic pattern, often found in the gallbladder space after surgical removal

### *Acute “Acalculous” Cholecystitis in Calculous Gallbladder*

Since gallstones are frequent in the general population, how should we label an acute cholecystitis of critically ill patients occurring in a calculous gallbladder?

## Interventional Ultrasound

Diagnostic aspiration has been discussed in “Ultrasound-Guided Aspiration of Gallbladder Bile.”

Percutaneous cholecystostomy is a bedside alternative to surgery [17,18]. Some authors find this procedure easy and rather safe [14,17]. Rates of null mortality and 2–5% morbidity are related [19]. Technical requirements are the same as those described for aspiration [20]. Kits are available, with laterally perforated pigtail catheters, preventing parietal perforation and dislocation of material. The procedure provides a decrease in pressure upstream of an obstacle located in the biliary tract. It was even shown to be effective in sepsis without obvious causes [14]. Other teams mistrust this technique, arguing that a fragile wall can easily be perforated [15]. We add two arguments against this procedure. Since histological proof is unavailable, no conclusion can be drawn from why the situation evolves. Above all, acute acalculous cholecystitis is more an ischemic than an infectious disorder. The gallbladder wall should therefore be removed, more than its content.

From a methodological point of view, in a population with clinical and ultrasound patterns suggestive of acute acalculous cholecystitis, it would be valuable to compare the progression of operated versus non-operated patients. Such a study includes the risk of allowing a genuine acute cholecystitis to evolve [21] and the benefit of avoiding useless laparotomy, i.e., which are ethical issues. Note simply that this methodological detail is absent in published studies [14].

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