

Courvoisier's Gallbladder: Law or Sign?

J. Edward F. Fitzgerald · Matthew J. White ·
Dileep N. Lobo

Published online: 4 February 2009
© Société Internationale de Chirurgie 2009

Abstract

Background Various descriptions of Courvoisier's law, sign, or even gallbladder, this eponymous "law" has been taught to medical students since the publication of Courvoisier's treatise in 1890.

Methods We reviewed Courvoisier's original "law," the modern misconceptions surrounding it, and the contemporary evidence supporting and explaining his observations.

Results Courvoisier never stated a "law" in the context of a jaundiced patient with a palpable gallbladder. He described 187 cases of common bile duct obstruction, observing that gallbladder dilatation seldom occurred with stone obstruction of the bile duct. The classic explanation for Courvoisier's finding is based on the underlying pathologic process. With the presence of gallstones come repeated episodes of infection and subsequent fibrosis of the gallbladder. In the event that a gallstone causes the obstruction, the gallbladder is shrunken owing to fibrosis and is unlikely to be distensible and, hence, palpable. With other causes of obstruction, the gallbladder distends as a result of the back-pressure from obstructed bile flow.

However, recent experiments show that gallbladders are equally distensible in vitro, irrespective of the pathology, suggesting that chronicity of the obstruction is the key. Chronically elevated intraductal pressures are more likely to develop with malignant obstruction owing to the progressive nature of the disease. Gallstones cause obstruction in an intermittent fashion, which is generally not consistent enough to produce such a chronic rise in pressure.

Conclusion We hope that reminding clinicians of Courvoisier's actual observations will reestablish the usefulness of this clinical sign in the way he intended.

Introduction

Various descriptions of Courvoisier's law, Courvoisier's sign, or even Courvoisier's gallbladder, this eponymous "law" has been taught to medical students and surgical trainees since the publication of Courvoisier's famous treatise on the pathology and surgery of the biliary tract in 1890 [1]. As one of the classic clinical signs in medicine, Courvoisier's "law" has stood the tests of both time and modern radiologic imaging remarkably well.

Despite this, the "law" has sadly become confused, misquoted, and discredited in recent years. Does a palpable gallbladder in a jaundiced patient suggest malignant disease? Where is that malignancy located? Is the jaundice painful or pain-free? As with all eponyms, the lack of a clear descriptor in the title has allowed a variety of incorrect mutations to occur, with subsequent rejection of a "law" far-removed from his original observations. Unfortunately, few modern authors have revisited Courvoisier's original text, finding it easier to quote (or misquote) others and in doing so perpetuate these errors.

J. E. F. Fitzgerald · D. N. Lobo (✉)
Division of Gastrointestinal Surgery, Nottingham Digestive
Diseases Centre Biomedical Research Unit, Nottingham
University Hospitals, Queen's Medical Centre,
Nottingham NG7 2UH, UK
e-mail: Dileep.Lobo@nottingham.ac.uk

J. E. F. Fitzgerald
Medical Education Unit, University of Nottingham Medical
School, Nottingham NG7 2UH, UK

M. J. White
University of Nottingham Medical School, Nottingham
NG7 2UH, UK

In this article we review Courvoisier’s original “law” and the modern misconceptions surrounding it. We also discuss the contemporary clinical evidence supporting and explaining his observations.

Courvoisier and his “law”

Ludwig Georg Courvoisier (1843–1918) (Fig. 1) was born in Basel, Switzerland in 1843. After graduation from medical school at the local university, he learned his future craft under the tutorship of Czerny and Billroth in Vienna and Spencer Wells and William Ferguson in London. On returning to Switzerland, his early career was spent in a small hospital in Riehen, where he pursued his special interest in surgery of the biliary tract. After 12 years there he was appointed Professor of Surgery at Basel and went on to become one of the leading surgical figures of his time [2]. Among many notable achievements, he is credited with being the first to perform a choledocolithotomy successfully and for developing the early operative techniques for cholecystectomy. In addition to his surgical expertise he was a respected entomologist and botanist [3].

Many will be surprised to learn that Courvoisier never stated a “law” in the context of a jaundiced patient with a palpable gallbladder. He described 187 cases of common bile duct obstruction, making the observation that



Fig. 1 Ludwig Courvoisier. (Courtesy of Wellcome Library, London)

Table 1 Original German text from Courvoisier’s *Casuistisch-statistische Beiträge zur Pathologie und Chirurgie der Gallenwege* with the English translation

Theile ich sämmtliche für diese Untersuchung verwendbaren 187 Fälle in 2 Hauptgruppen, so erhalte ich 87 Steinobstructionen, 100 andre Obstructionen. Wegen ihrer annähernd gleichen numerischen Stärke lassen sich beide gut vergleichen! Nun collidiren mit den Steinobstructionen weitaus am häufigsten die Gallenblasenatrophien (70:87 Fällen = 80,4%), viel seltener die Ectasien (17:87 = 19,6%). Die andern Verschlüsse dagegen treffen viel seltener mit Atrophie (8:100), viel häufiger mit Ectasie (92:100) zusammen. Oder anders ausgedrückt: Bei Steinobstruction des Choledochus ist Ectasie der Gallenblase selten; das Organ ist vorher schon gewöhnlich geschrumpft. Bei Obstruction anderer Art ist dagegen Ectasie das Gewöhnliche; Atrophie besteht nur in 1/12 dieser Fälle [1]

[In a collection of 187 cases, two main groups were usable for this investigation: 87 stone obstructions and 100 other obstructions. Because of their approximately same numerical strength, they can be compared well. Gallbladder atrophy with stone obstruction (70:87 cases = 80.4%) was more common than the rare ectasia (17:87 = 19.6%). With other causes of obstruction to the choledochus, it is rarer to see atrophy (8:100) than ectasia (92:100). Or differently expressed, with stone obstruction of the choledochus, ectasia of the gallbladder is rare; usually, the organ has already shrunk. Ectasia is usual with obstruction of other kinds; atrophy exists in only 1 of 12 of these cases]

gallbladder dilatation seldom occurred with stone obstruction of the bile duct. The original German text of this observation, along with our contemporary translation into English, is shown in Table 1. It is unclear when this observation became a “law”; and, as with many laws in medicine, this status has resulted in much recent criticism due to numerous exceptions to the rule.

On returning to his original text, another important misconception can be corrected. For many clinicians and authors, Courvoisier’s law equates to malignancy, yet no mention is made to the underlying causes of a palpable gallbladder in the presence of an obstructed bile duct—rather, that stone obstruction is less likely. Courvoisier simply referred to “other causes” without specifying diagnoses; he noted that despite this finding atrophy still occurs in 1 of every 12 cases not due to stone obstruction. However, later in the text, Courvoisier [1] stated, “In my surgical patients I found 35 cases with obstruction to the choledochus; 17 had stones, 18 had strictures or compression by tumours. Of the 17 with stones, only 4 had ectasia of the gallbladder, compared with 16 of the 18 with other causes. Of the 20 ectasias only 4 had stone obstruction; 16 were due to other causes.”

The latter point is important for clearing the confusion surrounding his “law.” Modern literature is full of misquotations, some examples of which are listed in Table 2. However, reviewing historical publications reveals that originally Courvoisier’s observations were not always misrepresented, as the quotation from 1957 indicates [4]:

Table 2 Examples of erroneous definitions of Courvoisier's law in current textbooks

- "Malignant biliary stricture ... If malignant obstruction is below the level of the cystic duct, the gallbladder is distended and may be palpable (Courvoisier's law)."
- Summerfield JA (2004) Diseases of the gallbladder and biliary tree. In: Warrell DA, Cox TM, Firth JD, et al, editors. *Oxford Textbook of Medicine*. Vol 2. 4th edition. Oxford University Press, Oxford, p 707
- "Ludwig Courvoisier (1843–1918) of Basel recognised the reservoir effect of the gallbladder leading to massive distension in the setting of distal malignant obstruction, as in pancreatic cancer. We know this clinical feature of a palpable gallbladder in malignant biliary obstruction as the Courvoisier's sign."
- Norton JA, Bollinger RR, Chang AE, et al (2003) *Essential Practice of Surgery: Basic Science and Clinical Evidence*. Springer Verlag, New York, p 404
- "Hepatomegaly and/or jaundice are found in the majority of patients at presentation, and co-existence of a palpable distended gallbladder suggests presence of a carcinoma at the lower end of the bile duct (Courvoisier's sign). This is however an unreliable sign as it can also occur with cholelithiasis."
- Souhami RL, Moxham J, editors (2003) *Textbook of Medicine*. 4th edition. Elsevier, London, p 832
- "A palpable non-tender gallbladder in a jaundiced patient suggests neoplastic obstruction of the common duct (Courvoisier's sign), most often due to pancreatic cancer...."
- Way LM, Doherty GM (2003) *Current Surgical Diagnosis & Treatment*. 11th edition. Lange Medical Books, New York, p 644

"When the common bile duct is obstructed by a stone, dilatation of the gallbladder is rare ... when the duct is obstructed in some other way, dilatation is frequent."

Furthermore, no indication of the site of the obstruction in the bile duct (proximal or distal to the site of insertion of the cystic duct) is specified in these original observations, nor is there a comment on whether this is painful or painless.

Verghese et al. [2] wisely suggested renaming this clinical finding "Courvoisier's gallbladder" in light of exceptions to the "law." In this way, a palpable, distended gallbladder can be interpreted in the context of the underlying pathophysiology and the clinical picture surrounding it, without blindly following a "law."

Clinical correlates

Although not described as such by Courvoisier, the popular view that jaundice and a palpable gallbladder equate to an underlying neoplasm is supported by a trend reported in the literature. Notable exceptions [5–8], listed in Table 3, mean that this trend falls well short of forming a "law."

Subsequent observational studies have broadly supported the trend described by Courvoisier, although figures vary according to whether the distended gallbladder is detected at

Table 3 Exceptions to Courvoisier's law

Calculous obstruction in Hartmann's pouch
Chronic pancreatitis
Autoimmune pancreatitis [5]
Parasitic biliary obstruction (e.g., <i>Ascaris</i>) [6]
Acquired immunodeficiency syndrome (AIDS)-associated cholangiopathy [7]
Congenital choledochal cysts [8]
Common hepatic duct obstruction—proximal to the insertion of the cystic duct (e.g., lymph nodes at the porta or hilar cholangiocarcinoma)
Double pathology: distal malignancy in the presence of a fibrosed gallbladder Xanthogranulomatous cholecystitis

clinical examination, radiologic imaging, or subsequent surgery. One retrospective review found that 41.9% of jaundiced patients with common bile duct stones had an enlarged gallbladder at subsequent surgery compared to 80% of patients with a malignant obstruction [9]. A more recently reported series showed that 83% of palpable gallbladders resulted from distal tumors, with 15% due to stone obstruction of the distal common bile duct [10].

During an era where computed tomography (CT) is readily available and used routinely in the characterization of abdominal masses, the latter paper [10] also emphasized the importance of a CT-based Courvoisier's sign. Clinical examination identified only 53% of the distended gallbladders, whereas CT identified 87% prior to surgery. Regardless of the point at which it is identified, the finding carries the same significance. Typical CT appearances of such a finding are shown in Fig. 2, and examples of exceptions to the rule are demonstrated in Figs. 3 and 4.

When forming a differential diagnosis in the presence of obstructive jaundice, it is important to remember that the converse of Courvoisier's law is not true—the risk of malignancy cannot be excluded in the absence of a palpable gallbladder. In theory, obstruction proximal to the level of the insertion of cystic duct reduces flow of bile distal to it, thereby reducing biliary pressure in the gallbladder. Hence, the gallbladder may not be distended. However, the absence of a palpable gallbladder can still be useful when estimating the level of obstruction.

Etiology and pathophysiology

The classic explanation for Courvoisier's finding is based on the underlying pathologic process. With the presence of gallstones come repeated episodes of infection and subsequent fibrosis of the gallbladder. In the event that a gallstone causes the obstruction, the gallbladder is shrunken owing to fibrosis and is unlikely to be distensible and, hence, palpable. With other causes of obstruction, the

Fig. 2 Contrast-enhanced abdominal computed tomography (CT) scans of a patient with jaundice due to a distal cholangiocarcinoma. The intrahepatic ducts are dilated (arrows), and there is no mass lesion in the pancreas. The bile duct is dilated (arrowhead), and the distended gallbladder (GB) extends well beyond the inferior edge of the liver (L)

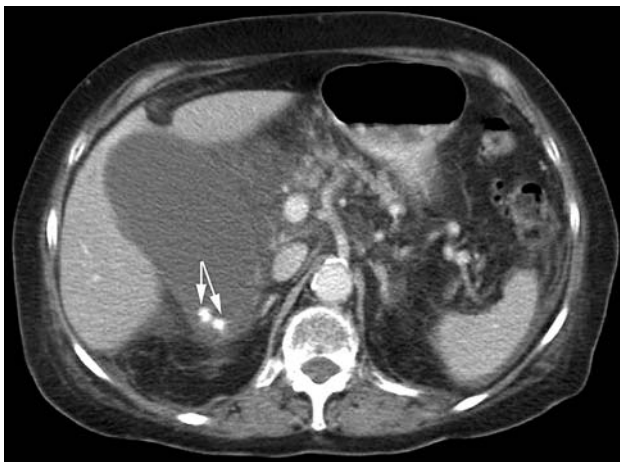
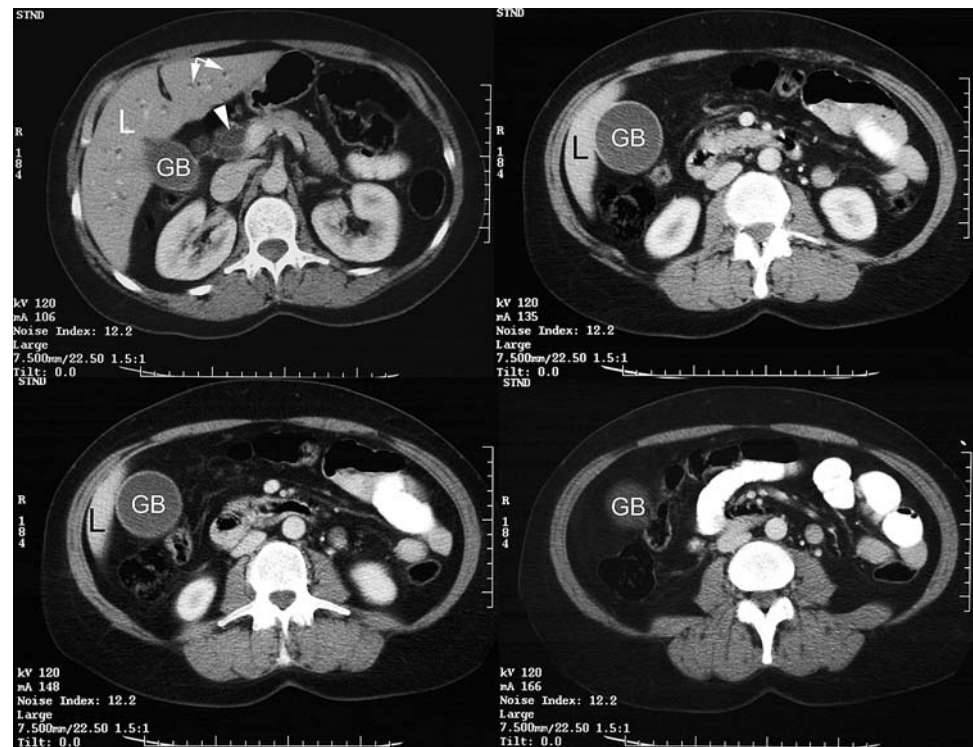


Fig. 3 Example of an exception to the rule. This patient had jaundice due to a stone obstructing the bile duct. However, the gallbladder was distended and palpable because of stones in Hartmann's pouch (arrows) obstructing gallbladder outflow

gallbladder is distended as a result of the back-pressure from obstructed bile flow. Courvoisier [1] himself described these changes as follows:

“The explanation of this difference does not seem to be difficult. According to former diagnostic representations the stones in the choledochus originate from the gallbladder. On their way the stones irritate the cystic duct as well as the gallbladder. This irritation causes a chronic inflammation which can lead to wall changes. The chronic changes can lead to a

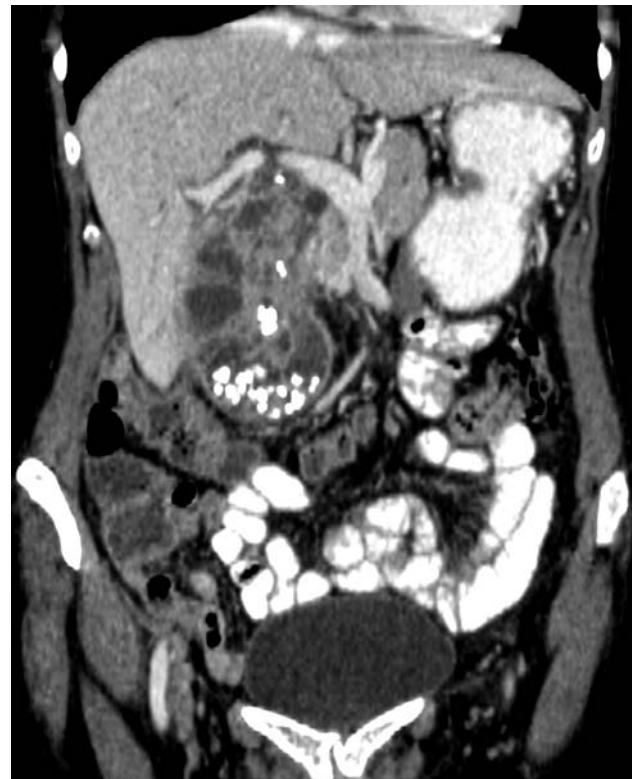


Fig. 4 Coronal reconstruction of an abdominal CT scan in a 65-year-old woman with a right upper quadrant mass demonstrated calcified gallstones and an enhancing soft tissue mass in the gallbladder. Calcification was noted in this mass, with enhancing radial septae extending to the gallbladder wall. Histologic examination of the resected gallbladder showed xanthogranulomatous cholecystitis

shrinking of the duct and of the gallbladder. If the wall of the gallbladder is altered, the strongest bilious congestion will not be able to enlarge them, indeed. The other obstructions, especially those due to tumour compression show a normal collection of the bile and a flexible gallbladder (able to enlarge on pressure)!”

This original explanation has been rejected by those who believe malignant obstruction cannot give rise to such dilation of the gallbladder [11]. The counter argument, seemingly without experimental evidence, is that the gallbladder is not capable of such acute distension and that resultant back-pressure on the hepatic system would cause liver failure long before the organ became palpable.

However, subsequent research has established that the underlying trend observed by Courvoisier remains true, although the original explanation may not be. Chung examined a series of 41 patients to correlate the degree of biochemical jaundice with common bile duct pressure at operation [12]. Comparing chronic calculous cholecystitis with and without common duct obstruction, periaampullary malignant obstruction, and normal patients, the results were unexpected. In vitro, gallbladders were equally pliable in all patients; however, the ductal pressures were markedly higher in those with dilated gallbladders. This suggests that the chronicity of the obstruction is the key. Chronically elevated intraductal pressures are more likely to develop with malignant obstruction owing to the gradual, progressive nature of the disease. Gallstones, although also able to cause obstruction, do so in an intermittent fashion, generally not consistent enough to produce such a chronic rise in pressure. The progressive, initially incomplete nature of malignant obstruction ensures that pressures build gradually without precipitating liver failure.

This plausible theory also fits well with an interesting finding reported by Munzer [10]. Here the phenomenon of a “fading” gallbladder is described, where a clinically palpable gallbladder disappeared on repeated examination. This occurred in 20% of cases, only to reappear again in three-fourths of them. This finding is attributed to squeezing of the gallbladder, overcoming an incomplete obstruction and milking bile out of the gallbladder, only for it to reaccumulate later.

One exception to the hydrostatic back-pressure theory is reported in the literature. Several cases have been described in which there is presence of a palpable gallbladder and obstructive jaundice, but in which gallbladder obstruction arises from a carcinoma in the ampullary region, together with common hepatic duct obstruction due to the carcinoma extending through the tissue in Calot’s triangle. In these instances hydropic dilation of the gall bladder was noted in the absence of bile within it. Although adhering to

Courvoisier’s original observations, the clinical findings are the result of hydrostatic obstruction at two independent sites [13].

Chen et al. [14] evaluated the usefulness of Courvoisier’s law as applied to ultrasonography, comparing 24 patients with malignant obstruction, 50 with calculous obstruction, and 50 normal patients. Gallbladder volume was found to be similar in patients with both malignant and nonmalignant obstruction (93.0 ± 11.3 vs. 86.7 ± 8.5 ml). They also found a linear relation between gallbladder volume and serum bilirubin in patients with malignant biliary obstruction and patients with calculous obstruction without gallbladder stones. These results indicate that gallbladder size is dependent on the degree of obstruction when the gallbladder is not affected by fibrosis (i.e., gallstones not present in the gallbladder). Their findings highlight that it may not always be a malignancy that gives rise to the dilation; yet Courvoisier made no claim that it was.

Conclusions

Ludwig Courvoisier made his astute observations during an era when there was no access to ultrasonography, CT, endoscopic retrograde cholangiopancreatography or magnetic resonance cholangiopancreatography. There is no doubt that over the years his observations have been misinterpreted, and subsequent criticisms are no longer based on his original statements. Courvoisier himself did not seek to establish a “law,” and statistically his findings do not stand up to such a title. Although some authors have tried in vain to set the record right [15–17], many articles and textbooks continue to perpetuate a fallacy. Many would believe this misnomer adds credence to the current arguments for abolishing eponyms in medicine [18]. However, used correctly, Courvoisier’s “law” is a useful guide for establishing a differential diagnosis, and it recognizes the contribution of Courvoisier to biliary surgery.

At approximately 119 years after Ludwig Courvoisier’s treatise was published, we hope that reminding clinicians of his actual observations will reestablish the usefulness of this clinical sign in the way he intended.

Acknowledgments The authors thank Dr. Zeno Stanga for his help in translating the original German text of Courvoisier’s *Casuistisch-statistische Beiträge zur Pathologie und Chirurgie der Gallenwege*.

References

1. Courvoisier LG (1890) *Casuistisch-statistische Beiträge zur Pathologie und Chirurgie der Gallenwege*. Verlag von F.C.W. Vogel, Leipzig

2. Verghese A, Dison C, Berk SL (1987) Courvoisier's "law"—an eponym in evolution. *Am J Gastroenterol* 82:248–250
3. Ludwig Courvoisier (1843–1918). Courvoisier's sign. *JAMA* 204: 627 (1968)
4. Rhoads JE (1957) Pancreas. In: Allen JG, Harkins HN, Moyer CA, Rhoads JE (eds) *Surgery: principles and practice*. Pitman Medical Publishing, London, pp 715–740
5. Ooi K, Merrett N (2004) Autoimmune pancreatitis in a patient presenting with obstructive jaundice and pancreatic mass. *HPB (Oxford)* 6:126–127
6. Lim JH, Kim SY, Park CM (2007) Parasitic diseases of the biliary tract. *AJR Am J Roentgenol* 188:1596–1603
7. Fournier AM, Michel J (1992) Courvoisier's sign revisited: two patients with palpable gallbladder. *South Med J* 85:548–550
8. Lapointe R, Gamache A, Pare P (1984) Bile-duct cyst with cyst-lithiasis: a case report. *Can J Surg* 27:271–273
9. Viteri AL (1980) Courvoisier's law and evaluation of the jaundiced patient. *Tex Med* 76:60–61
10. Munzer D (1999) Assessment of Courvoisier's law. *Saudi J Gastroenterol* 5:106–112
11. Watts G (1985) Courvoisier's law. *Lancet* 2:1293–1294
12. Chung RS (1983) Pathogenesis of the "Courvoisier gallbladder". *Dig Dis Sci* 28:33–38
13. Ginzburg L, Payson BA (1957) A variant of the Courvoisier syndrome in carcinoma of the gallbladder. *Ann Surg* 146:976–982
14. Chen JJ, Changchien CS, Tai DI et al (1994) Gallbladder volume in patients with common hepatic duct dilatation: an evaluation of Courvoisier's sign using ultrasonography. *Scand J Gastroenterol* 29:284–288
15. Bromley PJ, Keller FS (2001) Images in clinical medicine: Courvoisier's gallbladder. *N Engl J Med* 345:1542
16. Verghese A, Berk SL (1986) Courvoisier's law. *Lancet* 1:99
17. Schneiderman H (2004) It's not the law. *Can Med Assoc J* 171: 312
18. Woywodt A, Matteson E (2007) Should eponyms be abandoned? Yes. *BMJ* 335:424