Editorial

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Acalculous Biliary Pain: Motility Dysfunction and Functional Pain

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Most patients with gallstones experience a satisfactory outcome with relief of symptoms following surgery. Such a high response may in part be attributable to high patient expectations that their symptoms will worsen without surgery, their assumption being that surgery will provide a cure. Presumably this biliary-type pain originates either from obstruction of the gallbladder contracting against a fixed or functional obstruction at the cystic duct, or at the level of the sphincter of Oddi (SO), or from the inflammation that result [1]. The basis of such pain is less clear if gallbladder smooth muscle contractility is defective, as occurs in cholesterol gallstone disease. Although biliary pain heralds cholelithiasis and cholecystitis, most (80%) people harboring gallstones never experience pain. Gallstones and abdominal pain are thus not necessarily synonymous. It is, therefore, not surprising that chronic abdominal pain persists in up to 50% following cholecystectomy [2]. Many of these complaints are nonspecific, but 14% have true biliary pain attributable to a definable cause, eg, SO dysfunction. The figures for surgical failures might be higher with the advent of laparoscopic cholecystectomy, which has dramatically increased the rate of surgeries over the past decade. Predictors of this 'post cholecystectomy syndrome' include psychological vulnerability, chronic symptoms before cholecystectomy and pain six weeks after cholecystectomy [3].

The situation becomes even less clear when no structural abnormality is evident. Such 'functional' biliary type pain requires careful investigation to eliminate all structural abnormalities, particularly very small gallstones, as the basis for recurrent biliary-type pain [4]. This includes repeating Trans-abdominal ultrasound, which detects stones larger than 3 to 5 mm in diameter, performing an endoscopic ultrasound for tiny stones smaller than 3 mm in diameter and using accurate microscopy of gallbladder bile to detect microlithiasis. Although it is best to centrifuge bile immediately and use microscopy to detect cholesterol microcrystals and/or bilirubin granules, if a delay is unavoidable, bile can be frozen for later analysis. The cholecystokinin (CCK)-provocation test can cause pain in some normal individuals, depending on the rate of CCK infusion [5]. It does not, however, predict a symptomatic benefit from cholecystectomy and should be abandoned when evaluating acalculous biliary pain. More accurate is the quantitative measurement of gallbladder emptying under controlled conditions with a slow infusion of CCK – CCK cholescintigraphy. In well-defined patients with acalculous gallbladder disease and low ejection fractions on CCK ultrasonography, 59% to 75% continue to have symptoms. Conversely, some 90% experience complete or substantial relief following cholecystectomy [6].

Here, impaired gallbladder emptying appears to be a marker of acalculous biliary pain. The basis for such impaired gallbladder emptying is a defect in the contractile machinery. Again, the dilemma is, what causes the pain? The biliary tract is a low-pressure conduit in which the gallbladder acts as a reservoir to decompress and regulate its pressure. Bile ducts lack a smooth muscle layer, leaving the SO a prime suspect, especially when pain persists after cholecystectomy. The sphincter may be stenotic or exhibit dyskinesia. The diagnostic criteria outlined by the Rome II consensus conference include the gold standard test, SO manometry. This invasive procedure is best reserved for those with objective evidence of intermittent biliary obstruction. Patients suspected of having SO manometry should undergo a preliminary evaluation – a nuclear medicine scan that quantifies the transit time from the hepatic hilus to the duodenum. Morphine provocation appears to increase its sensitivity [7]. Those with SO stenosis have sufficient criteria for

a fixed organic basis and benefit from going directly to sphincterotomy. All functional biliary pain, however, might not relate to motility abnormalities of the gallbladder or the SO. Abnormal manometry of the sphincter may reflect a mere generalized motility disorder of the gut, perhaps involving the duodenum and jejunum. Conversely, visceral hypersensitivity, a potential basis for pain in functional gastrointestinal disorders, represents an abnormal sensitivity to a relatively innocuous stimulus [8,9]. The basis for this could be modified receptor sensitivity at the level of the biliary tract or adjacent viscus, neuronal hyperexcitability in the dorsal horn of the spinal cord and/or altered central modulation of sensory inputs. Such biliary-type pain may even be part of the irritable bowel syndrome. Thus, acalculous biliary pain means no obvious organic basis, but 'functional' is giving way to dysfunction, be it motor or sensory. Further, understanding the basis for the pain implies a potential for medical therapy, drugs acting specifically on the rich neural connections between the gallbladder, SO and the adjacent stomach and duodenum. Functional pain now has meaning [10].

References

1. Rankin J Cerebral vascular accidents in patients over the age of 60: II. Prognosis. *Scott Med J.* 2, 200–215 (1957).
2. Quinn TJ, Dawson J, Walters MR *et al.* Functional outcome measures in contemporary stroke trials. *Int J Stroke.* 4, 200–205 (2009).
3. Lees KR, Bath PMW, Schellinger PD *et al.* Contemporary outcome measures in acute stroke research: choice of primary outcome measure. *Stroke.* 43, 1163–1170 (2012).
4. Sulter G, Steen C, Keyser JD *et al.* Use of the Barthel index and modified Rankin scale in acute stroke trials. *Stroke.* 30, 1538–1541 (1999).
5. Grotta J Why do all drugs work in animals but none in stroke patients? 2 Neuroprotective therapy. *J Intern Med.* 237, 89–94 (1995).
6. Weaver CS, Leonardi-Bee J, Bath-Hextall *et al.* Sample size calculations in acute stroke trials: a systematic review of their reporting, characteristics, and relationship with outcome. *Stroke.* 35, 1216–1224 (2004).
7. Weaver CS, Leonardi-Bee J, Bath-Hextall *et al.* Can we improve the statistical analysis of stroke trials? Statistical reanalysis of functional outcomes in stroke trials *Stroke.* 38, 1911–1915 (1911).
8. Duncan PW, Jorgensen HS, Wade DT *et al.* Outcome measures in acute stroke trials: a systematic review and some recommendations to improve practice. *Stroke.* 31, 1429–1438 (2000).
9. Grotta J Calculation of sample size for stroke trials assessing functional outcome: comparison of binary and ordinal approaches. *Int J Stroke.* 3, 78–84 (2008).
10. Bath PMW, Lees KR, Schellinger PD *et al.* Statistical analysis of the primary outcome in acute stroke trials. *Stroke.* 43, 1171–1178 (2012).