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# LETTERS TO THE EDITOR

## Gallstones and Coronary Heart Disease: Some Authors Have a Lot of Gall!

TO THE EDITOR: A recent article by Méndez-Sánchez *et al.* (1) suggested a strong link between gallstones and cardiovascular disease. The study consisted of 473 asymptomatic Mexican subjects (25% with gallstones), and reported a univariate odds ratio and 95% confidence interval (OR = 4.01, 95% CI: 1.99–8.09) and a multivariate result (OR = 2.84, 95% CI: 1.33–6.07). This study has a myriad of problems. The first issue relates to the title of the article. There appears to be a lack of consistency between the terms "cardiovascular disease" in the title and "coronary heart disease" (CHD) in the body of the article, which is it? The second issue with this study is the sample size, the numbers in each group were extremely small (of those with CHD there were 19 with gallstones and 16 without gallstones) and thus the power for this study is inadequate to accurately determine the effect sizes reported. Another issue is that the abstract reports that the final multivariate model was adjusted for age, gender, and body mass index, however, in the statistical analyses section it states that the final logistic model was adjusted for age, gender, blood pressure and glucose, but not body mass index. So it remains uncertain as to which variables were actually adjusted for in the final model? A major flaw in this study is the lack of confounders that were adjusted for in the final model. The authors should have considered a broader range of potential confounding factors such as smoking, alcohol use, diabetes, infection (*Helicobacter pylori*), physical activity, triglycerides, and cholesterol to reduce bias (Fig. 1). In addition, traditionally the epidemiology of cholelithiasis in Mexican populations has a 3–3.5:1 female to male ratio (2), however, this particular study has a female to male ratio of 0.75:1. The gender differences in this study relate to lack of random selection and the larger proportion of older subjects with gallstones and I assume older males who would be more likely to have CHD, thus biasing the results of this study.

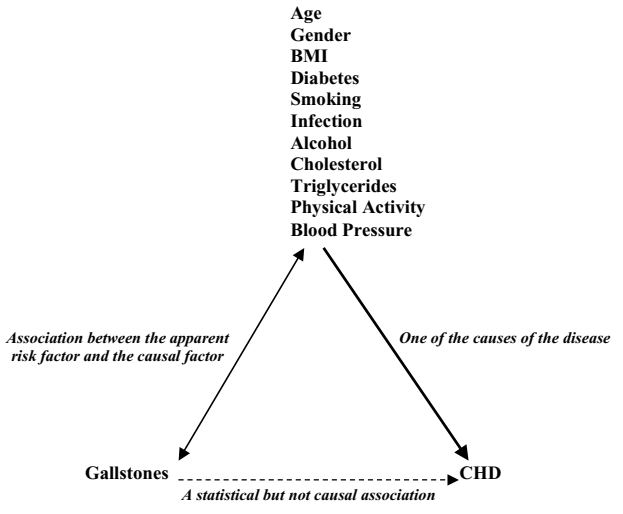


**Figure 1.** The potential confounding factors in the association between gallstones and coronary heart disease (CHD).

Previously, only a handful of studies had assessed a possible link between gallstones and CHD (3–7). The findings of these studies remain mixed and therefore the relationship remains inconclusive. Unfortunately, these studies were also flawed; some were conducted on patients who had undergone cholecystectomy, other studies failed to assess the effects of specific types of gallstones, some lacked adequate assessment of confounders including socio-economic status, most had a small sample size, others were retrospective in design rather than prospective. Future studies must be designed using orthodox epidemiological methods and statistical analysis which, must include a complete range of confounders and not just two or three potential confounding factors.

*Guy D. Eslick, Ph.D.*
*M.Med.Sc. (Clin Epi), M.Med.Stat.*
*School of Public Health*
*The University of Sydney*
*Sydney, New South Wales, Australia*
*Department of Medicine*
*The University of Sydney*
*Nepean Hospital, Penrith*
*New South Wales, Australia*

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## Response to Letter of Guy D. Eslick

TO THE EDITOR: Although disagreeing with Dr. Guy D. Eslick comments, we want to thank him for his letter on our article (2). In the first paragraph he stated that ". . .the article has a *myriad of problems*." One of the elemental lessons in writing scientific literature is to avoid the lack of precise information and the use of adjectives. He asks about a lack of consistency in the title *versus* the body of the article, v.gr. cardiovascular disease *versus* coronary heart disease. We answer this with a question: Is not coronary heart disease a cardiovascular disease? Secondly, he refers to sample size "the numbers in each group were *extremely small*." He based his comment on the comparison of 19 patients with coronary heart disease with 119 patients with gallstones (15.96%) and in 16 patients with coronary heart disease with 354 without gallstones (4.52%), and he concluded the power of the study is inadequate to accurately determine the effect sizes reported. However, the statistical difference between these figures was very significant ($p < 0.0001$), such as it could be observed in the Table 1 of the paper. Statistical power is defined as the probability to find differences statistically different. Thus, if the sample size had not been adequate, surely we had not found statistical significance. In relation with the variables included in the multivariate models they are correctly pointed out in the abstract and the results (Table 2): age, gender, and BMI were variables controlled. In the statistical analysis we omitted to point out that blood pressure and glucose were other variables tested to adjust in the multivariate logistic regression analyses, but we did not say that it was the "final model" as Dr. Eslick wrote.

Dr. Eslick mentioned that "a broader range of potential confounding factors" should be considered. It may be Dr. Eslick did not read the last paragraph in *Physical Examination* (page 2) where blood pressure measurements are mentioned and in *Analytical Procedures* (page 2) where the determination of serum concentrations of glucose, insulin, total cholesterol, HDL-C, LDL-C, triglycerides, aspartate aminotransferase, alanine aminotransferase, total protein, albumin, and total bilirubin were included. These variables were tested in a multivariate logistic regression analysis but they were not significant statistically. So we did not mention them.

We recognize a bias in the selection of the sample to favor the male sex and older ages in the group of gallstone. This deviation was corrected with the adjustment by age and gender in the logistic models. The figures of 3–3.5:1 as female to male ratio to cholelithiasis that Dr. Eslick mentions are based on Mexican American population and this is not representative of the Mexican population (2).

Dr. Eslick finishes his letter with: ". . . must include a complete range of confounders and not just two or three potential confounding factors." It appears that Dr. Eslick minimized or ignored the variables included in the multivariate model (Table 2).

Finally, we have added to the title of Dr. Eslick's letter:

**Gallstones and coronary heart disease: some authors have a lot of gall!** ***and some others have a lot of stones!***

*Nahum Méndez-Sánchez, M.D., Ph.D.*
*Jesús Bahena-Aponte, M.D.*
*Norberto C. Chávez-Tapia, M.D.*
*Daniel Motola-Kuba, M.D.*
*Karla Sánchez-Lara, B.S.*
*Guadalupe Ponciano-Rodríguez, M.D.*
*Martha H. Ramos, M.D.*
*Misael Uribe, M.D.*

*Departments of Biomedical Research and Gastroenterology & Liver Unit*
*Medica Sur Clinic & Foundation*
*Mexico City, Mexico*

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## Insulin-Like Growth Factor-1: A Common Metabolic Pathway in the Origin of Both Gallstones and Coronary Heart Disease

TO THE EDITOR: In the contribution of Mendez-Sanchez *et al*. (1), multi-regression analysis shows that presence of gallstones (GS) is independently associated with presence of coronary heart disease (CHD) and an increased waist girth. Despite their interesting observation with a possible role for hormones such as leptin, or other components of the metabolic syndrome, they do not propose any explanation with regard to their observation. The aim of our letter is to propose a novel pathophysiological pathway that could be involved. Disturbances in the growth hormone/insulin-like growth factor-1 (IGF-1) phenotype could be a possible candidate. The amount of circulating IGF-1 is mostly genetically determined and secreted by hepatocytes (2). In a recent paper, we showed that postprandial gallbladder emptying and concomitant cholecystokinin (CCK) release after a mixed meal is related to the patient's individual IGF-1 status. Indeed, low levels of plasma IGF-1 in patients with a growth hormone deficiency give rise to less postprandial gall bladder emptying due to less postprandial release of CCK and a decreased gallbladder sensitivity to CCK (3). Disturbances in postprandial gallbladder emptying are considered a risk factor for GS formation: impaired emptying allows time for nucleation of

cholesterol crystals from supersaturated bile and their subsequent aggregation into macroscopic stones. On the other hand, the total mass of truncal or visceral fat is negatively associated with plasma IGF-1 levels (2). Of special interest, recent cardiovascular research noticed that patients with a low level of IGF-1 (Dan Monica study) or polymorphisms in the IGF-1 promotor gene exhibit an increased risk on heart disease (4, 5). Mendez-Sanchez *et al*. found all these factors, the presence of both GS and HD and an increased waist girth, significantly clustered in their study.

All these previous results are consistent with the hypothesis that low plasma levels of IGF-1 may lead to the development of both GS and CHD. Additional determination by Mendez-Sanchez *et al.* of IGF-1 status (IGF-1, free IGF-1, and IGF BP-3) in their patients could support this hypothesis.

*Marcel Th.B. Twickler, M.D., Ph.D (UU), Ph.D (Paris VI)*
*Department of Endocrinology,*
*Radboud University Nijmegen Medical Centre*
*the Netherlands*

*Maarten-Jan M. Cramer, Ph.D., M.D.*
*Heart Lung Center Utrecht*
*Department of Cardiology*
*University Medical Centre Utrecht (UMCU)*
*the Netherlands*

*Kare J. van Erpecum M.D., Ph.D.*
*Department of Gastroenterology*
*UMCU, the Netherlands*

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# Transient Pneumobilia Following Upper Gastrointestinal Endoscopy

TO THE EDITOR: Pneumobilia results from an abnormal communication between the biliary tract and a viscus or abdominal cavity caused by an iatrogenic or spontaneous bilioenteric fistula. Spontaneous pneumobilia is a potentially alarming finding, suggesting a fistula or other pathology. We present a case of transient, benign pneumobilia occurring after upper gastrointestinal endoscopy. We discuss the possible mechanism of this type of pneumobilia and review the different cases of pneumobilia described in the literature.

A 79-yr-old man presented for preoperative evaluation before scheduled surgical repair of a noncomplicated asymptomatic inguinal hernia. He had no history of abdominal surgery or gallstones. The patient reported no chills, fever, jaundice, dark urine, or light stools, but did report new onset of a cough and mild production of yellow sputum. Physical examination findings were within normal limits but a rectal examination revealed dark brown guaiac-positive stool. The results of laboratory testing were consistent with mild anemia but the patient's liver enzymes level and coagulation profile were within normal limits. A diagnostic upper endoscopic examination revealed diffuse superficial mucosal erosions in the duodenal bulb without active bleeding. A routine chest radiograph revealed a left mediastinal mass and right lower-lobe pneumonia.

A computed tomography (CT)-guided fine-needle biopsy of the mediastinal mass performed several hours after the gastrointestinal endoscopy revealed small cell lung carcinoma. Multiple air bubbles within the intrahepatic and the extrahepatic bile ducts that were incidentally noted at the level of the liver sections on the chest CT scan (Fig. 1) suggested the additional diagnosis of asymptomatic pneumobilia. A repeat abdominal CT study performed 4 days later, however, showed no pneumobilia, bowel obstruction, congenital anomalies, gallstones, or other biliary abnormalities. The patient had no subsequent hepatobiliary disease on follow-up a year later.

Pneumobilia usually results from communication between the biliary and gastrointestinal or bronchoalveolar tracts. The most common causes of pneumobilia are the surgical creation

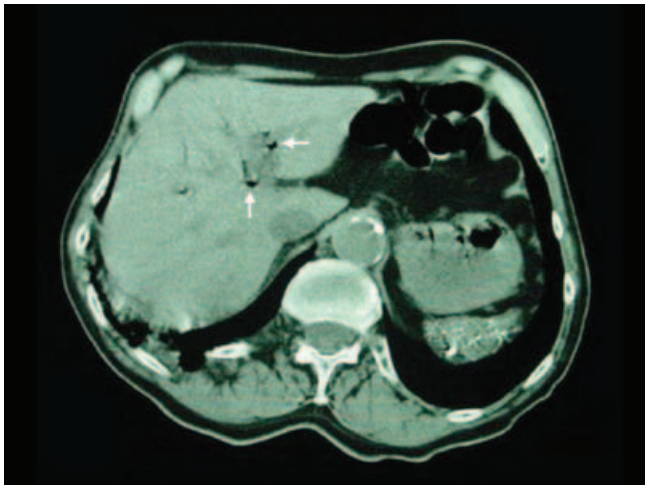

**Figure 1.** Chest CT scan showing incidental finding on the lung windows at the level of liver sections. The central distribution of air bubbles (arrows) within the intrahepatic biliary tree is consistent with pneumobilia.

of a biliary-enteric anastomoses and iatrogenic fistulas (1). Except in cases of iatrogenic bilioenteric anastomoses, the finding of air in the biliary tree of the liver is concerning for occult pathology. Spontaneous bilioenteric fistulas usually occur as a complication of passage of biliary stones (2), peptic ulcer disease (3), or gastrointestinal malignancy (1). Pneumobilia is also common after open transduodenal sphincteroplasty and endoscopic retrograde cholangiopancreatography with biliary sphincterotomy (4). Anecdotal cases of pneumobilia occurring in association with small bowel obstruction (5) and emphysematous cholecystitis (6), Crohn's disease, lymphoma, and parasitic disease of the liver have also been reported (7).

Transient pneumobilia has been attributed to incompetence of the sphincter of Oddi (8). We believe that the increased duodenal air pressure resulting from endoscopy may have induced transient sphincter incompetence in this case. A similar phenomenon may explain reflux of air into the biliary tree during blunt trauma (9), small bowel obstruction (4), and mask ventilation (7).

The incidental radiologic finding of pneumobilia within hours of upper endoscopy is benign and is probably underdiagnosed in routine clinical practice. To emphasize its transient occurrence, we chose to name this finding "*pneumobilia fugax.*" However, pneumobilia without obvious explanation, and especially if associated with signs and symptoms of biliary obstruction or cholangitis, requires further investigation.

*Fadi Braiteh, M.D.*[1]
*William Ramsey, M.D.*[2]
[1]*Phase I Program, Division of Cancer Medicine, The University of Texas M. D. Anderson Cancer Center, and the University of Texas Graduate School of Biomedical Sciences at Houston, Houston, Texas; and* [2]*Division of Gastroenterology at Hospital of Saint Raphael, Yale School of Medicine, New Haven, Connecticut*

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# Symptom-Based Outcome Measure for Nonulcer Dyspepsia

TO THE EDITOR: Dear Sir, Frase *et al.* reviewed and discussed questionnaires used to evaluate dyspepsia and GERD (1). Given that their review considered articles published prior to July 2004 only, the authors did not mention a dyspepsia questionnaire published in *Digestive Disease and Science* in December 2004 (2).

Entitled "Porto Alegre Dyspeptic Symptoms Questionnaire" (PADYQ), our questionnaire is a unidimensional instrument that evaluates symptoms in terms of frequency, duration, and intensity, as appropriate, as suggested by Frase *et al.* in their article. The time frame used, which is in accordance with McColl (3), comprises the 30 days prior to the date when the questionnaire is applied.

We agree with the opinion of Frase *et al.* that questionnaires should be rigorously evaluated through a validation process. The PADYQ showed high levels of internal consistency, reproducibility, responsiveness, face validity, discriminant validity, and concurrent validity. Moreover, a factorial analysis identified three domains in our questionnaire: pain; nausea/vomiting; bloating/early satiety.

The questionnaire is not copyrighted and may be used by other research teams in other countries. An English version of the questionnaire may be obtained and translated into other languages, with the proviso that the original name and acronym of the questionnaire be maintained, and the original paper be quoted (2).

*Guilherme B. Sander, M.D.*
*Luiz Edmundo Mazzoleni, M.D., Ph.D.*
*Eduardo A. Ott, M.D.*
*Carlos Fernando Francesconi, M.D., Ph.D.*
*Gastroenterology Division*
*Hospital de Clinicas de Porto Alegre*
*Universidade Federal do Rio Grande do Sul*
*Porto Alegre, Brazil*

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## Epidemiology of GIST

TO THE EDITOR: Tran *et al.* recently stated they had conducted the first population-based study of malignant gastrointestinal stromal tumor (GIST), and that there were only 1,458 cases diagnosed in the United States between 1992 and 2000 (1). There are several limitations to this study, calling that number into question. The authors included only SEER cases encoded with "confirmed malignant behavior." Currently, all GISTs are considered potentially malignant, and the majority of resected "benign" GISTs recur. It is extremely likely a large number of GISTs, coded as leiomyomas or other benign malignancies, or not included in the registry at all, were missed. Tran did not confirm the diagnosis of GIST using modern criteria. The investigators were forced to include a hodge-podge of coded tumor types (ranging from "glomus tumors" to "mesenchymoma"), so the actual percentage of true GISTs is wholly unknown. Also, the authors only included "sarcomas" arising from a limited number of GI sites. GISTs can arise from anywhere in the GI tract, so tumors originating in gall bladder, pancreas, mesentery, et cetera would not be included in the estimate.

Several other important data collections support the concept that GISTs occur with much greater frequency than estimated by Tran. A recent Southwest Oncology Group metastatic GIST trial accrued 746 patients in 9 months (2). When the study was conducted, nonresectable GIST was a uniformly fatal disease, so the prevalence did not dramatically exceed the incidence. It is difficult to comprehend the rapidity of this accrual if only 94 new cases (the number predicted by the authors' data) actually occurred during that period. Nilsson reported a population-based study in which the authors pathologically reviewed all cases of mesenchymal and nervous system GI tumors arising in a defined Swedish population, culling the true GISTs (3). They estimated a yearly incidence/prevalence of 14.6/129 per million, respectively. Another population-based study was reported by Tryggvarson, who identified all GISTs diagnosed in Iceland in a 14-year period and reported an incidence of 11 cases per million (4). There is no biologic reason to suspect major differences would occur between Scandinavian and American populations; extrapolation of the Nilsson data to the United States leads to an estimated annual incidence of 4,320 GISTs, a number consistent with the rapid accrual to nearly all clinical trials.

While GIST remains a rare cancer, its actual incidence would seem to parallel that of Hodgkin's disease, or chronic myelogenous leukemia. It is important to recognize the frequency of this cancer, as effective therapy (imatinib) now exists (2).

*Charles Blanke, M.D.*
*ONSV Cancer Institute*
*Portland, OR*

*Burton L. Eisenberg, M.D.*
*Dartmonth-Hitchcock Medical Center*
*Lebanon, NH*

*Michael Heinrich, M.D.*
*ONSV Cancer Institute*
*Portland, OR*

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## External Validation for Fibrosis Predicting Index in Hereditary Hemochromatosis

TO THE EDITOR: We read with great interest the article by Olynyk JK *et al.* (1) assessing the relation between the duration of hepatic iron exposure and development of fibrosis in hereditary hemochromatosis (HH). It is very appealing from a clinical point of view because we could avoid some of the biopsies for prognostic purposes. Thus, a simple index (age × hepatic iron content) with a well-defined cut-off value would allow us to know who among our HH patients have a high-grade fibrosis.

We have estimated the index from Olynyk in a cohort of 21 HH patients previously published (2). The results we obtained (for a prevalence of fibrosis of 23.8%) are close to those from the original article: sensitivity, 80% (95% CI: 28.36–99.49) and specificity, 68.75% (95% CI: 41.34–88.98). The point estimates of sensitivity and specificity from Olynyk may somehow raise overoptimistic expectations, and we would like to see the interval estimates as we have just done in our cohort. Using Olynynk published data, these would be, for

a prevalence of 30%, sensitivity 100% (95% CI: 81.47–100) and specificity 66.67% (95% CI: 50.45–80.43).

While the clinical utility of MR imaging for hepatic iron concentration quantification is unquestionable since the previous published papers by us and others (2, 3), its performance as a diagnostic tool for fibrosis is lower. The proposed index is very promising but, in our opinion, it should not be used alone but in conjunction with other less than perfect predictive parameters (4, 5).

Physicians caring for HH patients will welcome a further study combining the prediction power of MRI, age, and other relevant clinical data. While this occurs, we salute any effort like the one discussed here, to provide prognostic information in a noninvasive manner.

*Agustin Castiella, M.D.*
*Gastroenterology, Mendaro Hospital*

*Jose I. Emparanza, M.D., Ph.D.*
*Clinical Epidemiology, Donostia Hospital*
*Spain*

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# Gastroenterologists Should Decline NonBeneficial PEG Tube Placements

TO THE EDITOR: In his recent commentary "The PEG 'Consult'," Dr. Scott expertly discusses the ethical issues surrounding percutaneous endoscopic gastrostomy (PEG) tube placements by gastroenterologists (1). He rightly focuses on whether gastroenterologists should function as technicians, inserting PEG tubes on request, or as clinicians, assessing the appropriateness of the procedure for each patient.

He compares the perceived role of gastroenterologists to that of radiologists, who typically do not question the clinical decisions made by requesting physicians. PEG tube placement, however, is not a diagnostic test but a surgical intervention. As such, it seems more appropriate to compare gastroenterologists in this setting with surgeons, who routinely refuse to perform requested procedures from which they do not expect patients to benefit.

While the author focuses on PEG tube placements in dementia, it should be noted that they also have proven ineffective in the prevention of aspiration pneumonia (2) or for nutritional support in terminal cancer (3), two other conditions for which they are commonly used. In fact, benefit has been shown only in early head and neck cancer, malignant bowel obstruction, amyotrophic lateral sclerosis, and acute stroke with dysphagia persisting one month after hospital discharge (4).

As the author implies, all physicians, including gastroenterologists, have an ethical obligation not to perform nonbeneficial invasive procedures regardless of who requests them. Doing so, in fact, may constitute a clinical form of torture (5). Time- or outcome-limited trials, suggested for cases in which consensus cannot be reached, are in my experience rarely successful and usually serve only to transform the decision to withhold artificial nutrition to the more difficult decision to withdraw it.

*William M. Plonk, Jr., M.D.*
*Department of Internal Medicine*
*University of Virginia Health System*
*Charlottesville, Virginia*

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