CORRESPONDENCE

Readers are encouraged to write Letters to the Editor concerning articles that have been published in GASTROENTEROLOGY. Short, general comments are also considered, but use of the Correspondence Section for publication of original data in preliminary form is not encouraged. Letters should be typewritten double-spaced and submitted in triplicate.

Occult Blood Screening

Dear Sir:

We read with interest the review of occult blood screening by Simon (1) in GASTROENTEROLOGY. However, we wish to point out some important points that have been incorrectly stated.

It is stated that the trials at the Minnesota and the Sloan-Kettering Cancer Centers are the only controlled studies presently on-going. The study in Nottingham (2), erroneously quoted in the table of "Selected Uncontrolled Studies of Occult Blood Screening," is indeed a controlled study. At the time of publication this study had recruited 20,525 individuals who were randomly allocated into test and control groups. The report gave the follow-up at 1 yr of both test and control groups. This program has now recruited 35,000 individuals and a total of 120,000 individuals will ultimately be randomized and observed for 10 yr. It is also worth pointing out that unlike the Sloan Kettering and Minnesota programs, which have as control groups either subjects attending for sigmoidoscopy, health care checks, or interested volunteers, the control group is not contacted and subjects are simply followed up using the family practitioner and hospital pathology records as well as the Regional Cancer Registry. They remain unaware of the screening program so that the effect of screening can be more clearly measured.

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1. Simon JB. Occult blood screening for colorectal carcinoma: a critical review. Gastroenterology 1985;88:820–37.
2. Hardcastle JD, Farrands PA, Balfour TW, et al. Controlled trial of faecal occult blood testing in the detection of colorectal cancer. Lancet 1983;ii:1–4.

Reply. My apologies to Hardcastle and his colleagues for misleadingly grouping their Nottingham study (1) with uncontrolled screening trials. As noted, however, control subjects are unaware of their participation and are not specifically followed; instead, any cancers among them are garnered indirectly from pathology registers, etc. Moreover, the stated purpose of the study was "to compare the yield of neoplasia in a test group offered haemoccult screening with that in a control group" (1), rather than to assess long-term prognostic outcome. For these reasons I decided against discussing the Nottingham program with the Sloan-Kettering and Minnesota controlled studies. Nevertheless, the ongoing Nottingham trial is important, and I sincerely regret my error.

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1. Hardcastle JD, Farrands PA, Balfour TW, et al. Controlled trial of faecal occult blood testing in the detection of colorectal cancer. Lancet 1983;ii:1–4.

Yersinia enterocolitica Peritonitis

Dear Sir:

We read with interest the article in GASTROENTEROLOGY by Capron et al. (1) on spontaneous *Yersinia enterocolitica* peritonitis in idiopathic hemochromatosis.

In patients with iron overload, *Y. enterocolitica* septicemia has been frequently reported (2,3), however, *Y. enterocolitica* peritonitis has been very rarely described (1,4).

In the last few years we observed 3 cases of *Y. enterocolitica* peritonitis in children affected by thalassemia major.

Case 1. This case, published in detail elsewhere (5), is briefly summarized here: an 8-yr-old thalassemic male, who had undergone splenectomy 1 yr before his present illness, was hospitalized with a working diagnosis of acute abdomen. Laparotomy revealed the presence of creamy, purulent exudate in the peritoneal cavity without evidence of gastrointestinal perforation or appendiceal involvement. Suppurative and necrotic mesenteric lymphadenitis was elicited. Postoperatively, the patient was treated with cefoxitin and gentamicin and was discharged after 20 days. Peritoneal fluid culture yielded *Y. enterocolitica* biotype 4, serogroup 0:3, phage type VIII. Autologous strain agglutination tests during acute and convalescent phase revealed *Y. enterocolitica* antibody titer of 1:2000 and 1:200, respectively.

Case 2. A 13-yr-old thalassemic male entered the hospital with a diagnosis of acute abdomen. He had undergone splenectomy 5 yr earlier. At laparotomy, cloudy peritoneal fluid was encountered. The appendix appeared gangrenous and several mesenteric nodes were enlarged and suppurative. Postoperatively, the patient received netilmicin and was discharged after 15 days. Stool and peritoneal cultures yielded *Y. enterocolitica* biotype 4, serogroup 0:3, phage type VIII. Stool cultures obtained from the patient's pet dog were positive for the same strain of *Y. enterocolitica*. Serodiagnosis performed only during the acute phase of disease revealed a *Y. enterocolitica* 0:3 antibody titer of 1:2000.

Case 3. A 6-yr-old thalassemic male was admitted to the hospital with a diagnosis of acute appendicitis. There was no history of spenectomy. Laparotomy revealed purulent exudate in the peritoneal cavity, mesenteric lymphadenitis, terminal ileitis, and gangrenous appendicitis. Pus was encountered in the mesenteric lymph nodes. Cultures of peritoneal fluid and blood were negative. No pathogenic bacteria were grown from stool cultures. However, the patient had been treated with cefuroxime for 3 days before surgery. Serodiagnosis revealed a high titer (1:1280) of antibodies against *Y. enterocolitica* 0:3 during the acute phase and a subsequent decline (1:160) after 2 mo. The serum samples also revealed the absence of anti-*Y. enterocolitica* 0:9 and *Brucella* sp agglutinins. On the basis of the serologic results, the patient was treated with tobramycin. He became afebrile after 48 h and was discharged 9 days later.

The present 3 cases support the view expressed by Capron et al. (1) that a diagnosis of *Y. enterocolitica* infection be considered in patients with iron overload who present with abdominal syndromes and represent the pediatric counterpart of the 2 cases (1,4) of *Y. enterocolitica* peritonitis in adults with iron overload.

It is noteworthy that in all our 3 cases *Y. enterocolitica* peritonitis was associated with mesenteric lymphadenitis as a stepping-stone from intestinal to peritoneal infection.

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1. Capron JP, Capron-Chivrac D, Tossou H, Delamarre J, Eb F. Spontaneous *Yersinia enterocolitica* peritonitis in idiopathic hemochromatosis. Gastroenterology 1984;87:1372–5.
2. Bouza E, Dominguez A, Meseguer M, et al. *Yersinia enterocolitica* septicemia. Am J Clin Pathol 1980;74:404–9.
3. Rabson AR, Hallett AF, Koornhof HJ. Generalized *Yersinia enterocolitica* infection. J Infect Dis 1975;131:447–51.
4. Rabson AR, Koornhof HJ. *Yersinia enterocolitica* infections in South Africa. S Afr Med J 1972;46:798–803.
5. Levi Della Vida MV, Campanelli G, Boemi L. *Yersinia enterocolitica* peritonitis in a child with Cooley's disease and splenectomy. Ital J Gastroenterol 1982;14:31–3.

Classification of Pancreatitis—Cambridge and Marseille

Dear Sir:

The March 1983 Cambridge (1) and the March 1984 Marseille (2) Workshop meetings to improve the classification of pancreatitis recommended the first major changes in the classification and nomenclature of pancreatic inflammatory disease since the first Marseille meeting in 1963. These revisions of the 1963 Marseille classification of pancreatitis are most welcome and timely, and the participants and authors are to be complimented on their products.

These new classification schemes have now been published. We believe these proposals deserve careful analysis regarding their usefulness in improving communication and understanding of pancreatic inflammatory disease among individuals involved in patient care, research, and advancing our knowledge of the natural history of pancreatitis.

Features in Common and Differences

Both the Cambridge and the Marseille classifications stress that structural and functional damage in chronic pancreatitis are irreversible. Both definitions do away with the often difficult clinical distinctions between chronic and chronic relapsing pancreatitis. We agree this is a sensible step.

Chronic Pancreatitis

In both workshops, the view was expressed that histologic changes in chronic pancreatitis are irreversible, may be progressive, may lead to loss of exocrine and endocrine pancreatic function, and are often associated with abdominal pain. The Marseille conference also delineated a distinct morphologic form of chronic pancreatitis, which they labeled obstructive chronic pancreatitis. Structural and functional changes tend to improve in this form of chronic pancreatitis if the ductal obstruction is removed.

Acute Pancreatitis

The Cambridge and Marseille 1984 conferees expressed very similar views regarding the clinical description of acute pancreatitis. The presence of abdominal pain, elevation of enzymes in blood and urine, and recognition that there may be systemic response of varying severity and that the attacks can recur were cited in both workshops. Complications of acute pancreatitis noted by the Cambridge group included necrosis, hemorrhage, phlegmon, pseudocyst, and abscess. The Marseille group described necrosis, hemorrhage, pseudocyst, but not abscess.

The 1984 Marseille definition of acute pancreatitis goes beyond the clinical description of the Cambridge group and outlines essential morphologic criteria necessary to the diagnosis. These are as follows: *mild form*, peripancreatitis fat necrosis and interstitial edema; *severe form*, extensive peri- and intrapancreatic fat necrosis, parenchymal necrosis, and hemorrhage. These lesions may be localized or diffuse. Clinical morphologic and functional abnormalities return to normal after each clinical attack.

Inclusion of the morphologic and functional criteria in the definition of acute and chronic pancreatitis creates a quandry for the clinician at the bedside attempting to label a patient having his initial attack of pancreatitis. The fact that morphologic and functional information are required for the distinction between acute and chronic pancreatitis, but are rarely available at the time of the initial attack is at the heart of the dilemma. Biopsies of the pancreas and pancreatic function tests are rarely performed at the time of the initial attack of pancreatitis. Yet, morphologic and functional information about the pancreas are essential to making the distinction between acute and chronic pancreatitis according to the Marseille definition, as well as to defining the gradations of severity of the attack. Should the patient's physician postpone making a decision as to whether the attack should be labeled acute or chronic pancreatitis until after recovery of the patient from the attack, when function studies are feasible, or until after death or at operation, when histologic material becomes available? Yes, would have to be the answer according to the Marseille classification.

The Cambridge definition of acute pancreatitis based on clinical criteria can be applied by the physician at the time of the initial attack. Should the patient, after further investigation, be found to have permanent structural damange, as evidenced on computed tomography scan or endoscopic retrograde cholangiopancreatography, the initial acute attack, based on clinical criteria, may be accurately viewed as an exacerbation of chronic pancreatitis. The inclusion of structural data is helpful because such data are usually available, whereas the histologic data are not. There were additional differences. The Cambridge group believed that the etiology of pancreatitis was pertinent to prognosis and management of the patient with either acute or chronic pancreatitis and should be noted. The Marseille group did not.

Areas for Further Study

Both workshops agreed that our understanding of pancreatitis was far from complete. Further investigations were recommended in the following areas.