Opinion from the United States

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In recent years improved understanding of the clinical pathological correlation and the natural history of acute pancreatitis has resulted from information acquired through computed tomography (CT) with vascular enhancement, CT-guided aspiration for infection assessment, endoscopic retrograde cholangiopancreatography, ultrasound, visceral angiography and operative findings. Stimulated by this flow of new information, a constantly evolving series of classification systems of acute pancreatitis have been produced.


Sarles [10] in 1965 published a morphologic classification of pancreatitis after the 1963 International Symposium in Marseilles. He asserted the lesions of acute pancreatitis were reversible and those of chronic pancreatitis progressive with sclerosis and loss of exocrine parenchyma. The Marseilles classification system became widely but often incorrectly applied. Clinicians, as Sarles pointed out, persisted in employing the terms, “acute,” “chronic,” and “recurrent” as descriptors of the clinical cause of disease rather than histopathologic descriptions of the pancreas which were usually unknown to the clinicians [11].

Application of the new technology of ultrasound, CT, angiography and ERCP to the study of inflammatory disease of the pancreas increased understanding of the natural history and clinical pathologic correlation of acute and chronic pancreatitis and led to the Cambridge 1983 and Marseilles 1984 symposia on the classification of pancreatitis [12, 13]. The Cambridge group advanced the notion that the terms “acute” and “chronic” should incorporate the clinical course of the patient and pancreatic function (exocrine and endocrine) as well as the histopathology and morphology as delineated by ultrasound, CT, angiography and ERCP. The participants felt it was important to quantitate the ultrasound, ERCP, CT and angiographic changes and correlate these findings with the patient’s
clinical course to better understand the connection between ductal and parenchymal changes, pancreatic function, and clinical severity of pancreatitis. They noted the etiology of pancreatitis influences the natural history of the disease and how patients will be managed and should be stated.

The 1984 Marseilles classification [12] shared many features of the 1983 Cambridge classification of pancreatitis including the clinical description of acute pancreatitis. Both symposia agreed that the presence of abdominal pain, elevation of enzymes in blood and urine were characteristics of pancreatitis and that the attacks varied in severity and acute pancreatitis could recur. Complications of acute pancreatitis cited by both groups were necrosis, hemorrhage, and pseudocyst. The Cambridge group additionally described phlegmon and abscess. The Marseilles criteria for diagnosis of acute pancreatitis in 1963 and 1984 required histologic criteria: "mild," – peripancreatic necrosis and interstitial edema; "severe," extensive peri and intrapancreatic fat necrosis, parenchymal necrosis and hemorrhage. These lesions which could be local or diffuse were said to return to normal after the attack and there would be no loss of exocrine or endocrine function. This is an over simplification in the case of necrotizing pancreatitis. Some patients with alcoholic pancreatitis following recovery from the acute attack may have some recovery of exocrine and endocrine function while some patients with biliary pancreatitis will not recover exocrine and endocrine function [14, 15].

The Marseilles-Rome Classification of 1988 [16] differs from the earlier versions. The 1988 version references the complications of acute pancreatitis including infected necrosis, abscess and fluid collections. The etiology of pancreatitis was included for the first time. The 1988 Marseilles-Rome classification of acute and chronic pancreatitis continued to be based on histopathologic descriptions of the pancreas.

The flood of information about the pathogenesis and natural history of pancreatitis derived from the use of ultrasound, CT, ERCP and angiography in patients with acute pancreatitis was largely responsible for the proliferation of classification systems in the 1980s.

The 1992 Atlanta symposium to develop clinically based classification system for acute pancreatitis was predicated on information derived from widespread utilization of CT with vascular enhancement, and clinicians need for a classification system for acute pancreatitis which was clinically applicable. The Atlanta Classification System and definitions of acute pancreatitis were preceded by two proposals initiated by Frey, Bradley, and Beger [17] and Frey [18] which had many similarities to those adopted by the international multidisciplinary group. During the late 1980s and early 1990s the widespread use of CT with bolus vascular enhancement in acute pancreatitis improved our understanding of necrotizing pancreatitis. The necrosis of the pancreas was found to occur most often within hours of the onset of symptoms. In the absence of infection, there was little extension of the areas of necrosis within the pancreatic parenchyma. Peripancreatic necrosis and fluid collections on the other hand evolved over a period of weeks and the outcome was variable. Disruption of the major and