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Chronic Hepatitis

An Update on Terminology and Reporting

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The terms chronic active hepatitis (CAH), chronic persistent hepatitis (CPH), and chronic lobular hepatitis (CLH) have become obsolete, and their use without further specifications should be discontinued. This recommendation has become necessary because these names have changed from descriptive terms, intended for grading, to terms that are used either as morphologic diagnoses or disease designations or both, depending on individual preferences. Because this practice has caused serious misunderstandings, many authors and two international groups have recommended the use of a clear etiologic terminology. For the reporting practice of pathologists, we recommend that the pathologist routinely sign out biopsy samples with features of chronic hepatitis by indicating etiology, grade, and stage. An example would be autoimmune hepatitis, severe, stage 3. The stage in this case would indicate the presence of well-developed septal fibrosis but no nodular regeneration. Obviously, for the etiologic diagnosis, morphologic findings must be integrated with clinical and laboratory data. If this information is not available, clear morphologic diagnoses should be reported. Thus, instead of CPH, the diagnosis should be portal hepatitis, cause undetermined. This reporting practice eliminates ambiguous terminology and avoids the risk of inappropriate treatment as might occur, for example, when a term such as CAH is used to describe Wilson's disease and is misunderstood to mean autoimmune hepatitis. For a transitional period and to facilitate relearning, the terms CAH, CPH, and CLH can be reported in parentheses behind the etiologic diagnosis.
Key Words: Chronic active hepatitis—Chronic persistent hepatitis—Definitions—Terminology.

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HISTORICAL CONTEXT OF TERMINOLOGY

Most of the terms traditionally used to describe chronic hepatitis had their roots in the 1950s and 1960s, an era when the causes of chronic hepatitis were poorly understood. The concept of chronic viral hepatitis arose from military experiences in World War II, when soldiers with acute episodes of jaundice subsequently developed evidence of chronic liver disease (17). Lupoid hepatitis, now recognized as autoimmune hepatitis, became a popular term in 1956 (22). The distinction between viral and autoimmune liver disease was often problematic and imprecise, however, given the limited number of diagnostic tests available.

The term *chronic persistent hepatitis* (CPH) had its roots in the mid-1950s as "persistent hepatitis," which was used to describe a nonprogressive variant of viral hepatitis (30). "*Chronic active hepatitis*" (CAH) also had its origins around this time (26). The terms CPH and CAH were solidified in 1968 in a classification of chronic hepatitis published by an international group of liver experts (9); "chronic lobular hepatitis" (CLH) was popularized in 1971 (25). The authors of the 1968 classification system had intended that terms such as CPH and CAH would refer to degrees of disease activity within a diagnostic category rather than represent separate disease entities (10). At that time, the pathogenesis of chronic hepatitis and the respective roles of viral infection and chronic immune response were still unclear, precluding precise etiologic diagnoses.

Whereas greater insight into the etiology and pathogenesis of chronic hepatitis has developed over the ensuing decades, terminology and reporting practices have largely failed to keep pace and have in fact become sources of confusion. In particular, the differential diagnosis of the morphologically descriptive terms CPH and CAH has grown considerably, although, as stated, these terms initially referred to morphologic patterns of injury in individuals with presumed viral or autoimmune disease. As an example of the expanded differential diagnosis, we quote from a 1992 abstract in which autoimmune hepatitis is discussed; "CAH is a morphological lesion that is seen in a number of other

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liver disorders, notably acute hepatitis A, chronic hepatitis B, primary biliary cirrhosis, primary sclerosing cholangitis, alcohol- and drug-induced liver damage, and Wilson's disease" (23). It should be noted that most liver textbooks, including our own (21), list more or less similar differential diagnoses for CAH.

Perhaps an even less desirable consequence has been the gradual misconception that CPH and CAH are etiologic diagnoses unto themselves. As a result of this blurred meaning for CAH, it is now possible to find published therapeutic trials in which the subjects are treated for "chronic active hepatitis" without reference to whether the disease is viral, autoimmune, or of other etiology (1).

Interestingly, the danger of confusing descriptive terms intended for grading with etiologic diagnoses was recognized years earlier by Drs. Popper and Schaffner, who proposed purely morphologic terms such as "periportal hepatitis" to distinguish clearly between a biopsy finding and clinical disease (25). Unfortunately, this approach never gained popular acceptance, and therefore the name CAH continues to be defined by its main histologic feature, piecemeal necrosis.

The terms CPH and CAH are also undesirable on a linguistic basis. Principles of terminology discourage the use of different names for mild and severe forms of the same disease (e.g., in previous terminology, chronic hepatitis B might be diagnosed as either CAH or CPH) or the same name for different diseases (e.g., CAH referring to viral, autoimmune, metabolic, or biliary disease).

Dissatisfaction with current nomenclature eventually led to a number of editorials from both clinicians and pathologists calling for the development of new classification systems for chronic hepatitis with improved terminology and greater standardization of reporting (8,11,20,27,29,31). Committees were also commissioned by both the International Association of the Study of the Liver (IASL) and the World Congresses of Gastroenterology to study, discuss, and publish current thoughts on the classification and terminology of chronic hepatitis (10,13). Because much of this recent literature has been published in clinical journals, the aim of this article is to summarize recent developments in the classification, terminology, and reporting of chronic hepatitis for the practicing anatomic pathologist.

CLASSIFICATION OF CHRONIC HEPATITIS

Chronic hepatitis is not a single disease, but rather a clinical and pathologic syndrome that may have a variety of causes (10). Traditionally, chronicity has been defined clinically as continuing disease for at least 6 months (19). This definition still has some practical utility, but asymptomatic disease must also be taken into account; for example, both hepatitis C and autoimmune hepatitis (5) may remain asymptomatic for long periods. Thus, in many cases histologic evidence of chronicity can be used to infer disease of longer than 6 months' duration and permit therapy for chronic disease. Indeed, an international panel has formally recognized autoimmune hepatitis as an a priori chronic disease and thus recommended eliminating the descriptor "chronic" from autoimmune hepatitis (16). Metabolic diseases, such as Wilson's disease, can also be considered a priori chronic.

The chronic hepatitides consist of chronic necroinflammatory diseases in which hepatocytes rather than biliary structures appear to be the main target of attack. Chronic cholestatic diseases, such as primary biliary cirrhosis (PBC) and primary sclerosing cholangitis (PSC), and metabolic disorders, such as Wilson's disease and alpha-1-antitrypsin (A1AT) deficiency, are not always included under the heading of chronic hepatitis (10). However, they may show similar morphologic features, and there is thus practical merit in considering them in the broader spectrum of chronic hepatitis (13). Alcoholic and nonalcoholic steatohepatitis are also causes of chronic liver damage; however, they are sufficiently different morphologically from the chronic hepatitides that nothing is gained by placing them in the latter category. A practical current classification of chronic hepatitis based on putative or proven pathogenesis is shown in Table 1.

DEFINITIONS OF CHRONIC HEPATITIS

An international working party of the World Congresses of Gastroenterology 1994 compiled a list of

TABLE 1. *Etiologic classification of chronic hepatitis and chronic biliary diseases*

Chronic (nonbiliary) hepatitis
Autoimmune hepatitis
Chronic hepatitis B
Chronic hepatitis B and D
Chronic hepatitis C
Chronic drug hepatitis
Chronic hepatitis, unclassified as to viral or autoimmune etiology (including cryptogenic chronic hepatitis)
Wilson's disease of the liver
Alpha-1-antitrypsin disease of the liver
Chronic biliary disease
Primary biliary cirrhosis
Primary sclerosing cholangitis
Autoimmune cholangitis

recommended terms and definitions for chronic hepatitis (13), many of which continue to emphasize the 6-month or longer duration used traditionally. It should be emphasized that the 6-month requirement is of limited utility in individual cases and is best applied to multicentric and other large trials where assurance is needed that the study groups are indeed composed of chronic cases. For autoimmune hepatitis, the 6-month requirement, and thus the adjective "chronic," are no longer needed (16). The definitions are summarized below:

Autoimmune Hepatitis

This is an unresolving, predominantly periportal hepatitis, usually with hypergammaglobulinemia and tissue autoantibodies, which is in most cases responsive to immunosuppressive therapy. Objective and standardized criteria for the diagnosis of autoimmune hepatitis using biochemical, serological, and clinical features were recently published by an international group (16). Based on serologic patterns, multiple subtypes exist (16). The disease is considered a priori chronic.

Chronic Hepatitis B

This is an inflammatory disease of the liver which is caused by hepatitis B virus (HBV), is of 6 months or longer duration, and has the potential to progress to cirrhosis or to be associated with cirrhosis.

Chronic Hepatitis D

This is an inflammatory disease of the liver which is caused by hepatitis D virus (HDV), in conjunction with HBV infection, is of 6 months or longer duration, and has the potential to progress to cirrhosis or to be associated with cirrhosis.

Chronic Hepatitis C

This is an inflammatory disease of the liver which is caused by hepatitis C virus (HCV), is of 6 months or longer duration, and has the potential to progress to cirrhosis or to be associated with cirrhosis.

Chronic Hepatitis, Unclassified as to Viral or Autoimmune Origin

This is an inflammatory disease of the liver of 6 months or longer duration that resembles chronic viral and autoimmune hepatitis morphologically, but information on viral or autoimmune injury is not available or diagnostic clinical or morphologic findings cannot be identified. A synonym for the latter setting is the term cryptogenic chronic hepatitis (10).

Chronic Drug Hepatitis

This is an inflammatory disease of the liver that lasts 6 months or longer and is caused by an adverse drug effect, which may be (a) a direct toxic effect of a drug or of a metabolite(s) of a drug or (b) an idiosyncratic reaction of a drug or a metabolite(s) of a drug.

Primary Biliary Cirrhosis

This is a chronic cholestatic, granulomatous, and destructive inflammatory disease of interlobular and septal bile ducts, which is believed to be caused by autoimmunity and has the potential to progress to cirrhosis or to be associated with cirrhosis.

Primary Sclerosing Cholangitis

This is a disease characterized by chronic, progressive, fibrosing inflammation of the bile ducts which usually affects both the extrahepatic and intrahepatic biliary ductal systems, leading to biliary cirrhosis and hepatic failure. Its cause is unknown.

Wilson's Disease of the Liver

This is a chronic liver disease caused by an autosomal-recessive disorder of copper metabolism, which has the potential to progress to fulminant hepatic failure or to chronic hepatitis and cirrhosis or to be associated with one of these complications.

A1AT Deficiency Disease of the Liver

A chronic liver disease associated with or caused by an autosomal-recessive disorder of protein metabolism, typically with abnormally low values of serum A1AT, this disease may lead to chronic hepatitis and cirrhosis or may be associated with these conditions. In the pediatric age group, A1AT may be associated with nonsyndromatic paucity of intrahepatic bile ducts or with neonatal hepatitis with giant cell transformation.

Systemized Nomenclature of Medicine (SNOMED) coding can be readily applied to all these terms. The appropriate SNOMED International codes are listed in detail elsewhere (13).

MORPHOLOGIC FEATURES

The various etiologic types of chronic hepatitis share a number of histologic characteristics that may vary over time in an affected individual. Most of these common morphologic features allow the pathologist to assess the grade (severity of inflammatory activity) and stage (degree of fibrosis) of the disease process but do not always allow a definitive distinction between the various etiologies. In general, lobular inflammation predominates in acute forms of hepatitis, and portal and periportal inflammation predominates in chronic hepatitis. Chronic hepatitis with flares of disease activity commonly shows lobular hepatitis together with portal and periportal inflammation and fibrosis (5). A brief summary of the major histopathologic features of the chronic hepatitides is listed below; a more detailed review was recently published (14). A schematic drawing of the three main morphologic patterns—portal, periportal and lobular hepatitis—is provided in Fig. 1.

Portal Inflammation (Portal Hepatitis)

Portal inflammation (Fig. 1A) is common to all forms of chronic hepatitis and is composed mainly of a mixture of lymphocytes, plasma cells, and macrophages. Plasma cells sometimes predominate in autoimmune hepatitis. Bile duct damage with lymphocytic cholangitis is frequently seen in hepatitis C (7); nondestructive lymphocytic cholangitis is also common in autoimmune hepatitis (5), primary biliary cirrhosis, primary sclerosing cholangitis, and autoimmune cholangitis. The presence of lymphoid aggregates or follicles, although not pathognomonic, is typical of chronic hepatitis C and is seen in about half to three-fourths of cases (3,28).

In practical terms, if portal inflammation is prominent but a definite acute hepatitic episode had not

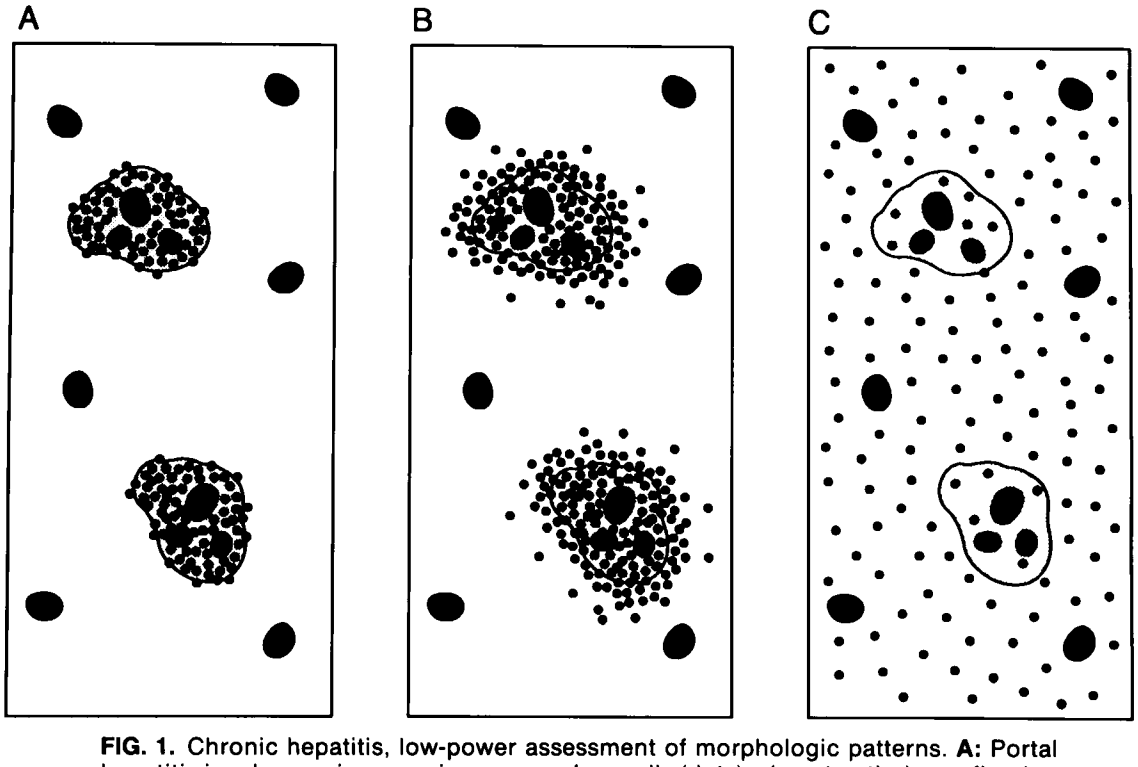

FIG. 1. Chronic hepatitis, low-power assessment of morphologic patterns. **A:** Portal hepatitis involves an increase in mononuclear cells (dots), almost entirely confined to portal areas. At scanning magnification, this results in portal areas being sharply delimited. **B:** In periportal hepatitis, an increase in mononuclear cells (dots) in the periportal parenchyma (zone 1) occurs, commonly associated with piecemeal necrosis and lobular inflammation of variable degree. The result is a low-power impression of portal-dominant inflammation; however, the portal areas are less sharply defined than in portal hepatitis. **C:** Lobular hepatitis is characterized by lobular inflammation, with or without disarray and necrosis. Pure lobular hepatitis is a feature of acute hepatitis; however, lobular hepatitis in conjunction with considerable portal and periportal inflammation is typical of "flares" of chronic viral or autoimmune hepatitis.

been evident clinically in the preceding 6 months, it is still reasonable for the pathologist to diagnose chronic hepatitis. If portal inflammation is not accompanied by prominent periportal or lobular inflammation, the process can be described as *inactive*.

Periportal Inflammation (Periportal Hepatitis)

Periportal inflammation (Fig. 1B) is commonly accompanied by evidence of local hepatocyte damage; this necroinflammatory process is referred to as lymphocytic piecemeal necrosis (4). The composition of these inflammatory infiltrates is identical to those in the portal tracts; in autoimmune hepatitis, plasma cells are frequently prominent. As a consequence of the necroinflammatory process, collagen is deposited. The presence of periportal hepatitis is evidence of inflammatory disease activity and contributes to the grading of the chronic hepatitis (below).

Lobular Inflammation (Lobular Hepatitis)

Lobular inflammation (Fig. 1C) is usually accompanied by evidence of hepatocellular damage. In contrast to the portal and periportal inflammation, however, lobular inflammation usually consists of single small clusters of mononuclear cells rather than confluent sheets. Hepatocellular damage is generally manifested by scattered necrotic hepatocytes (acidophilic or Councilman bodies), hepatocellular nuclear disarray (anisonucleosis), mitotic activity, and hepatocellular swelling. The degenerative and regenerative hepatocellular changes are frequently more impressive than the number of inflammatory cells.

The severity of lobular necroinflammatory changes varies considerably. Spotty hepatocellular necrosis describes death or dropout of minute clusters of hepatocytes, whereas confluent necrosis affects entire lobules. Bridging necrosis refers to necroses that link portal tracts with portal tracts or with terminal hepatic venules; most observers reserve this term for linkage of portal tracts to terminal venules (10). The degree of lobular damage can be semiquantitated and determines, in concert with the periportal inflammation, the degree of activity (grade) of the chronic hepatitis (see below).

Fibrosis

Over time, chronic hepatitis leads to progressive fibrosis, which begins in portal areas, extends to periportal zones, and eventually links portal tracts to other portal tracts and to terminal hepatic venules. Portocentral septa are thought by some to be indicative of more advanced stage of fibrosis than portoportal septa (10). Unfortunately, these patterns are sometimes difficult to distinguish. After fibrous septa have formed, regenerative nodules, indicative of cirrhosis, may appear.

Steatosis

With the exception of hepatitis C, in which approximately 70% of cases show fatty change (3), steatosis is uncommon in chronic hepatitis. In chronic hepatitis C, the fatty change is generally of mild degree and nonzonal; Mallory's hyaline is uncommon, and when present it tends to be found in the periportal/periseptal regions, particularly in advanced stages.

Cholestasis

If PBC and PSC are excluded from the chronic hepatitides, the presence of bile is an uncommon feature of chronic hepatitis. In particular, chronic viral hepatitis is rarely cholestatic. A minority of cases with autoimmune hepatitis show cholestasis (5).

Immunohistochemistry

Commercially available antibodies detect hepatitis B surface antigen, hepatitis B care antigen, HDV, and HCV. Antigen detection of HCV in formalin-fixed tissue is technically demanding, however (12); the methods should still be considered research procedures. Reverse transcription double polymerase chain reaction may show more promise in the future (2), particularly if in situ techniques become widely applicable (24). In general, serum testing and histology will be sufficient for reaching an etiologic diagnosis, and immunohistochemical techniques should be reserved for ambiguous cases.

SYSTEMATIC APPROACH TO ANALYSIS AND REPORTING

In the clinical evaluation of chronic hepatitis, biopsy specimens may be obtained for several reasons. In some cases, the main objective is to determine an *etiology* for clinically evident hepatitis or to explain occult laboratory abnormalities. Histologic features may support or exclude the presumed clinical diagnosis or provide evidence of an alternative or superimposed process (e.g., steatohepatitis). In other cases, the etiology has been established clinically, but *grading* of necroinflammatory activity is

required, either in anticipation of potential antiviral or immunosuppressive therapy or to assess the results of prior therapy. Clinical and laboratory assessments may not reflect the degree of observed necroinflammatory activity. In the precirrhotic phases of chronic hepatitis, the *stage* of the disease, that is, the degree of fibrosis, is difficult to predict clinically. Thus, a third subset of patients undergo liver biopsy primarily for the purpose of staging. Early stage disease is more likely to benefit from aggressive treatment with antiviral drugs or immune modulators than late-stage disease.

Complete pathology reports should address all these issues (Tables 1–4). The information can be provided in the diagnosis itself, in the microscopic description, or as a comment. Our group and others (5,10,20) advocate a synoptic approach to reporting that includes the etiology, stage, and grade of the chronic hepatitis in the final diagnosis.

TABLE 3. *Staging of chronic hepatitis*

Staging Terminology		
Semiquantitative	Descriptive	Criteria
0	No fibrosis	Normal connective tissue
1	Portal fibrosis	Fibrous portal expansion
2	Periportal fibrosis	Periportal or rare portal-portal septa
3	Septal fibrosis	Fibrous septa with arthitectural distortion; no obvious cirrhosis
4	Cirrhosis	Cirrhosis

Etiology

The etiologic portion of the final diagnosis should generally contain one of the diseases listed in Table 1. Whenever possible, the morphology should be correlated with clinical history, serologic studies, serum liver enzyme activities, and tests of liver function to reach an etiologic diagnosis. Special histochemical or immunohistochemical stains, in situ hybridization, or polymerase chain reaction techniques may be necessary in some circumstances. If such information is unavailable and no specific morphologic features are appreciated, a descriptive morphologic diagnosis such as that illustrated in Fig. 1 should be used, provided a comment about possible etiologies is also included. For instance, if no specific etiology can be inferred from the histology and if clinical information is lacking, "periportal hepatitis, etiology undetermined" is a reasonable diagnosis. Some additional examples are shown in Table 4. In contrast, the term cryptogenic chronic hepatitis should be used when thorough clinical and laboratory investigation fails to reveal a likely etiology and specific morphologic features are not seen.

Grade

Grading is a measure of the severity of the necroinflammatory process; the old terms CPH, CAH, and CLH essentially represent a grading (but not staging) system (10). The histological activity index (HAI) of Knodell et al. (18) is a detailed grading system that has been in existence for over a decade and has been widely used in the context of grading in therapeutic trials. In this system, numerical values for periportal/bridging necrosis (0–10), intralobular necrosis (0–4), portal inflammation (0–4), and fibrosis (0–4) are added to obtain a score. It should be noted in this context that only the first three categories reflect disease activity; the last category reflects stage. An updated version was recently published (15).

Simplified schemes for grading disease activity

TABLE 2. *Grading of disease activity in chronic hepatitis*[a]

Grading Terminology		Criteria	
Semiquantitative	Descriptive	Lymphocytic piecemeal necrosis	Lobular inflammation and necrosis
0	Portal inflammation only; no activity	None	None
1	Minimal	Minimal, patchy	Minimal; occasional spotty necrosis
2	Mild	Mild; involving some or all portal tracts	Mild; little hepatocellular damage
3	Moderate	Moderate; involving all portal tracts	Moderate; with noticeable hepatocellular change
4	Severe	Severe; may have bridging fibrosis	Severe; with prominent diffuse hepatocellular damage

[a] When a discrepancy exists between criteria, the more severe lesion should determine the grade.

TABLE 4. *Sample reporting options for chronic hepatitis*

Clinical and laboratory information available
Chronic hepatitis C, mildly active, with bridging fibrosis (or stage 3)
Chronic hepatitis B, severely active, with periportal fibrosis (or stage 2)
Autoimmune hepatitis, minimally active, with cirrhosis (or stage 4)
Primary biliary cirrhosis, stage 2
Clinical and laboratory information unavailable
Portal hepatitis, cause undetermined, without fibrosis
Lobular hepatitis (drug-induced? unresolved viral?)

have been outlined (10,14,20,27) and may be more appropriate in daily patient care settings. We recommend a synthesis of these schemes as outlined in Table 2 and illustrated in Fig. 2. Either semiquantitative numeric values or descriptive terms can be used, depending on local preferences. The main determinants of activity are lymphocytic piecemeal necrosis and lobular necroinflammatory activity rather than intensity of portal inflammation; however, portal inflammation tends to parallel the former. When a discrepancy exists between piecemeal and lobular necrosis, the more severe lesion should determine the grade. Grading should be applied to chronic viral and autoimmune hepatitis, but it is of little use in biliary diseases and other types of chronic hepatitis. In some practices, clinicians prefer use of the HAI of Knodell et al. (8). With this system, only the first three categories should be used, resulting in a 0–18 scale (10).

Stage

The stage of chronic hepatitis refers to the degree of fibrosis subsequent to necroinflammatory insults. In our experience, reliable staging requires connective tissue stains, such as Masson's trichrome stain, because with hematoxylin and eosin stains the stage of the process is often underestimated. Detailed staging systems have been proposed (6), but they seem more applicable to scientific studies than to daily practice. A practical scheme that represents a modification of other proposals (10,18,27) is described in Table 3 and illustrated in Fig. 3. In stage 2, rare portal–portal septa may be seen, but numerous septa leading to a subjective distortion of architecture are indicative of stage 3 disease. Bridging fibrosis with nodular regeneration is indicative of cirrhosis.

Practical Applications

When mild inflammation is present and confined to the portal areas, that is, portal hepatitis or "triaditis," the diagnosis of chronicity may rest entirely on the clinical information. It may be difficult to distinguish between a true chronic hepatitis versus

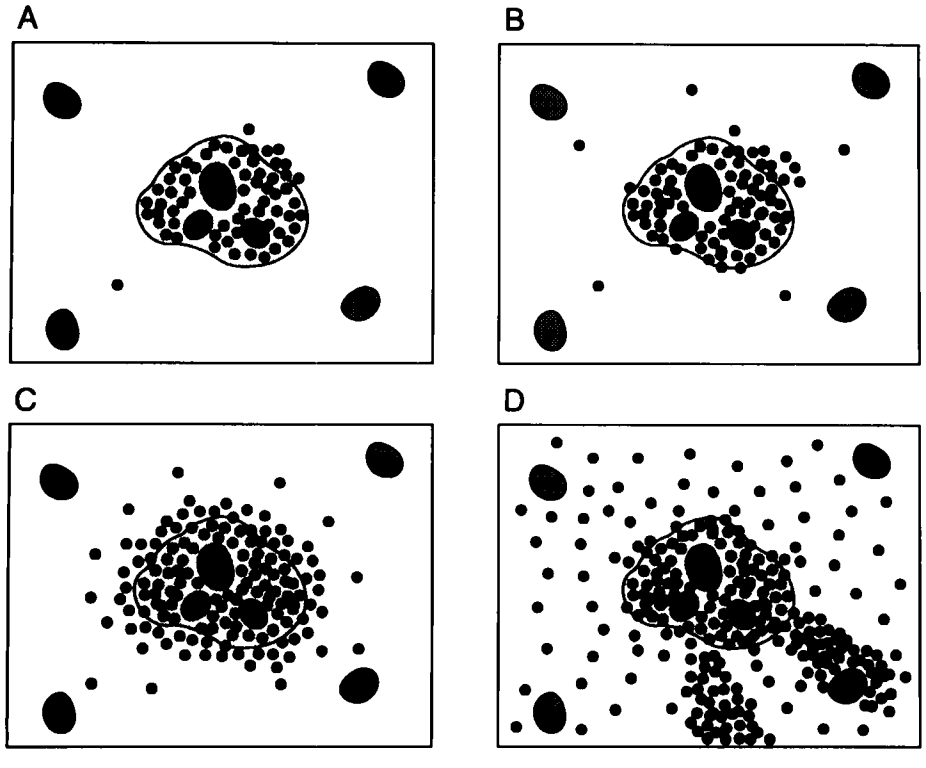

FIG. 2. Grading of chronic hepatitis, schematic diagram. **A:** Minimal activity (grade 1) with mild portal inflammation but scant piecemeal necrosis and no lobular necrosis. **B:** Mild activity (grade 2) with mild portal inflammation, piecemeal necrosis, and scant lobular spotty necrosis. **C:** Moderate activity (grade 3) with moderate portal inflammation, piecemeal necrosis, and lobular spotty necrosis. **D:** Severe activity (grade 4) with marked portal inflammation, brisk piecemeal necrosis, considerable spotty necrosis, and areas of confluent necrosis resulting in bridging. When discrepancies exist between piecemeal and lobular necrosis, the more severe lesion should determine the grade.

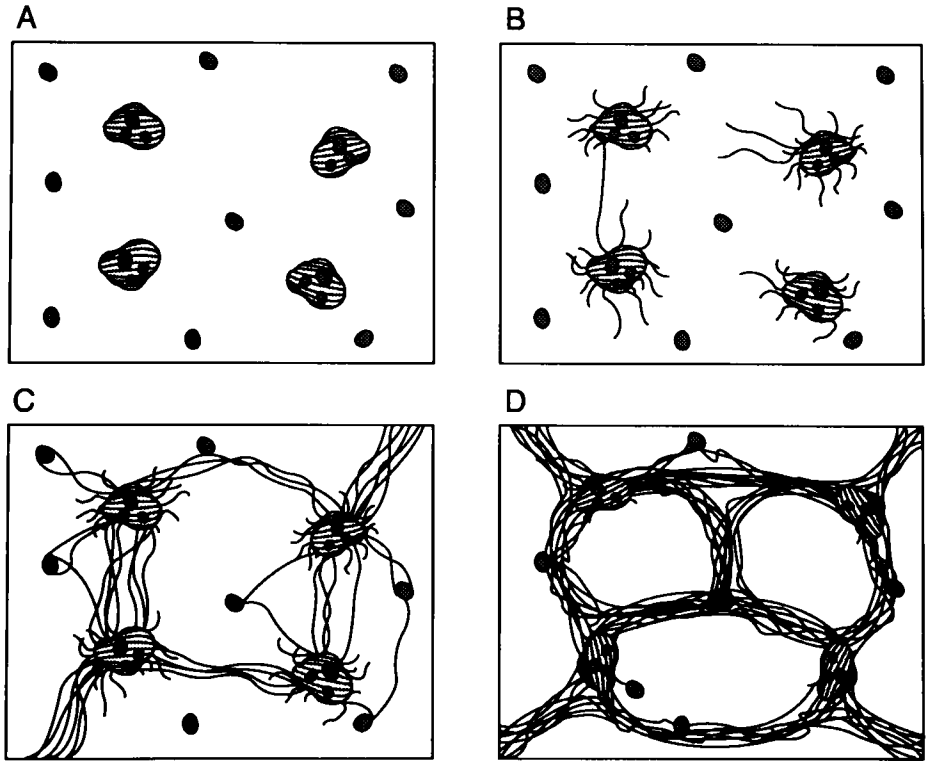

FIG. 3. Staging of chronic hepatitis, schematic diagram. **A:** Portal fibrosis (stage 1) characterized by mild fibrous expansion of portal tracts. **B:** Periportal fibrosis (stage 2) showing fine strands of connective tissue in zone 1 with only rare portal–portal septa. **C:** Septal fibrosis (stage 3) manifested by connective tissue bridges that link portal tracts with other portal tracts and central veins, minimally distorted architecture, but no regenerative nodules. **D:** Cirrhosis (stage 4) showing bridging fibrosis and nodular regeneration.

the nonspecific inflammation that may accompany a variety of systemic conditions or be in the vicinity of mass lesions. If the inflammation is accompanied by lymphocytic piecemeal necrosis or significant lobular inflammation, chronic hepatitis can be suspected. Furthermore, if significant fibrosis is present (stage 2 or), chronic hepatitis or chronic biliary disease is likely present. In the absence of the above features, the term nonspecific reactive hepatitis is reasonable.

Some sample diagnoses shown in Table 4 are based on etiology, grade, and stage, as described in Tables 1, 2, and 3. Note that for biliary diseases (PBC, PSC, autoimmune cholangitis) the principle of diagnosing a stage is fairly well established, but grade is not considered relevant. Likewise, for Wilson's disease and A1AT deficiency, etiology and stage are clearly important, but grade is of questionable relevance and perhaps best dealt with in a descriptive comment. Parenthetically, steatohepatitis can also be reported by stating etiology, grade, and stage.

Whereas many of the traditional terms used to describe chronic hepatitis and cirrhosis have become obsolete (Table 5), for a transitional period, it might be prudent to place the terms CAH, CPH, and CLH in parentheses to ease introduction of the improved terminology. As advances in the understanding of chronic liver disease continue, the terminology will undoubtedly continue to evolve. □

TABLE 5. *Chronic hepatitis and cirrhosis: obsolete terms*[a]

Chronic hepatitis and related conditions
Chronic active hepatitis, chronic aggressive hepatitis, chronic active liver disease, plasma cell hepatitis, lupoid hepatitis, and other synonyms for autoimmune hepatitis
Chronic persistent hepatitis
Chronic lobular hepatitis
Chronic nonsuppurative destructive cholangitis
Pericholangitis
Cirrhosis
Portal cirrhosis
Postnecrotic cirrhosis
Posthepatitic cirrhosis
Posthepatitic cirrhosis
Laënnec's cirrhosis
Nutritional cirrhosis

[a] Modified from ref. 13.

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