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甲方： 北京大学第三医院

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电话: 18910192576

乙方：上海于诚医药科技有限公司

联系人： 李欣梅

地址：上海市中山西路 2020 号华宜大厦 1 号楼 8 楼

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Classification and Grading of Gastritis: The Updated Sydney System

Dixon, Michael F. M.D., F.R.C.Path; **Genta, Robert M. M.D.**; **Yardley, John H. M.D.**; **Correa, Pelayo M.D.**; the Participants in the International Workshop on the Histopathology of Gastritis, Houston 1994

单位：

From the University of Leeds, England (M.F.D.); VAMC and Baylor College of Medicine, Houston, Texas (R.M.G.); Johns Hopkins University School of Medicine, Baltimore, Maryland (J.H.Y.); Louisiana State University Medical Center, New Orleans, Louisiana (P.C.), and the participants in the International Workshop on the Histopathology of Gastritis, Houston, Texas, September 1994: Kenneth P. Batts, Rochester, Minnesota, U.S.A.; Beverly B. Dahms, Cleveland, Ohio, U.S.A.; M. Isabel Filipe, London, England; Rodger C. Haggitt, Seattle, Washington, U.S.A.; Jules Haot, Brussels, Belgium; P.K. Hui, Hong Kong; Juan Lechago, Houston, Texas; Klaus Lewin, Los Angeles, California, U.S.A.; Johan A. Offerhaus, Amsterdam, The Netherlands; Ashley B. Price, Harrow, UK; Sixto Recavarren, Lima, Peru; Robert H. Riddell, Hamilton, Ontario, Canada; Pentti Sipponen, Helsinki, Finland; Enrico Solcia, Pavia, Italy; Manfred Stolte, Bayreuth, Germany; Hidenobu Watanabe, Niigata, Japan.

Address correspndence and reprint requests to Dr. Robert M. Genta, Pathology 113, VAMC, 2002 Holcombe Blvd., Houston, TX 77030, U.S.A.

This article represents a *consensus* of opinions expressed by members of the Houston Gastritis Workshop as interpreted by the primary authors. Individual members do not necessarily subscribe to all the opinions contained in the article.

Abstract

The Sydney System for the classification of gastritis emphasized the importance of combining topographical, morphological, and etiological information into a schema that would help to generate reproducible and clinically useful diagnoses. To reappraise the Sydney System 4 years after its introduction, a group of gastrointestinal pathologists from various parts of the world met in Houston, Texas, in September 1994. The aims of the workshop were (a) to establish an agreed terminology of gastritis; (b) to identify, define, and attempt to resolve some of the problems associated with the Sydney System. This article introduces the Sydney System as it was revised at the Houston Gastritis Workshop and represents the consensus of the participants. Overall, the principles and grading of the Sydney System were only slightly modified, the grading being aided by the provision of a visual analogue scale. The terminology of the final classification has been improved to emphasize the distinction between the atrophic and nonatrophic stomach; the names used for each entity were selected because they are generally acceptable to both pathologists and gastroenterologists. In addition to the main categories and atrophic and nonatrophic gastritis, the special or distinctive forms are described and their respective diagnostic criteria are provided. The article includes practical guidelines for optimal biopsy sampling of the stomach, for the use of the visual analogue scales for grading the histopathologic features, and for the formulation of a comprehensive standardized diagnosis. A glossary of gastritis-related terms as used in this article is provided.

Gastritis has a broad histopathological and topographical spectrum and leads to patterns of disease that have been well recognized and characterized. Although pathologists have generally agreed on the morphological aspects of the lesions they observed, the great variety of names used to define the different disease patterns has resulted in considerable confusion. Thus, many of the controversies and disagreements on the different types and pattern of gastritis may have been caused more by semantics than by real differences of opinion on the biology of disease.

The discovery of *Helicobacter pylori* totally altered our concepts of etiology, as it has become apparent that infection with this organism is the major cause of nonautoimmune chronic gastritis. Furthermore, investigations of gastritis prompted by the discovery of *H. pylori* have led to the recognition of other distinctive forms, such as lymphocytic and reflux gastritis. To take account of these developments and in an attempt to remove diagnostic confusion, a working party met before the World Congress of Gastroenterology in Sydney in 1990 to establish guidelines for the classification and grading of gastritis. The resulting “Sydney System” had both endoscopic and histological divisions, but only the latter will receive attention in this report. The histological arm emphasized the importance of combining topographical, morphological, and etiological information into a schema that would help generate reproducible and clinically useful diagnoses (70,78).

To reappraise the Sydney System 4 years after its introduction, a group of pathologists from various parts of the world with an interest in gastric pathology met in Houston, Texas, in September 1994. The aims of the workshop were twofold: first, to establish an agreed terminology of gastritis; and, second, to identify, define, and attempt to resolve some of the problems associated with the Sydney System. Overall, the general principles and grading as originally proposed in the Sydney System were retained, but grading has been aided by the provision of a visual analogue scale. Furthermore, the terminology of the final classification has been improved to emphasize the distinction between the atrophic and nonatrophic stomach.

This article introduces the Sydney System as it was revised at the Houston Gastritis Workshop. The reader may find it helpful to refer to the original description of the histological arm of the Sydney System (78). The present article, however, can be used independently as a practical guide to the revised classification and grading.

Readers are reminded that the recommendations made in this article reflect the consensus reached by the workshop participants. A consensus is ultimately a collection of opinions, which in this case are based on the collective experience of 20 gastrointestinal pathologists from various parts of the world. Individual physicians will have to decide to what extent they want to adhere to these recommendations and how they want to adapt them to the social, economic, and medical realities of the populations they serve.

CLASSIFICATION

The proposed revised terminology is based on the nosological patterns of gastritis. The names used for each entity were selected because they are generally acceptable to gastrointestinal pathologists; however, no name or definition can be universally acceptable, and we recognize that with some of our choices we may unintentionally rekindle old controversies or generate new ones.

The spectrum of gastritis encompasses several groups of nosological entities that for convenience can be divided into three broad categories: acute, chronic, and special (or "distinctive") forms. The main concern of this report is the assessment of chronic gastritis. Acute gastritis and the special forms of chronic gastritis are covered in lesser detail in later sections herein. Vascular gastropathies are briefly discussed. Whereas the recognition of epithelial dysplasia (82,83) and the early lesions of mucosa-associated lymphoid tissue (MALT) lymphoma (56) in the context of chronic gastritis are of great importance, an extensive discussion of these topics was not considered central to the main objectives of a workshop devoted to the classification, definition, and clinicopathological correlates of gastritis.

Table 1 presents the updated classification of chronic gastritis still based on topography, morphology, and etiology. Gastritis is separated into two major categories based on the presence or absence and topographic distribution of atrophy (Fig. 1). In addition, several well-characterized special forms are also enumerated. When known, possible etiologies are mentioned. It should be noted, however, that some causative agents, such as *H. pylori*, may play a role in more than one type of gastritis. It should also be stressed that an individual patient may have histopathological evidence of more than one type of gastritis, probably as a result of being exposed to more than one etiological agent. A common example of such a situation is the simultaneous presence of gastritis due to chronic ingestion of nonsteroidal anti-inflammatory drugs (*chemical gastritis*) and chronic active gastritis associated with *H. pylori* in the same patient (79). Finally, it is important to realize that, although it is possible to establish the etiopathogenesis of gastritis in most patients, in some cases the pathologist can identify only nonspecific types and patterns of inflammatory and other changes in their gastric biopsies. These patients' condition must be categorized as *unclassifiable* or *type indeterminate*.

Type of gastritis	Etiologic factors	Gastritis synonyms
Nonatrophic	*Helicobacter pylori* ?Other factors	Superficial Diffuse antral gastritis (DAG) Chronic antral gastritis (CAG) Interstitial—follicular Hypersecretory Type B[a]
Atrophic		
Autoimmune	Autoimmunity	Type A[a] Diffuse Corporal Pernicious anemia-associated
Multifocal atrophic	*Helicobacter pylori* Dietary ?Environmental factors	Type B,[a] type AB[a] Environmental Metaplastic
Special forms		
Chemical[b]	Chemical irritation Bile NSAIDs ? Other agents	Reactive Reflux NSAID Type C[a]
Radiation	Radiation injury	
Lymphocytic	Idiopathic? Immune mechanisms Gluten Drug (ticlopidine) ? *H. pylori*	Varioliform (endoscopic) Celiac disease-associated
Noninfectious granulomatous	Crohn's disease Sarcoidosis Wegener's granulomatosis and other vasculitides Foreign substances Idiopathic	Isolated granulomatous
Eosinophilic	Food sensitivity ? Other allergies	Allergic
Other infectious gastritides	Bacteria (other than *H. pylori*) Viruses Fungi Parasites	Phlegmonous

NSAIDs, nonsteroidal anti-inflammatory drugs.
[a] Alphabetic designations of gastritis were abandoned in the original presentation of the Sydney System. That approach is also recommended here. Use of "Type B" to denote either atrophic or non-atrophic gastritis is considered to be especially misleading.
[b] Many participants favor substitution of *gastropathy* for *gastritis* to describe conditions that result from chemical injury.

TABLE 1. *Classification of chronic gastritis based on topography morphology and etiology*

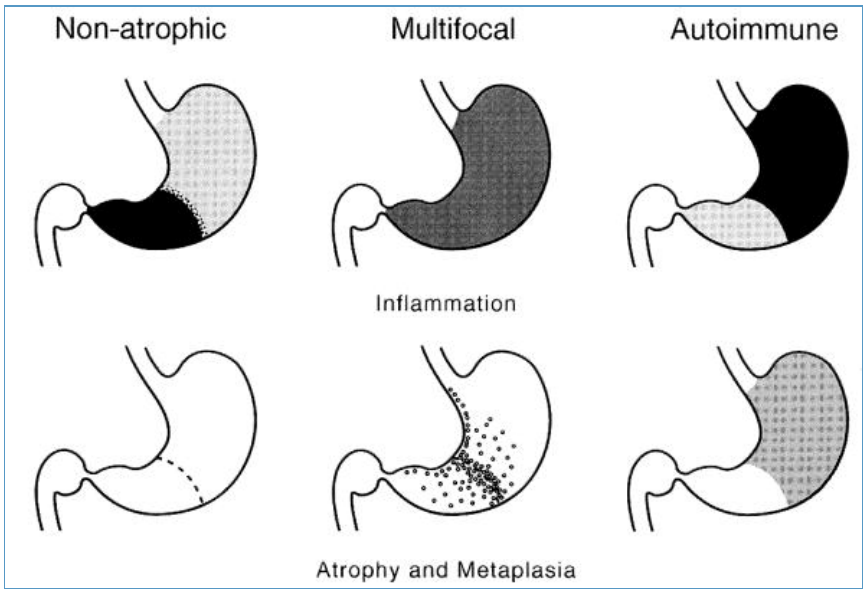


FIG. 1. Schematic representation of the distribution of inflammation and atrophy in different types of atrophic and nonatrophic chronic gastritis. In nonatrophic *Helicobacter pylori* gastritis, inflammation is either predominantly antral or almost uniformly distributed in antrum and corpus (upper left), and there is no significant atrophy (lower left). In atrophic *H. pylori* gastritis, inflammation is usually less intense and similar in the antrum and corpus (upper center); patches of atrophy with intestinal metaplasia arise initially in the area of the incisura angularis and in the transitional zone and may expand proximally and distally to form confluent patches of atrophic metaplastic mucosa (lower center). In autoimmune gastritis, both inflammation (upper right) and atrophy (lower right) are virtually restricted to the corpus.

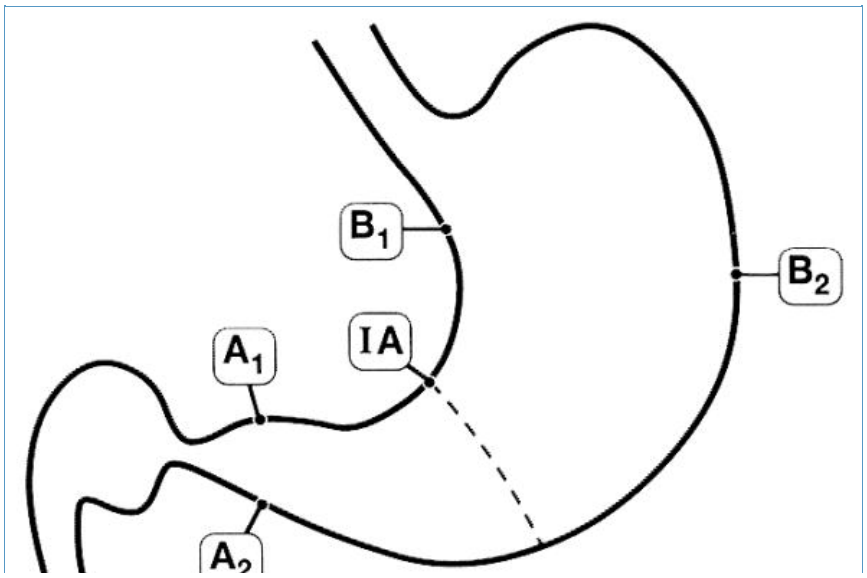
PRACTICAL CONSIDERATIONS

Biopsy Sites

Mapping studies in which multiple biopsy specimens have been taken from *H. pylori*-positive subjects confirm that the careful examination of four specimens (two antral and two corpus) has a high probability of establishing the correct *H. pylori* status (4,36). Corpus biopsies are particularly valuable for yielding positive results after treatment, especially where proton pump inhibitors have been used (54,98). Under these circumstances, organisms may become rare or disappear from the antrum but remain in the oxyntic mucosa, which may also develop cystic dilatations with hypertrophy of the parietal cells (100). Furthermore, biopsy specimens from the corpus are essential to establish the pattern of gastritis, which has important implications for the risk of associated diseases.

Whereas biopsy specimens from the antrum and corpus are adequate, therefore, to establish both the *H. pylori* status and the "background" level and distribution of gastritis, it is self-evident that additional biopsy specimens are required from any lesions that may be present and for mapping the distribution and extent of intestinal metaplasia and dysplasia. However, maximal degrees of atrophy and intestinal metaplasia are consistently found in the region of the incisura angularis (96,99,106), which is also the site most likely to reveal premalignant dysplasia (15,82). Thus, specimens from the antrum and corpus should be supplemented with a fifth or more biopsy specimens from the incisura (Fig. 2). The choice of additional sites from which biopsy specimens may be

taken is dependent on the local epidemiologic conditions with respect to the types of gastritis and the incidence of gastric carcinoma.



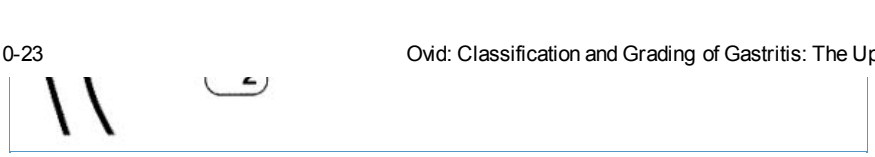


FIG. 2. Schematic representation of the recommended biopsy sites. One specimen each should be obtained from the lesser (A_1) and the greater curvature (A_2) of the antrum, both within 2 to 3 cm from the pylorus; from the lesser curvature of the corpus about 4 cm proximal to the angulus (B_1); from the middle portion of the greater curvature of the corpus, approximately 8 cm from the cardia (B_2); and one from the incisura angularis (IA).

Biopsy specimens from the three regions should be separately identifiable when submitted to the laboratory. Proper orientation is indispensable for optimal histological evaluation, but it is difficult to achieve with small pinch biopsies. It may be accomplished with larger biopsies either in the endoscopy suite when biopsy specimens are collected or in the histopathology laboratory at the time of embedding.

Communication Between the Endoscopist and Pathologist

Full and accurate clinicopathologic correlation in gastritis is consistently achievable only when the pathologist is aware of biopsy locations and of relevant endoscopic and clinical observations. Endoscopic information should include a brief narrative or a diagram describing focal lesions (e.g., thickened folds, polyps, masses, erosions, or ulcers) and abnormal-looking areas. Specific questions (e.g., Is there atrophy, intestinal metaplasia, ectopic mucosa?) may also help to alert the pathologist about the possibility of a process that might not be searched for otherwise. Endoscopic pictures forwarded warded to the pathologist can be particularly helpful. Clinical information furnished to the pathologist should include the medications taken by the patients within the last 2 months, particularly nonsteroidal anti-inflammatory drugs, proton pump inhibitors, antibiotics, and nonprescription antiacids. Other pertinent aspects of the medical history should also be included.

Special Stains

In addition to hematoxylin and eosin (H & E), many laboratories routinely undertake a special stain for *H. pylori*. This practice encourages proper assessment and may be more cost effective than subsequent requests for an extra stain. The choice of stain, for example, modified Giemsa, Warthin-Starry, or the new Genta stain (40), is a matter of local preference, but the use of a special stain is strongly recommended, particularly when H & E fails to reveal organisms in a biopsy specimen with chronic active inflammation. Immunostains are also available for the demonstration of *H. pylori* (8) and may be particularly useful in detecting coccoid forms (12). Thus, although many positive cases can be recognized in a good H & E stain, careful examination of a special stain is essential before declaring an inflamed biopsy specimen histologically negative for *H. pylori*. Many laboratories routinely employ an Alcian-blue (AB, pH 2.5)/periodic acid Schiff (PAS) stain on gastric specimens to demonstrate intestinal metaplasia.

Recommendations

A. For optimal assessment, five biopsy specimens are taken, two from the antrum within 2 to 3 cm from the pylorus, one from the distal lesser curvature, and the other from the distal greater curvature, two from the corpus about 8 cm from the cardia (one from the lesser and the other from the greater curvature), and one from the incisura angularis (Fig. 2).

B. Samples from antrum, corpus, and incisura angularis should be separately identifiable.

C. Transmission of information to the pathologist about the patient's endoscopic findings, clinical history, and biopsy sites is essential for successful clinicopathologic correlation in gastritis.

D. A special stain for *H. pylori* should be carried out before declaring an inflamed biopsy specimen negative.

E. An AB/PAS stain will facilitate the recognition of intestinal metaplasia.

GRADING THE MORPHOLOGICAL VARIABLES

The real value of classifying chronic gastritis lies in the relationship between certain patterns of inflammation and atrophy and their disease associations. In some instances, these patterns are sufficiently consistent for them to be predictive of peptic ulcer or gastric cancer risk. To translate the histopathological observations into well-defined topographic patterns or for comparison purposes, it is highly desirable to grade each relevant feature using a standardized reproducible scale. When the grading guidelines laid down in the Sydney System were applied by a small number of observers, moderately satisfactory levels of agreement were achieved (1). In an attempt to improve the agreement among different observers, at the Houston meeting the textual explanation of the concepts of *mild*, *moderate*, and *severe* (now replaced with *marked*, which has a more quantitative

connotation) was reduced to a minimum. Instead, a visual analog scale was provided to assist in grading. A study using an early version of the visual analogue scale showed excellent interobserver agreement for the density of *H. pylori*, moderate agreement for the intensity of active inflammation and poor agreement for the degree of atrophy (28). Since then, a new visual analog scale for the graded variables and a set of guidelines for its application have been designed (Fig. 3). Unpublished tests of this scale have shown extremely good agreement among observers in all variables, including atrophy; however, this too will require validation by rigorous interobserver studies on level of agreement.

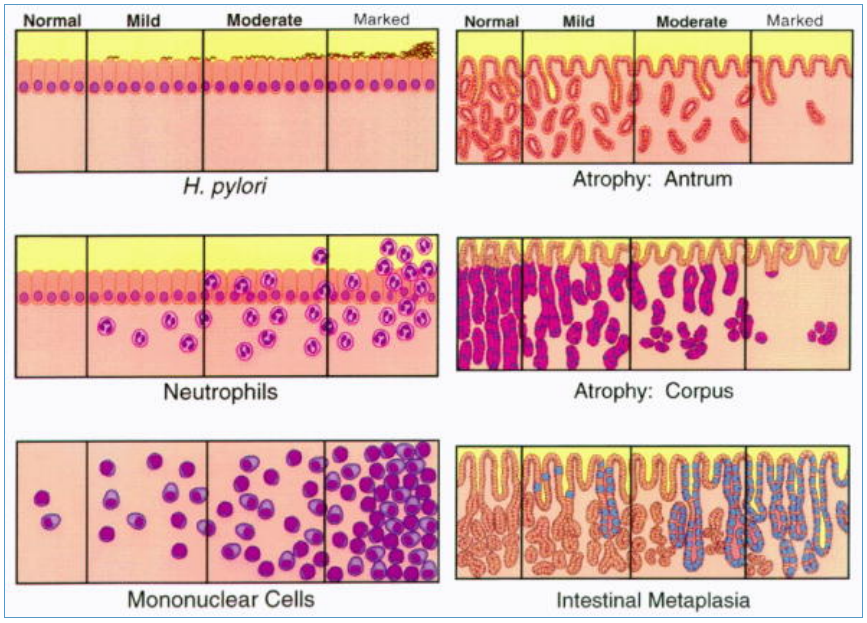


FIG. 3. Using the visual analogue scales: The observer should attempt to evaluate one feature at the time. The most prevalent appearance on each side should be matched with the graded panel that resembles it most closely. Observers should keep in mind that these drawings are not intended to represent realistically the histopathological appearance of the gastric mucosa; rather, they provide a schematic representation of the magnitude of each feature and, as such, have certain limitations. Thus, for example, the decreasing thickness of the mucosa usually observed with increasing atrophy is not depicted realistically. Particularly with *Helicobacter pylori* and neutrophils, there may be a considerable variation of intensity within the same biopsy sample; in such cases, the observer should attempt to average the different areas and score the specimen accordingly.

Grading the morphological variables is useful in the evaluation of *H. pylori* gastritis and its related forms (nonatrophic and atrophic gastritides in Table 1). Although an assessment of the intensity of the inflammatory components may be useful in some of the special forms (e.g., non-*H. pylori* infectious and lymphocytic gastritis), a formal report of the graded variables is not considered necessary when reporting chemical, radiation, granulomatous, or eosinophilic gastritis.

Graded Variables

H. pylori *density*

For clinical management purposes, the most important information is whether *Helicobacter* is present. Variations in *H. pylori* density may have a bearing on disease associations and have epidemiological importance (18,104,109). Difficulties applying the grading guidelines are particularly apparent in biopsy specimens in which portions of the gastric mucosa show intestinal metaplasia, which is usually not colonized by *H. pylori*. Observers have been uncertain about whether to grade the bacterial density on the gastric epithelium alone (ignoring the presence of the adjacent metaplasia) or to average the density of the bacteria over the length of the entire

specimen. We recommend the first approach, which limits the evaluation of *H. pylori* density to the areas where it normally resides.

Polymorphonuclear neutrophil activity

Notwithstanding the problems surrounding the term *activity*, it was considered worthwhile to retain it, as in chronic *active* gastritis, and to grade its severity. In this context, it provides a useful short-hand for "the presence of neutrophil polymorphs in a background of chronic inflammation." As such, it is a measure of continuing acute inflammation; given the likely role of neutrophil-derived reactive oxygen species and proteases (18), neutrophil "activity" is likely to be linked to tissue damage. Chronic inflammation in the absence of neutrophils is also "active" in the sense that cytotoxic T-lymphocytes and other cell effectors may play a role in tissue damage, and operate in glandular destruction in some patterns of gastritis. Neutrophil activity is an almost universal phenomenon in *H. pylori* gastritis. Biopsy specimens contain neutrophils in virtually all cases of *H. pylori*-positive cases if a sufficient number from both antrum and corpus is examined. Neutrophils may be seen in the lamina propria, within the epithelium (particularly in the region of the glandular neck), and within the foveolar lumen, where they may form "pit abscesses." The density of intraepithelial neutrophils has been correlated with the extent of mucosal damage and with the intensity of *H. pylori* infection (31,102). Neutrophils are a very sensitive indicator of the presence or absence of *H. pylori* and disappear within days of cure of infection. If neutrophilic polymorphs are seen in a post-treatment biopsy but organisms are not apparent, a careful search for *Helicobacter* using one of the special stains or immunostains should be carried out (45).

Variable numbers of eosinophils infiltrate the lamina propria in most types of gastritis, but their pathogenetic role is unknown (69). Therefore, routine grading of eosinophils is not required. Increased numbers of eosinophils are frequently seen in biopsy specimens from patients successfully treated for *H. pylori* gastritis; however, their density rarely approaches that found in eosinophilic gastritis (see below).

Chronic inflammation

The normal gastric mucosa contains only individual scattered chronic inflammatory cells (mononuclear cells) in the lamina propria. Thus, any increase indicates chronic gastritis. In *H. pylori* infection, the cellular infiltrate contains effectors of the immune response, including CD4+ and CD8+ T-lymphocytes, B-lymphocytes, plasma cells, monocytes, mast cells, and eosinophils.

A few mononuclear leukocytes are always present in the lamina propria of the gastric mucosa; however, a precise definition of chronic inflammation is hampered by lack of a universal standard for the quantity of mononuclear inflammatory cells in the normal mucosa. The latter can be heavily influenced by geographic location and other demographic variables of the persons studied and by observers' subjective impressions. From a pragmatic standpoint, it may be useful to think in terms of an "expected" rather than a "normal" level of chronic inflammatory cell infiltration.

Despite these limitations, the normal number of gastric mucosal mononuclear leukocytes in the lamina propria is viewed as a maximum of 2 to 5 lymphocytes, plasma cells and macrophages per highpower (×40 objective) microscopic field or, by another approach, two or three lymphocytes or plasma cells between foveolae (the area in which chronic inflammatory cells are most often found). Plasma cells are sparse or absent from the stomach of healthy persons; so their presence is an especially important indicator of a chronic inflammatory response. Some observers consider chronic inflammation to be present even when there are as few as one or two plasma cells per high-power field.

Occasional lymphocytes may also be observed in the epithelium of the normal stomach, especially in the surface (up to about 5 per 100 epithelial nuclei); when increased above that number, they also constitute chronic inflammation (see section on Lymphocytic Gastritis).

Grading the density of the chronic inflammatory cell infiltration provides a baseline measure with which to compare patient groups before treatment and with which to monitor the long-term effects of antibacterial therapy. Chronic inflammatory cells have been shown to be slow to disappear after eradication of *H. pylori*, and may take a year or more to fall to "normal" levels (39,97,113). Some researchers believe that even several years after cure the gastric mucosa, particularly in the antrum, remains infiltrated by a greater than normal number of chronic inflammatory cells. The density of mononuclear cells in the lamina propria should be graded in areas away from lymphoid follicles and their surrounding marginal zone of small lymphocytes.

Glandular atrophy

Atrophy of the gastric mucosa is defined as loss of glandular tissue. Atrophy leads to thinning of the mucosa and is a common denominator in all pathological processes, causing severe mucosal damage. Thus, the loss of glands may follow erosion or ulceration of the mucosa with destruction of the glandular layer or result from a prolonged inflammatory process where individual glands undergo destruction in a "piecemeal" fashion. When this loss occurs, it may be followed by fibrous replacement or by a collapse of the existing supporting matrix.

Recognition of minor degrees of atrophy in the antrum is difficult because of the greater amount of connective tissue normally present in this compartment and the more irregular distribution of the pits and glands

compared with the oxyntic mucosa. A useful way to gauge antral atrophy is the demonstration that the three to four gland cross sections that normally span the lower antral mucosa are reduced to two or fewer cross sections. Replacement of antral epithelium by intestinal metaplasia adds to the microscopic impression of atrophy and can be a useful indicator that atrophy is present, but the metaplasia itself is an independent process. Separation of

glands by chronic inflammation and fibrosis may make it difficult to recognize antral gland atrophy. Atrophy in the oxyntic mucosa is closely linked to loss of acid secretion (111) and to the development of intestinal metaplasia, which in turn is linked to an increased risk of gastric cancer (14,87-89). Extensive atrophy in antral mucosa, usually associated with intestinal metaplasia, also carries an increased risk of malignancy (90). Atrophy can be found in the absence of intestinal metaplasia, particularly in autoimmune gastritis, where there may be diffuse atrophy and pseudopyloric metaplasia in the corpus with minimal or no intestinal metaplasia. Atrophy should therefore be evaluated and graded independent of metaplastic changes.

Intestinal metaplasia

Intestinal metaplasia is common in chronic gastritis of all causes and increases in prevalence with disease duration. Metaplastic epithelium can be recognized morphologically by the presence of goblet cells, absorptive cells, and cells resembling colonocytes or by its enzyme or mucin content. Intestinal metaplasia has been categorized on the basis of morphology and enzyme histochemistry into *small intestinal* and *colonic* types or *complete* and *incomplete* forms and using mucin histochemistry into three main types according to its morphology and glycoprotein content (29,89). Acidic glycoproteins in metaplastic cells are best demonstrated with the AB/PAS technique at pH 2.5 when they stain blue or purple, in contrast to the Schiff-positive neutral mucins present in the surface and foveolar epithelium and the mucous glands of the nonmetaplastic gastric mucosa. In type I (which corresponds to *complete,* see glossary) goblet cells containing sialomucins are interspersed between nonsecretory absorptive cells with well-delineated brush borders. In type II, sialomucin-containing goblet cells are scattered among gastric-type cells containing either neutral mucin or sialomucins; type III is characterized by tortuous and branched crypts lined by tall columnar cells containing abundant sulfomucins with smaller numbers of goblet cells containing either sialomucins or sulfomucins. Sulfomucins are differentiated from sialomucins by staining with high iron diamine (HID)/AB using the appropriate concentration of ferric chloride and carefully controlled conditions (57).

Intestinal metaplasia is generally regarded as a condition that predisposes to malignancy. Complete (type I) metaplasia is believed to carry the lowest risk of gastric cancer, whereas those forms of metaplasia with *large intestinal* characteristics (type III metaplasia or incomplete forms) have been closely linked to carcinoma (81,108). A follow-up study in Slovenia reveals that compared with individuals with types I and II sulfomucin-negative IM, subjects with type III IM have a 2.7 to 5.8 times greater risk for the development of gastric cancer (29). Experience from other high-incidence countries, like Colombia and Japan, also point to a possible predictive value of type III metaplasia. At this time, however, the performance of special stains to define the types of intestinal metaplasia and the assessment of gastric cancer risk should be confined to research settings. More data are needed before specific guidelines for sampling strategy, endoscopic intervals, patient eligibility, and cost effectiveness of surveillance can be formulated.

Recommendations

A. The presence or absence of *H. pylori,* chronic inflammation, neutrophil polymorph activity, glandular atrophy, and intestinal metaplasia should be recorded in all cases of gastritis.

B. When present, each of these variables can be graded on a *mild, moderate,* or *marked* scale as indicated in the guidelines.

Other Histological Features (Nongraded Variables)

Surface epithelial damage, mucous depletion, and erosions

These may be striking features in some cases of active *H. pylori* chronic gastritis and might be correlated with type of cytotoxin production and peptic ulcer risk (53); however, neutrophilic polymorphonuclear inflammation usually accompanies epithelial damage and parallels “activity” (30,67); thus, grading of epithelial degeneration is not necessary in routine practice, but the presence of erosions should be noted (102). Because detachment of parts of the surface epithelium commonly occur during handling and processing, pathologists should be cautious in distinguishing this artifact from true erosions, which show fibrin deposition, neutrophilic infiltration, and regenerative changes in the adjacent epithelium. When low-grade inactive chronic gastritis without surface epithelial damage is seen, a search for *Helicobacter heilmannii* may be warranted (105).

Lymphoid follicles

Lymphoid aggregates with germinal centers are characteristic of chronic *H. pylori* gastritis and a hallmark of this diagnosis. There is sampling error in determining their prevalence, but if sufficient biopsy specimens are examined, they are found in 100% of *H. pylori* positive cases (38,101). Grading a feature whose prevalence is determined by the number of biopsy specimens taken is not indicated; however, the presence of follicles should be noted in the microscopic description. Lymphoid follicles in a *Helicobacter*-negative case suggest that the organisms have been missed (either overlooked or not present because of sampling errors) or that the infection has been cleared (37). If large or irregularly shaped lymphoid follicles are noted or large portions of the mucosa are occupied by a dense population of lymphocytes, the possibility of a mucosa-associated lymphoid tissue (MALT) lymphoma should be considered (56,116).

Foveolar hyperplasia

This condition is recognized by increased length and tortuosity of the foveolae combined with expansion of the proliferative compartment and an increase in nuclear size relative to the mucin-depleted cytoplasm. It arises as either a compensatory response to increased cell exfoliation from the surface or as a response to cytokine stimulation or other inflammatory mediators, such as transforming growth factor alpha (TFG[alpha]) (22). Thus some degree of foveolar hyperplasia may be seen in all forms of gastritis, but it is most pronounced in chemical gastritis (see below).

Pseudopyloric metaplasia

Discriminating between pseudopyloric and true antral glands in a biopsy can at times be of great value for correctly localizing and classifying atrophic gastritis, especially when the site of origin of a gastric biopsy is uncertain. Pseudopyloric glands differ from true "pyloric" (antral) glands in that endocrine cells associated with the metaplastic glands do not include G cells (i.e., cells that immunostain for gastrin), whereas they routinely accompany antral glands. Furthermore, pseudopyloric glands contain both pepsinogens I and II, whereas true antral glands show only pepsinogen II. Pseudopyloric glands in pernicious anemia also contain enterochromaffin-like (ECL) cells, normally present in the oxyntic but absent from the normal antral mucosa (94).

Pancreatic (acinar) metaplasia

Pancreatic acinar-like cells characterized by abundant cytoplasm, acidophilic and finely granular in the apical and middle portions and basophilic in the basal compartment, arranged in nests or lobules among gastric glands have been found in 1 to 2% of gastric biopsy and resection specimens (25). The presence of pancreatic metaplasia is associated with intestinal metaplasia and chronic gastritis; its significance, however, remains unclear.

Endocrine cell hyperplasia

Endocrine cell hyperplasia occurs chiefly as a consequence of functional changes in chronic gastritis. An increase in the number of endocrine cells is most prominent in autoimmune atrophic gastritis. The hypohydria or achlorhydria in that condition leads to G-cell hyperplasia in the antral mucosa and an accompanying rise in circulating gastrin (3). The high gastrin concentrations to which the histamine-producing ECL cells in the oxyntic glands are exposed causes the latter cells to undergo hyperplasia (9,95). Although in severe atrophic gastritis, ECL hyperplasia is often detected on slides stained with H & E, the more precise definition of endocrine cell hyperplasia depends on the use of special stains for argyrophilia (e.g., Grimelius) or immunostains for chromogranin or specific hormones. The ECL cell hyperplasia is typically seen as widespread chains of argyrophilic cells (termed *linear hyperplasia*) within the epithelium of the oxyntic glands or their atrophic or metaplastic replacements. There can also be associated hyperplastic micronodules of argyrophil cells in the lamina propria that are in all likelihood derived from residual endocrine cells within markedly atrophic glands. In a small proportion of patients with autoimmune gastritis, the ECL cell hyperplasia progresses to carcinoid tumor (9,46,52,96).

TOPOGRAPHY OF CHRONIC GASTRITIS

The epidemiologic associations of gastritis are closely related to different topographical patterns of inflammation and atrophy. Most individuals who acquire chronic *H. pylori* gastritis will exhibit more prominent gastritis in the antrum compared with the corpus (one grade higher). In the absence of atrophy and intestinal metaplasia, this mild, diffuse form of chronic gastritis involving both gastric compartments has no clear disease associations, and most affected subjects do not manifest any clinical symptoms. A minority of infected subjects develop marked chronic inflammation in the antrum with only mild inflammation in oxyntic mucosa, an antral-predominant gastritis; a subset of these patients are likely to develop duodenal ulcer. Infrequently, some *H. pylori*-infected individuals (estimated to be 14% in a UK study) (117) show a corpus-predominant pattern that overlaps with autoimmune gastritis. Inflammation restricted to the oxyntic mucosa in an *H. pylori* negative subject that is associated with diffuse glandular atrophy in the corpus is more characteristic of autoimmune gastritis. It remains possible, however, that the pernicious anemia phenotype could still be a long-term consequence of *H. pylori* infection (21). Corpus predominance is also the usual pattern in lymphocytic gastritis.

Atrophy and intestinal metaplasia can be either diffuse or multifocal. The latter is recognized when patchy glandular loss and intestinal metaplasia alternate with areas with a well-preserved glandular layer. Multifocal atrophic gastritis is associated with gastric ulcer disease and, in some populations, with the risk for gastric carcinoma. Whereas diffuse chronic gastritis and multifocal atrophy can be associated with *H. pylori* infection, the pattern may be encountered in *H. pylori* negative cases. A proportion of these will have had previous infection (58,118), but atrophy can result from other injurious factors, such as bile reflux, nonsteroidal anti-inflammatory drugs (NSAIDs), and dietary injury. These and other factors may work independently of, or synergistically with, *H. pylori* to bring about multifocal atrophy.

Recommendations

A. The pattern of chronic gastritis should be assessed and categorized in all cases in which the recommended biopsy specimen from the antrum and corpus are submitted. Most cases show more or less diffuse chronic inflammation, but a small proportion will show a two-grade difference between the antrum and corpus or vice

versa. These cases should be distinguished as *antral predominant* or *corpus predominant*, respectively.

B. The pattern of atrophy or intestinal metaplasia, if present, should be categorized as either *multifocal* or *diffuse*.

C. The pattern of inflammation and atrophy should be used in classifying the gastritis and as an indicator of its etiology.

ETIOLOGY

Irrefutable evidence documents that *H. pylori* is the major cause of gastritis. If the organisms are identified histologically, this finding should be incorporated in the diagnosis. Difficulties arise when biopsy specimens with chronic inflammation do not show *Helicobacter*, but these organisms were present in a previous biopsy, for example, before eradication therapy, or if the result of some other investigation, such as serology or a breath test, indicates that the infection is or has been present. To classify such cases as *idiopathic* would be patently inappropriate; yet a diagnosis of *Helicobacter-associated* based on the results of additional investigations rather than on the finding of organisms in the biopsy specimens could be misleading. Under these circumstances, a pragmatic approach would be to dispense with an etiological term and report the case as *chronic gastritis* without further qualification unless it happened to fall into a distinctive topographical category, in which case that would be appended. Its association with *H. pylori* would then be a matter for clinicopathological correlation. The finding of focal *H. pylori*-negative chronic active gastritis (e.g., only in a single specimen from the corpus) should also alert the pathologist to the possibility of Crohn's disease (19,27,62,76).

Even when *H. pylori* is identified, it must not be assumed that it is the sole etiologic factor. The eventual form of gastritis and its disease associations will reflect the interplay of *H. pylori* with one or more cofactors, which may be host-related (e.g., secretor status, blood group, and immune responses), or environmental (e.g., excessive salt intake and inadequate consumption of fresh fruit and vegetables).

Although it is generally acknowledged that the finding of corpus inflammation and atrophy, with or without metaplasia, in patients with pernicious anemia has an autoimmune basis, a definitive diagnosis of autoimmune gastritis on purely histological grounds is not warranted, especially when dealing with biopsy specimens and their attendant sampling limitations. Similar histologic changes are possible, albeit infrequent, in the late stages of *H. pylori* gastritis and in corpus biopsy specimens from patients with multifocal atrophic gastritis. On the other hand, the histologic changes noted in biopsy specimens and their topography at times can strongly suggest autoimmune gastritis. For instance, autoimmune gastritis should always be considered when corpus predominant atrophic gastritis is seen, particularly when there is little or no atrophy or metaplasia in accompanying antral biopsy specimens. Although *H. pylori* is rarely seen in this pattern (32), serological data suggest a role for the organism (58). (See also comments above about the possible value of endocrine cell and pepsinogen I and II staining in pseudopyloric metaplasia.) Other etiological agents, such as chemicals and infectious organisms, are discussed in the following section.

ACUTE GASTRITIS

The use of the term *acute gastritis* without qualifiers is discouraged because of the persisting confusion between *acute* and *active*. Although only rarely encountered in routine biopsy specimens, two principal categories of acute gastritis are recognized: (a) *acute hemorrhagic* or *erosive gastritis* usually related to acute chemical or irritant injury (64,65); and (b) acute *Helicobacter gastritis* (71,80,91). A third variety, *acute phlegmonous* or *suppurative gastritis*, is a rare and almost invariably fatal condition because of its association with septicemia. Most cases are recognized only at autopsy.

A wide range of substances has been implicated in causing acute damage to the gastric mucosa including alcohol, aspirin, cortisone, phenylbutazone, and a number of other NSAIDs, but a similar clinical picture is seen in patients with shock, for example, after major trauma or surgery, sepsis, burns, and hypothermia. Acute hemorrhagic or erosive gastritis is common, therefore, in patients in intensive care units.

Alcohol and NSAID-associated hemorrhagic gastritis and stress-related acute (hemorrhagic) gastritis initially involve all segments of the stomach; later, antral lesions predominate and duodenal lesions may also develop. Histologically, abnormalities are usually found only in areas immediately adjacent to the lesions. In these areas, the findings are similar whether the underlying cause is hypoperfusion, NSAIDs, or alcohol. In the subepithelial zone, there is diffuse edema of the lamina propria, capillary congestion, and variable degrees of interstitial hemorrhage. Erosions can be sparse or even not demonstrable, as they are often repaired with considerable rapidity, leaving thin, regenerative epithelium as the only evidence of their occurrence. Inflammation is slight or absent, usually consisting of no more than a few neutrophils. Endoscopically normal areas are also histologically normal, and if inflammation is present, it is often caused by *H. pylori* infection (63,65). In fact, because inflammation is typically sparse or absent, *gastropathy* is appropriate as a substitute for *gastritis*, and the former term is favored by some authors (7).

The acute phase of *Helicobacter* infection is rarely encountered in gastric biopsy specimens because the initial illness has trivial symptomatology and goes unnoticed by the patient. Thus, in most cases, the presence of an acute phase is inferred from serological findings. Biopsy specimens reveal marked degenerative changes in the

surface epithelium, including mucus depletion, cellular exfoliation, and syncytial regenerative changes. Neutrophil polymorphonuclear infiltration into foveolar and surface epithelium is conspicuous, and one can find “pit abscesses” and adherent polymorph exudate on the surface. There appears to be relatively equal involvement of antrum and body. The numbers of organisms present can be extremely variable. In the few documented cases of accidental *H. pylori* infection, the corpus element quickly subsides, but the antral element persists (71,80,91). Phlegmonous gastritis is an acute suppurative condition of the stomach in which pyogenic organisms gain access to the submucosa and spread diffusely throughout the organ. The submucosa is thickened by pus formation and edema, which may go on to form a mural abscess, but the mucosa remains intact (17). It is difficult to diagnose in biopsy specimens because the polymorph exudation is centered in the submucosa.

SPECIAL FORMS OF GASTRITIS

These cases are distinctive forms of gastritis in which the characteristic histological appearances of a recognizable etiological agent allows them to be distinguished from the generality of *H. pylori*-associated or nonspecific chronic gastritis.

Chemical or Reactive Gastritis

The diagnosis of chemical or reactive gastritis is indicated by the finding of foveolar hyperplasia, edema, and smooth-muscle proliferation in the lamina propria, together with only normal numbers or a minor increase in chronic inflammatory cells. Unless there is erosion, neutrophilic polymorphonuclear cells are not seen. This histological picture suggests an etiological role for some chemical irritant or drug; indeed, the entity was first recognized in patients who had undergone a partial gastrectomy with a gastroenteric anastomosis and who had bile reflux (74,75,92,93). Thus, the clinical information might point to a *bile-associated chemical gastritis* (*bile reflux gastritis*), whereas a history of NSAID use would indicate a *NSAID-associated chemical gastritis*. Some patients with reactive gastritis abuse alcohol, but in most no specific association can be identified (92). When such changes are detected in the biopsy specimens from a patient without a suggestive history, pathologists should alert the clinician about the possibility of unrecognized NSAID use. Although the Sydney Working party favored *reactive* over *chemical* gastritis, the Houston meeting remained divided on this issue. Some members have a strong preference for `chemical' as the term is more explicit. Likewise, the term *gastropathy* is favored by those who do not consider the mucosal reaction to be inflammatory (2,7). The choice of terminology is not considered crucial. Wider recognition of this histological entity is much more important.

Lymphocytic Gastritis

As the name implies, *lymphocytic gastritis* is characterized by the presence of large numbers of mature lymphocytes infiltrating the surface and foveolar epithelium (50). The increase in intraepithelial lymphocytes can be associated with marked chronic inflammatory cell infiltration of the lamina propria, activity, and focal erosions, or at the other extreme, only a minor increase in chronic inflammatory cells with no activity. The histological picture is readily distinguished from ordinary *H. pylori*-associated chronic gastritis. In the latter one finds four to seven lymphocytes per 100 epithelial cells, whereas 10 times this number can be found in lymphocytic gastritis (23). Most cases have counts between 25 and 40 lymphocytes per 100 epithelial cells, the diagnostic threshold for lymphocytic gastritis being generally taken as being greater than 25 intraepithelial lymphocytes (IELs) per 100 cells. The IELs are almost exclusively T-lymphocytes and the great majority (around 90%) are CD8+ suppressor cells. The condition is frequently associated with the endoscopic entity *varioliform gastritis*, which is characterized by nodular and eroded lesions running along the gastric rugae in the corpus (23,50,51) while in some cases the endoscopic and histological appearances overlap with those of Menetrier's disease (72,115). An association between lymphocytic gastritis and celiac disease is becoming increasingly recognized (20,114).

Granulomatous Gastritis

Granulomatous gastritis, as an unqualified term, is a diagnosis of last resort and should be attached to a case only after numerous alternatives have been considered. Granulomas may be present in the gastric mucosa in Crohn's disease, in sarcoidosis, a variety of infectious diseases (such as tuberculosis, histoplasmosis and anisakiasis), and as a reaction to endogenous and foreign materials (26,27,49). Even when the histological appearances of the granulomas are nonspecific, important clues to the ultimate diagnosis are given by careful evaluation of their distribution and morphology. In rare instances, granulomatous gastritis may be part of an immunemediated vasculitis syndrome (73), an accompaniment of gastric lymphoma (68), or take a xanthogranulomatous form akin to xanthogranulomatous cholecystitis (48). Only after excluding such causes should the diagnosis of isolated granulomatous gastritis be made.

Eosinophilic Gastritis

Eosinophilic gastritis is a rare condition that is thought to be one manifestation of a generalized involvement of the alimentary tract by an allergic reaction, eosinophilic gastroenteritis. The diagnosis should be made with caution. It is not sufficient to see conspicuous numbers of eosinophils; eosinophils must be the dominant cell type with little or no increase in other inflammatory cell types. A marked increase in eosinophils, usually focal, can be seen in some cases of Crohn's disease and parasitic diseases (55) and accompanying some peptic ulcers and carcinomas. Eosinophilic gastritis can be diagnosed with greater confidence when there is a history of allergic disease, such as asthma, food intolerance, and atopic eczema (70% of cases), and there is a peripheral blood eosinophilia (60,84). The stomach is the most common site of involvement and eosinophilic infiltration is maximal in the antrum, but the esophagus and small intestine are also frequently involved (42).

Collagenous Gastritis

This is characterized by a thick band of collagen immediately beneath the surface epithelium of the gastric mucosa (13); however, in a case of collagenous gastroduodenitis associated with collagenous colitis (103) the collagen band in the gastric mucosa was at the level of the foveolar isthmus. Only rare cases of this entity have been described, and its clinical associations remain unclear.

Radiation Gastritis

Gastritis resulting from irradiation of the stomach is uncommon, as is the reactive gastritis that can complicate intra-arterial chemotherapy (16). Early changes consist of necrosis of fundal glands, edema, and mononuclear cell infiltration. In a series of patients followed by serial biopsies there was prompt and complete reversal to normal (41).

Infectious Gastritis

Bacterial

Mycobacteria: In patients with disseminated tuberculosis, necrotizing granulomas identical to those found in other locations may be found in the gastric mucosa (110,112). Another mycobacterium that has gained prominence with the spread of acquired immunodeficiency syndrome (AIDS) is *Mycobacterium avium-intracellulare*. The stomach is rarely involved; when it is, the typical lesions consist of accumulations of foamy histiocytes in the lamina propria, sometimes with formation of illdefined granulomas without necrosis (11). Staining for acid-fast bacilli reveals large numbers of positive microorganisms both within the histiocytes and extracellularly, in the stroma (43).

Treponema pallidum: Like tuberculous gastritis, gastric syphilis was a medical curiosity in the years preceding the AIDS pandemic. Recently, however, an increasing number of cases has been reported in human immunodeficiency virus (HIV)-infected patients (11,59). When associated with secondary syphilis, syphilitic gastritis is characterized by a prominent mixed inflammatory infiltrate consisting predominantly of plasma cells and with mucosal ulcerations (43,47). Mononuclear vasculitis involving larger vessels in the submucosa and muscularis propria is a consistent feature of syphilitic gastritis and suggests the diagnosis. Spirochetes may be seen in sections stained with appropriate silver stains (Dieterle, Steiner, or Warthin-Starry). The infiltrate may be dense enough to cause the swelling of gastric folds, which may also undergo erosion and ulceration, sometimes mimicking the endoscopic appearance of lymphoma or infiltrating carcinoma (5).

Viral

Although many common enteroviruses are believed to infect the gastric mucosa, so few patients with acute gastroenteritis undergo gastric biopsy that no information is available on the morphologic features of these infections. The only viral infections of the stomach with a distinct pathologic appearance are those caused by cytomegalovirus (CMV); CMV gastritis is seen almost exclusively in children and immunocompromised patients. Usually it is associated with concurrent CMV infection of other sites of the digestive tract. Endoscopically, the gastric mucosa may appear completely normal or show erosions or shallow ulcers. Rarely, it may present as a grossly nodular mucosa that has been referred to as a pseudotumor (34). In children, a hypertrophic gastritis similar to Menetrier's disease may develop (61,85). The histopathologic appearance varies depending on the patient's ability to mount an immune response. In some patients, particularly those with very low CD4 counts, numerous CMV inclusions may be seen in epithelial and endothelial cells as well as in macrophages, with little or no inflammatory response in the adjacent tissues. In other patients, one may see a florid mixed inflammatory reaction with abundant granulation tissue, and typical CMV inclusions may be difficult to detect without using immunohistochemistry or in situ hybridization techniques (86). Intense eosinophil infiltration in the lamina propria accompanied by peripheral eosinophilia has been noted, suggesting an allergic component to the gastritis in some patients.

Fungal

Candida species, *Histoplasma capsulatum*, and *Mucoraceae* have been found in the stomach of immunocompromised subjects, particularly AIDS patients, with disseminated infections (11). Endoscopically, gastric candidiasis appears as whitish patches scattered on the mucosa; microscopically, yeast forms are seen lying on and sometimes invading the eroded gastric mucosa. Hyphae of *Candida* spp. may be found at the base of a large portion of gastric ulcers, but they are believed to represent the secondary colonization of preexisting peptic ulcers. Histoplasmosis is diagnosed when the typical intracellular organisms are found within macrophages of the mucosa, which may be grossly intact, eroded, or ulcerated. Gastric invasion by Mucormycosis agents, belonging to the *Phycomycetes* class, is rarely encountered and only in severely debilitated, immunocompromised patients. The typical lesions consist of extensive hemorrhage and necrosis accompanied by an abundant inflammatory infiltrate but without granuloma formation. Although the lesions are primarily mucosal, they can extend deeply into the wall and cause perforation (11,66).

Parasitic

The stomach is not a preferred site for human parasitic infections. *Cryptosporidium* spp. has been found

The stomach is not a preferred site for human parasitic infections. *Cryptosporidium* spp. has been found lining the gastric mucosa, which was virtually free of inflammation (10). Several cases of giardiasis have been reported (24,27), virtually all in stomachs with extensive atrophy and intestinal metaplasia. Because most patients also have *H. pylori* infection, it remains unclear whether *Giardia intestinalis* causes gastritis. *Strongyloides stercoralis* has been found in the stomach of patients with widespread infections. Larvae and, more rarely, adults are buried in the gastric mucosa (35). The responses range from the complete absence of inflammation to granulomas engulfing fragments of parasites. Rarely, widespread mucosal damage, hemorrhage, and necrosis have been described. *Anisakis* larvae ingested by eating raw fish may penetrate and even perforate the gastric wall. In the few surgically resected specimens available for examination during this event, large numbers of eosinophils have been observed at the site of penetration, whereas a granulomatous reaction usually surrounds the parasitic organisms in chronic cases (55).

VASCULAR GASTROPATHIES

Congestion, dilatation of capillaries and focal hemorrhages are commonly found in many biopsy specimens with *H. pylori* gastritis. It is therefore important for the pathologist to be able to distinguish these nonspecific changes of inflammation from primary vascular lesions that are not gastritides per se. These conditions are known as vascular gastropathies and are defined as abnormalities of gastric mucosal vessels with little or no accompanying inflammation. The most important of these conditions, and the only one that can be confidently diagnosed by histopathological examination of biopsy specimens is gastric antral vascular ectasia (“watermelon” stomach), a syndrome frequently associated with gastric atrophy and autoimmune and connective tissue disorders (33,44). Histologically, gastric antral vascular ectasia is characterized by an expansion of the mucosa due to fibromuscular hyperplasia. The lamina propria also contains markedly dilated mucosal capillaries, which are not increased in number but show a significant increase in cross-sectional area (107). In most cases, fibrin thrombi are found within the dilated capillaries. The presence of these thrombi is particularly important for the differentiation from other causes of mucosal congestion. Another vascular gastropathy that may be suspected, but usually not diagnosed, by histopathological examination of biopsy specimens is *portal hypertensive gastropathy* (77), a significant cause of chronic upper gastrointestinal bleeding in patients with portal hypertension. In contrast to watermelon stomach, the histological changes in portal hypertensive gastropathy are more prominent in the proximal stomach. One can observe variable dilatation, irregularity, and tortuosity of the mucosal, and, if present in the specimen, submucosal veins and capillaries. These changes, however, are not specific, and in patients with concurrent *H. pylori* gastritis it is virtually impossible to establish a histopathological diagnosis of portal hypertensive gastropathy.

REPORTING GASTRITIS

A comprehensive diagnosis of gastritis should comment on the presence or absence of the graded variables and an assessment of their extent using the current guidelines. Additionally, the presence of features such as foveolar hyperplasia, lamina propria edema, and smooth-muscle proliferation will be assessed in cases where the final diagnosis is reactive gastritis. Likewise, the presence of erosions, eosinophils, granulomas, and infectious agents (e.g., anisakiasis, giardiasis, and *Helicobacter heilmannii,* formerly *Gastrospirillum hominis*) should be noted.

From the foregoing, it can now be appreciated that the ideal diagnostic phrase with which to conclude a report of inflammatory gastric disease is one that embraces an etiological, a topographical, and a morphological component, for example, *active, antral predominant, chronic H. pylori gastritis,* or *NSAID-associated reactive gastritis*. In this regard, the Houston meeting endorsed the principles of the Sydney System. Sometimes, although the etiology is not necessarily established, it may be inferred from the morphological findings, as in *corpusrestricted chronic gastritis* and *atrophy* (*autoimmune type*). In many cases, however, the cause remains unknown or is only revealed by other investigations; and to preface each diagnostic phrase with “idiopathic” would be self-defeating and misleading. Under these circumstances, the morphological or topographical classification will suffice. For example, the diagnosis of *chronic gastritis with multifocal atrophy* (or *multifocal atrophic gastritis*), which carries implications for the risk of progression to cancer, is not enhanced by the addition of *idiopathic*. If, on the other hand, *Helicobacter* is identified, chronic *H. pylori gastritis with multifocal atrophy* or *Helicobacter-associated multifocal atrophic gastritis* is much more helpful. Several practical examples of how to formulate a diagnosis of gastritis by applying this system to different combinations of topographical and histopathological patterns are presented in the Appendix.

PROGNOSTIC IMPLICATIONS

The diagnosis of gastritis rests on a synthesis of the morphological and topographical findings. These findings are interpreted in the light of possible etiological factors to generate a clinically relevant opinion providing prognostically useful information with regard to the likely disease associations and outcomes. Pathologists should exert great caution when expressing prognostic opinions, paying particular attention to the epidemiological associations that may be peculiar to the area where they practice or the population they serve. For example, the prognostic implications of a diagnosis of multifocal atrophic gastritis in areas with high gastric cancer risk (e.g., certain regions of South America or Eastern Asia) may differ substantially from those in an area where gastric cancer is uncommon (e.g., North America). Such facts should always be kept in mind to help avoid exaggerated responses from clinicians and to minimize the possibility of inappropriate interpretations in countries where medical litigation is commonplace.

CONCLUSIONS

Classifications are not right or wrong; they cannot even be said to be good or bad except in relation to a purpose. The most that can be said about them is that they are useful or not useful (6). We do not know how useful these revisions of the Sydney System will prove to be. The next step is to test reproducibility of our proposed grading and the interobserver agreement on the final diagnoses. Only when the system has been shown to be reproducible and to have biological significance can it be claimed that this is a useful approach to gastric biopsy interpretation.

APPENDIX

Using the visual analogue scales: The observer should attempt to evaluate one feature at a time. The most prevalent appearance on each slide should be matched with the graded panel that resembles it most closely. Observers should keep in mind that these drawings are not intended to represent realistically the histopathological appearance of the gastric mucosa; rather, they provide a schematic representation of the magnitude of each feature and, as such, have certain limitations. Thus, for example, the decreasing thickness of the mucosa usually observed with increasing atrophy is not depicted realistically. Particularly with *Helicobacter pylori* and neutrophils, there may be a considerable variation of intensity within the same biopsy sample; in such cases, the observer should attempt to average the different areas and score the specimen accordingly.

How to formulate the diagnosis: These guidelines assume that at least two biopsy specimens from the antrum and two from the corpus are available. When a fifth specimen from the incisura angularis is available, it should generally be treated as an additional antral specimen and its scores averaged with the antral scores. If more prominent intestinal metaplasia or dysplasia is found in the specimen from the incisura, an appropriate note should be included in the diagnosis.

In some instances, fewer than two antral and two corpus biopsies will be available. Both the systematic approach to the evaluation of gastric biopsies and the grading of histological variables described here may be usefully applied to individual biopsy specimens; however, because the topographic arm of the Sydney System is founded on the concept that adequate sampling from both regions of the stomach is obtained, we would discourage pathologists from issuing a topographic diagnosis based on the examination of fewer than the recommended number of specimens. We also recommend that, particularly in scientific articles, the sentence “Gastric biopsies were evaluated according to the Sydney System” be used only when the system is applied in its intact form.

1. After examination of the paired biopsy specimens an “average” grade is given for each histological feature in that gastric compartment. In a research setting, it may be appropriate to score the graded variables in individual biopsies and calculate the average for each compartment.

2. If *H. Pylori* is detected in any of the specimens, the words *H. pylori* should be included in the diagnostic phrase. Absence of *H. pylori* in a case with chronic gastritis should be specifically mentioned.

3. If a two-point or greater difference exists between the graded inflammatory variables of antrum and corpus, a topographical qualifier (*antrum*- or *corpus*-predominant or restricted) should follow the words *active chronic gastritis*. If there is neither antrum nor corpus predominance, the unqualified term *active chronic gastritis* will be sufficient. This substitutes *pangastritis* used in the original Sydney System.

4. If present, intestinal metaplasia or atrophy should be mentioned with respective grading after the term *gastritis*. As there are no generally accepted criteria to diagnose multifocal atrophic gastritis, the use of the term is a matter of individual choice. If more than one site is affected by at least moderate atrophy or metaplasia, the term *focal* may be used.

5. In routine practice, there is rarely the need to report the grading of each histological feature; however, in treatment protocols or other research settings, it may be useful to add a list of the scores as a comment or a descriptive phrase. Alternatively, a matrix such as the used in the following examples may be used.

6. If the specimens clearly show a “special form” of gastritis, the diagnostic phrase should refer only to it (e.g., *lymphocytic gastritis*). Because of the uncertain role of *H. pylori* in some of these entities (lymphocytic gastritis, granulomatous gastritis), it is recommended that its presence or absence be mentioned. Grading of individual histological features may be included in a comment.

Example 1, Example 2, Example 3, Example 4, Example 5

Site	*H. pylori* colonization	Neutrophils	Mononuclear cells	Atrophy	Intestinal metaplasia
Antrum	Moderate	Moderate	Marked	Absent	Absent
Corpus	Mild	Absent	Mild	Absent	Absent

Diagnosis: *H. pylori*-chronic gastritis, active, antrum-predominant.

Example 1: *Patient with duodenal ulcer*

Site	*H. pylori* colonization	Neutrophils	Mononuclear cells	Atrophy	Intestinal metaplasia
Antrum	Marked	Moderate	Marked	Mild	Moderate
Corpus	Moderate	Mild	Moderate	Mild	Mild

Diagnosis: *H. pylori*-chronic gastritis, active, with focal atrophy and intestinal metaplasia. Alternatively, this could be diagnosed as *H. pylori*-associated multifocal atrophic gastritis

could be diagnosed as *H. pylori*-associated multifocal atrophic gastritis.

Example 2: *Asymptomatic volunteer with positive serology for* H. pylori

Site	*H. pylori* colonization	Neutrophils	Mononuclear cells	Atrophy	Intestinal metaplasia
Antrum	Absent	Absent	Absent	Absent	Absent
Corpus	Absent	Absent	Moderate	Marked	Moderate

Diagnosis: Corpus-restricted chronic atrophic gastritis, probably autoimmune. No *H. pylori* is identified.

Example 3: *Patient with suspected pernicious anemia*

Site	*H. pylori* colonization	Neutrophils	Mononuclear cells	Atrophy	Intestinal metaplasia
Antrum	Absent	Absent	Moderate	Absent	Absent
Corpus	Absent	Absent	Mild	Absent	Absent

Diagnosis: Chronic gastritis, inactive. No *H. pylori* is identified.
Comment: The histopathological features of these biopsy specimens are indicative of successfully treated *H. pylori* gastritis.

Example 4: *Patient treated for* H. pylori *infection 3 months before*

Site	*H. pylori* colonization	Neutrophils	Mononuclear cells	Atrophy	Intestinal metaplasia
Antrum	Absent	Marked	Moderate	Mild	Moderate
Corpus	Absent	Mild	Mild	Mild	Mild

Diagnosis: Chronic gastritis, active, with focal atrophy and intestinal metaplasia.
Comment: Although no organisms can be identified in any of the biopsy specimens examined, the prominent neutrophilic infiltrate on a background of chronic atrophic gastritis strongly suggests that *H. pylori* infection is present and ancillary diagnostic tests (urea breath test, serology) may be indicated. If these tests are negative, the possibility of Crohn's disease should be considered.

Example 5: *Patient with gastric ulcer*

GLOSSARY OF GASTRITIS-RELATED TERMS

Note: The terms defined here (identified by CAPITAL LETTERS) are those that are most relevant to description and classification of gastritis. The definitions given are in accordance with generally accepted usage and, when appropriate, illustrate how they are used in this article. Liberal cross-referencing is included (also shown in CAPITAL LETTERS).

ACTIVE INFLAMMATION (Synonyms: active chronic inflammation; chronic active inflammation.)

Polymorphonuclear neutrophil infiltration (PMN) occurring in the context of chronic inflammation. See also: ACUTE INFLAMMATION; CHRONIC ACTIVE GASTRITIS; CHRONIC INFLAMMATION; INACTIVE INFLAMMATION; INFLAMMATION.

ACUTE INFLAMMATION The presence of extravascular polymorphonuclear neutrophils in the absence of concurrent chronic inflammation. Acute inflammation in the gastric mucosa is most readily demonstrated as epithelial PMN infiltration, which is never a normal finding. See also: ACTIVE INFLAMMATION, GASTRITIS, and INFLAMMATION.

ACUTE GASTRITIS See GASTRITIS.

ANTRUM (Synonyms: pyloric antrum.) The distal ¼ to 1/3 of the stomach, extending distally from the corpus to the pyloric channel and duodenum distally. The antrum is lined by ANTRAL MUCOSA. See also: PYLORUS: TRANSITIONAL ZONE.

ANTRAL MUCOSA (Synonym: pyloric mucosa.) The type of mucosa found in the gastric antrum. Its characteristic feature is presence of coiled and branching antral glands. These are lined by mucus cells that are interspersed with endocrine cells (chiefly G and D types), and a few parietal cells. The latter are most numerous proximally near the oxyntoantral border. The foveolar component is taller in the antral mucosa than in the oxyntic mucosa. See also: PYLORUS

ATROPHY Taken literally, atrophy means *wasting*, with resulting reduction in size or amount of an organ, tissue, or cell type. Atrophy of the stomach affects primarily the glands, which become sparse and small. In atrophy of the gastric corpus and fundus, parietal and chief cells disappear from the oxyntic glands, and the residual glands may undergo pseudopyloric metaplasia. Mucosal atrophy can be noted grossly, especially in advanced, diffuse atrophy of oxyntic mucosa in the fundus and corpus where rugae disappear and underlying blood vessels become visible. On the other hand, mucosal thinning is often grossly inapparent in the antrum, probably because its overall thickness is preserved by accompanying inflammation, intestinal metaplasia, or foveolar hyperplasia. Atrophy is often associated with intestinal metaplasia, but these two features may occur independently of each other. In pernicious anemia (corpus-predominant atrophic gastritis), there may be little or no intestinal metaplasia in large areas of the atrophic stomach; in contrast, scattered goblet cells may be found in the surface and foveolar epithelium of many nonatrophic *H. pylori*-infected stomachs. When metaplastic epithelium replaces the specialized epithelium of the mucous glands in the antrum or oxyntic glands in the corpus, however, there is actual loss of functional glandular tissue, and therefore true atrophy. In such an instance, atrophy and intestinal metaplasia coincide.

A distinctive form of gastric atrophy occurs in patients who undergo subtotal gastrectomy (antrectomy)

A distinctive form of gastric atrophy occurs in patients who undergo subtotal gastrectomy (antrectomy). Since there is reduced gastrin stimulation, parietal cells diminish in number in the residual stomach and the oxyntic mucosa becomes thinned. See also: INTESTINAL METAPLASIA, MULTIFOCAL ATROPHIC GASTRITIS, and Text, section 4 (iv).

BODY See CORPUS.

BODY MUCOSA See OXYNTIC MUCOSA.

CARDIAC MUCOSA The type of mucosa that lines the region of the cardia. It has antral-like glands, but unlike antral mucosa, there are no G cells. Scattered oxyntic glands may be present in the most distal portions of the cardia where it merges with the corpus.

CARDIA The most proximal section of the stomach. The cardia begins at the gastroesophageal junction (normally identifiable as the Z-line) and continues for one to a few centimeters distally to the cardio-oxyntic transitional zone. Histologically, it is lined by CARDIAC MUCOSA.

CHRONIC ACTIVE GASTRITIS Inflammation of the stomach characterized by the simultaneous presence of a mononuclear cell infiltrate and neutrophilic polymorphonuclear inflammation. Because this term may convey an entity (like chronic active hepatitis) rather than a phase of a single disease, it is preferable to use the term *active* as a descriptor, as in the original Sydney System (e.g., chronic gastritis, active). See also: ACTIVE INFLAMMATION: ACUTE INFLAMMATION: INACTIVE INFLAMMATION: GASTRITIS.

CHRONIC INFLAMMATION Inflammation in which mononuclear leukocytes, including lymphocytes, plasma cells, and macrophages (monocytes) predominate. Occasional small lymphoid aggregates without germinal centers are to be expected in the normal stomach, especially at the base of the oxyntic mucosa, but presence of germinal centers is prima facie evidence of chronic inflammation. Prominent numbers of eosinophils and mast cells, and fibrocytes with fibrosis when there is tissue repair, are often included in the overall picture of chronic inflammation. See also: INFLAMMATION and text section.

CHRONIC GASTRITIS See GASTRITIS.

CORPUS (CORPORAL) MUCOSA See OXYNTIC MUCOSA and CORPUS.

CORPUS The proximal 2/3 to ¾ of the stomach lying between the cardia and fundus proximally and the antrum distally. In the undistended state, rugae are observed grossly, and oxyntic mucosa is seen histologically. See also: RUGAE; TRANSITIONAL ZONE.

DIFFUSE ANTRAL GASTRITIS (DAG) (Synonym: antrum-predominant gastritis, nonatrophic.) Chronic gastritis characterized by a mononuclear infiltrate in the lamina propria of antrum and antrum-corpus junction without loss of gastric glands (hence nonatrophic). The corpus may show mild perifoveolar (superficial) inflammation.

EOSINOPHILIC INFLAMMATION (Synonym: eosinophilic gastritis.) Excessive infiltration by eosinophilic leukocytes. Some observers consider the presence of any eosinophils in gastric mucosa to be abnormal, but most believe that a few eosinophils may be present in the normal lamina propria. On the other hand, intraepithelial eosinophils, which usually occur in the context of generalized mucosal eosinophilia, are always viewed as abnormal.

FOLLICULAR GASTRITIS (Synonym: *Helicobacter*-associated gastritis.) Chronic gastritis in which there are large numbers of prominent lymphoid follicles in addition to the mononuclear infiltrate. Because *H. pylori* infection is also regularly present, it is considered the underlying cause of follicular gastritis.

FOVEOLAE (Singular: FOVEOLA) (Synonym: gastric pits.) The mucous cell-lined channels in the upper mucosa that open onto the surface and to which the gastric glands connect from below. The foveolae and their associated mucus-secreting surface epithelium appear similar everywhere in the stomach. They are longer in the antrum than in the corpus. See also: HYPERPLASIA.

FOVEOLAR HYPERPLASIA Increased length and tortuosity of the foveolae combined with expansion of the proliferative compartment and an increase in nuclear size relative to the mucin-depleted cytoplasm. Thus some degree of foveolar hyperplasia may be seen in all forms of gastritis but it is most pronounced in chemical gastritis.

FUNDIC (FUNDAL) MUCOSA See OXYNTIC MUCOSA and FUNDUS.

FUNDUS The dome-like subdiaphragmatic portion of stomach that lies above an imaginary horizontal line passing through the gastroesophageal junction. Like the more distal gastric corpus, the fundus shows rugae and is lined by oxyntic mucosa.

GASTRITIS Literally, inflammation of the stomach. *Acute gastritis* and *chronic gastritis* are sometimes used as alternative terms for acute inflammation and chronic inflammation respectively, but they carry the potential disadvantage of being misinterpreted as denoting specific nosologic entities or as indicating clinical duration of a patient's illness. Consult the text for descriptions and classification of gastritis subtypes. See also: INFLAMMATION and GASTROPATHY.

GASTROPATHY Literally, any type of gastric disease. The term is sometimes used in lieu of *gastritis* for nonneoplastic conditions in which inflammation is minimal or absent (e.g., *hemorrhagic gastropathy, chemical gastropathy*).

GLANDS, GASTRIC The principal secretory structures of the mucosa, occupying its lower portion and connecting to foveolae. The morphology, cell types, and functions of gastric glands vary between regions (cardia, corpus/fundus, and antrum). See also: ANTRAL MUCOSA; CARDIA; FOVEOLAE; OXYNTIC MUCOSA.

HYPERPLASIA Increased size of a tissue or component cell type that results from an increase in total number of cells. *Endocrine cell hyperplasia* and *foveolar hyperplasia* are especially relevant to gastritis. See also: ATROPHY.

INACTIVE INFLAMMATION (Synonym: Inactive chronic inflammation.) Term is used at times to describe chronic inflammation that is *not* accompanied by acute inflammation. See also: ACTIVE INFLAMMATION; CHRONIC INFLAMMATION; INFLAMMATION.

INCISURA ANGULARIS (Synonyms: Angulus, incisura, gastric notch; angular notch.) The notch-like indentation of the gastric wall on the lesser curvature. It is located at about the junction of the proximal 2/3 and distal 1/3 of the stomach. It also approximately marks the junction of the gastric antrum and corpus. See also: TRANSITIONAL ZONE (MUCOSA).

INFLAMMATION Excessive tissue infiltration by leukocytes. See also: ACTIVE INFLAMMATION; ACUTE INFLAMMATION; CHRONIC INFLAMMATION; EOSINOPHILIC INFLAMMATION; GASTRITIS; INACTIVE INFLAMMATION.

INTESTINAL METAPLASIA Replacement of glandular and/or foveolar epithelium by intestinal epithelium. This is the commonest type of metaplasia found in the stomach. Depending on the cell types that are present and the types of mucins being secreted, intestinal metaplasia takes several forms: (a) complete, or small intestinal, or type I; (b) incomplete, or colonic, or types II and III. See also: METAPLASIA and text section.

METAPLASIA The presence in a tissue of cells normally found only in other locations. Intestinal, pseudopyloric, and pancreatic metaplasia are, in descending order, the most common forms seen in gastritis. See also: ATROPHY; INTESTINAL METAPLASIA; PSEUDOPYLORIC METAPLASIA; PANCREATIC METAPLASIA.

MULTIFOCAL ATROPHIC GASTRITIS (MAG) Chronic gastritis characterized by loss of glands (atrophy) in multiple foci, more abundantly found in the areas of the incisura angularis, the antrum-corpus junction, and the lesser curvature of the antrum. The independent foci may grow to coalesce with age. These foci are usually the site of intestinal metaplasia.

OXYNTIC MUCOSA (Symptoms: fundic (or fundal) mucosa; body mucosa; corpus (or corporal) mucosa; proximal gastric mucosa.) Acid-secreting mucosa (*oxy* = sour or acid). The glands in oxyntic mucosa are straight tubes that constitute HCl-producing parietal cells along with scattered mucus cells in their upper portion and mainly chief (zymogenic) cells in their lower portion. There are also scattered argyrophilic endocrine cells (principal type: ECL cell).

PANCREATIC METAPLASIA Collections of pancreatic acini replacing gastric glands. See also: METAPLASIA.
PANGASTRITIS Term introduced in the original Sydney System report to denote that both the antrum and corpus show involvement. As it can be misunderstood to mean that the gastritis has a diffuse distribution in both compartments, it is not used in this report.

PITS See FOVEOLAE

PSEUDOPYLORIC METAPLASIA Replacement of oxyntic glands by mucus-secreting glands that resemble those seen in the “pylorus” (antrum). Pseudopyloric glands are thought to arise by hyperplasia of surviving mucous neck cells. See also: METAPLASIA and text section (Other histological features).

PYLORIC MUCOSA See PYLORUS; ANTRAL MUCOSA.

PYLORIC METAPLASIA Metaplastic glands that fully replicate those found in the antrum. The term is sometimes also used as a synonym for pseudopyloric metaplasia. See also: METAPLASIA.

PYLORUS Term that can denote either the distal gastric opening and its associated sphincter or the entire distal 3rd of the stomach (hence pyloric antrum, pyloric glands, etc.).

RUGAE Prominent folds located on the corpus and fundus of the stomach. They consist of full-thickness oxyntic mucosa and its underlying submucosa.

SUPERFICIAL GASTRITIS Older term used to describe chronic and often active gastritis that is concentrated in the upper, foveolar portion of the mucosa beginning just below the surface epithelium. In the classification system proposed by Whitehead, superficial gastritis was contrasted with atrophic gastritis. Most cases formerly termed superficial gastritis are due to *H. pylori* and would be equivalent to “Non-atrophic gastritis” in the classification system presented in this report. The term is not used in this report.

TRANSITIONAL ZONE (MUCOSA) (Synonyms: Border zone; intermediate zone; junctional zone.) A general term for regions where two types of mucosae meet. When alluding to a transitional zone, its location should be specified (e.g., cardio-oxyntic, oxyntoantral, antroduodenal). Verification of a transitional zone requires microscopic study since its exact location may not be evident grossly although the landmarks can be helpful. For instance, the oxyntoantral transitional zone, which is frequently noted in gastric biopsy specimens and can be up to several centimeters in width, is generally found near the incisura angularis. Histologically, transitional zones typically show a mixture of the architectural features and cell types found in the two mucosae. [Context Link]

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Key Words: Gastritis; Classification; Diagnosis; Sydney System; Gastroenterology; gTerminology

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影像图库

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Table 1

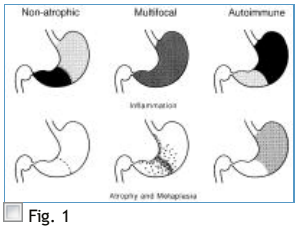
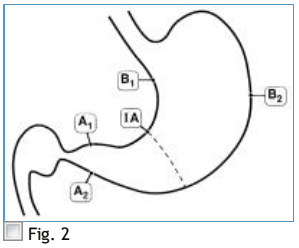


Fig. 1



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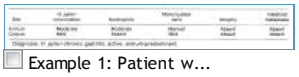
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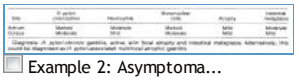
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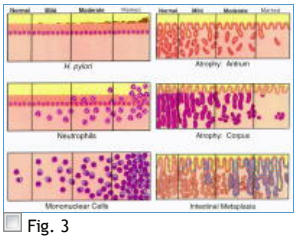
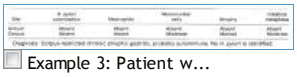


Fig. 3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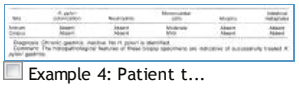
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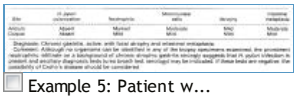
Example 5: Patient w...

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