CLASSIFICATION OF CHRONIC GASTRITIS IN THE POST-SYDNEY ERA: A DISCOURSE

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ABSTRACT

Chronic gastritis is defined histologically as an increase in the number of lymphocytes and plasma cells in the gastric mucosa. The definition and classification of this entity had been a subject of intensive research, long before the discovery of Helicobacter pylori by Warren and Marshal. The Sydney system of classification was introduced at a time when there was a dire need for greater consistency in nomenclature so as to be able to compare clinical studies emanating from pathology reports of gastric biopsies. The system achieved this objective to a large extent but histology reports continued to lack information on the prediction of risk of gastric cancer development. There was a need for clear guidelines on clinico-endoscopic surveillance for gastric cancer.

In an attempt to address this shortcoming, an international group of gastroenterologists and pathologists called Operative Link for Gastritis Assessment (OLGA) proposed a system which grades chronic gastritis according to gastric cancer risk, comparable to the histology reporting format that proved very successful in chronic hepatitis.

Helicobacter pylori is the etiologic agent of chronic gastritis in the vast majority of cases as its worldwide epidemiology overlaps that of chronic gastritis. It is also the most important risk factor for the development of non-cardia gastric cancer, being responsible for almost 90% of such cases worldwide. In gastric cancer development, Helicobacter pylori-induced chronic gastritis is the first step in the so-called "Correa" cascade.

The Kyoto consensus on Helicobacter pylori gastritis is the first global consensus for gastritis and touched on various aspects, including classification, designation of Helicobacter pylori gastritis as an infectious disease, recommendation that all infected subjects be treated regardless of whether they have clinical manifestations or not, separation of Helicobacter pylori gastritis from functional dyspepsia, and the interaction of Helicobacter pylori with other microbiota in the digestive tract.

The Maastricht Consensus on Helicobacter pylori management which was developed by the European Helicobacter Pylori Study Group (EHPSG) has moved from the 1st edition published in 1997 to the 5th which was released in 2015 (Maastricht V). The aspect of this latest version that touched on chronic gastritis is consistent with the provisions of the Kyoto consensus.

In a similar vein, the International Classification of Diseases (ICD) is currently being revised from ICD-10 to ICD-11. The formative stage of ICD-11 (ICD-11 β component) is still being compiled and is expected to capture the issue of etiologic classification of chronic gastritis which was conspicuously absent in ICD-10.

Image-enhanced endoscopy is a fast growing technique in the diagnosis of gastric premalignant lesions. It is expected that in the not too distant future, there would be classification systems for chronic gastritis that would be based on this new technology.

INTRODUCTION

Chronic gastritis has been with mankind for a long time and has actually received its fair share of attention from clinicians, pathologists and researchers, but two events in the development of science and technology marked a turning point in the understanding of this entity. First was the advent of fibreoptic gastrointestinal endoscopy in the 1960s. The period, 1968 to 1990 witnessed an explosion in technological advancements that eventually revolutionized the practice of gastroenterology in general and gastrointestinal endoscopy in particular. This period can rightly be described as the golden era of gastrointestinal endoscopy. The second event was the discovery of Helicobacter pylori in 1982 by Warren and Marshal[1]. This bacterium is now proven beyond doubt to be the etiologic agent of chronic gastritis in the vast majority of cases.

As a direct consequence of these two events, there was a deluge of research efforts by basic scientists, physicians, pathologists, and microbiologists. The result was that there was confusion in nomenclature and classification of chronic gastritis. At the World Congress of Gastroenterology in Sydney in 1990, a new classification system called "The Sydney system" was introduced[2-7]. This was revised 4 years later to produce The Updated Sydney system[8]. The Sydney system combined histological parameters of activity, chronicity, atrophy, intestinal metaplasia, Helicobacter pylori, topographical distribution and aetiopathogenic information for reporting the pathology of gastritis in endoscopic biopsies[4,8].

Despite the robustness of the Sydney system, some shortcomings have been raised especially the lack of any models for the prediction of risk of malignancy in premalignant lesions (atrophy and metaplasia)[9-12]. In an effort to address this challenge the "Operative Link on Gastritis Assessment or OLGA" and "Operative link for gastric intestinal metaplasia assessment or OLGIM" were introduced in 2005[13,14].

Other efforts at updating the classification of chronic gastritis in the post-Sydney era include the Maastricht V/Florence consensus report on the management of Helicobacter pylori infection[15], International classification of diseases and related health problems[16,17] and the Kyoto global consensus report on Helicobacter pylori gastritis[18].

In this review an attempt is made at appraising some of the major classification models that have been developed after the Sydney system.

Synopsis of Pre-Sydney Era

Prior to the use of fibreoptic gastroscopes, there were several efforts to classify chronic gastritis but this review will be concerned with studies that followed the introduction of fibreoptic gastroscopes. The advent of fibreoptic gastroscopes fitted with devices for taking mucosal biopsies has facilitated the study of gastric mucosal lesions. It is now possible to obtain full-thickness gastric mucosal samples that are free from autolysis or operative artefact, from any part of the stomach at the same examination. These factors have facilitated research in chronic gastritis.

In 1972, Whitehead[19] introduced topography into the classification of chronic gastritis and described gastritis involving the antral, fundal, corporal and pyloric regions of the stomach. Morphologically, he also proposed superficial and atrophic gastritis and

for activity (granulocyte infiltration in the epithelium and interstitium) he used the terms active and inactive. Whitehead was also the first to introduce grading of atrophy into mild, moderate and severe; and also introduced the evaluation of metaplasia in the routine histopathological assessment of gastric lesions. It is evident that the foundation and template for what was to later become the Sydney system was laid by Whitehead and some other researchers of his time.

Strickland and Mackay in 1973[20] suggested the inclusion of immunological and etiological information along with topographic and morphological data. They introduced the terms "type A" gastritis and "type B" gastritis for gastric corporal inflammation which mostly corresponds to pernicious anemia (parietal cell antibody positive); and inflammation of gastric antrum. The exact cause of type B was not known but was thought to be related in some cases to bile-reflux. Subsequent resea3moni[21] to describe chronic gastritis in which inflammation extended from corpus to pre-pyloric region of the stomach, and further used type AB+ to describe the same topographical pattern plus associated parietal cell antibody seropositivity.

Correa in 1980[22] subdivided chronic gastritis into autoimmune chronic gastritis with pernicious anemia, hypersecretory gastritis and environmental gastritis. In this schema, the gastritis associated with ulcer was termed hypersecretory and all others outside autoimmune and hypersecretory were termed environmental. Correa published other papers on this subject in which he changed focus from a classification based on etiology to one based on topography. The terms "diffuse antral", "diffuse corporal" and "multifocal" gastritis were used by Correa. In 1988[23] he classified chronic gastritis into "atrophic" and "non-atrophic" gastritis. Also in 1988, Wyatt and Dixon[24] introduced "type C" gastritis for chemical or drug-induced gastritis (Table 1).

Genesis of the Sydney System

From the foregoing, it is evident that the discovery of Helicobacter pylori in 1982 was preceded by ample knowledge about classiûcation of gastritis, its biological course and consequences. However, the discovery was a watershed in the understanding of the etiology of chronic gastritis, and established that the commonest form is that caused by Helicobacter pylori. This exciting discovery attracted many researchers in the field with the result that some confusion in terminology and classification crept into the field. On account of this, a working party was set up in the late 1980s to promulgate a common language applicable to the new knowledge about the biology and natural course of chronic gastritis, provide simple guidelines for the documentation of the microscopic appearances in biopsy specimens together with an easily understandable classiûcation for clinical and research purposes and provide guidelines for the reporting and classiûcation of the endoscopic appearances of the gastric mucosa.

At the World Congress of Gastroenterology in Sydney in 1990, the party presented its work, "The Sydney System: A New Classiûcation of Gastritis" which was subsequently published as six papers in the Journal Gastroenterology and Hepatology[2-7]. These encompassed the pathology, the endoscopic aspects, the microbiology, autoimmunity and epidemiology of chronic gastritis.

The Sydney System

The Sydney system emerged at a very auspicious time as the necessary templates and ingredients for its accomplishment were already on ground. Helicobacter pylori had just been discovered and its role in the causation of majority of cases of chronic gastritis was becoming evident. Furthermore, there was substantial body of existing knowledge in the subject of chronic gastritis. However, the existing knowledge needed to be harmonized in such a way that research findings could be compared. It became necessary to promulgate common language and guidelines to unify the vast

Table 1: Some classification systems of chronic gastritis in the pre-Sydney era

Year	Author/Classification	Highpoints	Reference
1972	Whitehead	1. Topography: antral, fundal, corporal,pyloric 2. Morphology: superficial, atrophic 3. Activity: active, inactive (neutrophilic infiltration) 4. Grading of atrophy: mild, moderate, severe 5. Evaluation for metaplasia	19
1973	Strickland & Mackay	1. Type A (Autoimmune), predominantly corporal 2. Type B (Non-autoimmune), predominantly antral	20
1975	Glass & Pitchumoni	1. Type A (as above) 2. Type B (as above) 3. Type AB (extend from corpus to antrum) 4. Type AB+ (AB plus parietal cell antibody seropositivity)	21
1980	Correa	Autommune, Hypersecretory, or Environmental	22
1988	Correa	1 .Diffuse antral, diffuse corporal, multifocal 2. Atrophic, non-atrophic.	23
1988	Wyatt & Dixon	Type C (for chemical/drug-induced form)	24
1990	Sydney	1. Acute, chronic, special forms 2. Topography: antral, corporal, pangastritis 3. Morphology: inflammation, activity, atrophy, Helicobacter pylori (all graded) 4. Etiology/pathophysiology (prefix)	2-7
1996	Updated Sydney	1. Sydney 2. 5th biopsy from angulus 3. Visual analogue scale for graded variables	8

knowledge in the biology and natural course of chronic gastritis.

The system described the type, severity, extent and where possible the etiology of chronic inflammation. Key features of the system are:

- *Topography:* There were three topographical variants (antral gastritis, corporal gastritis and pangastritis)
- *Etiology:* This was added as a prefix if known, for example, Autoimmune corpus gastritis, Helicobacter pylori antral gastritis.
- *Morphological variables:* There were 5 morphological variables (chronic inflammation, activity, atrophy, intestinal metaplasia and Helicobacter pylori)

Morphological Variables

The morphological variables are graded and are recorded separately for gastric antrum and body. The grading is semi-quantitative and utilizes absent, mild, moderate and severe to denote the degree of severity of each variable. The system is fairly comprehensive and robust and has continued to meet the expectations of clinicians and pathologists to a reasonable extent albeit some shortcomings.

1. ***Chronic inflammation:*** This is represented by the presence of mononuclear cells (lymphocytes, histiocytes and plasma cells) in the lamina propria. The normal mucosa tends to have some mononuclear cells in the lamina

propria such that there is no consensus on what constitutes normal. Usually, the number of mononuclear cells in the lamina propria of the stomach in normal people is affected by geographical location, age, sex and other demographic variables[25]. In general, 2 to 5 mononuclear cells per high power field (x40 objective) or 2 to 3 mononuclear cells between foveolae is considered normal. Chronic inflammatory cells can be used to monitor response to treatment. They are slow to disappear after eradication of Helicobacter pylori.

2. ***Activity:*** This is defined as the presence of coexistent neutrophil infiltration. It is a measure of host reaction against Helicobacter pylori and therefore is a strong marker of progression of the inflammation to atrophy. Neutrophils can be seen in the lamina propria, within the epithelium or within the foveolar lumen, where they form pit abscesses. They disappear within days of eradication of the infection. Variable number of eosinophils infiltrate the lamina propria but are not routinely graded in the Sydney system.
3. ***Glandular atrophy:*** This is defined as loss of glandular tissue as a result of prolonged inflammation. Loss of gastric glandular tissue may be associated with replacement by connective tissue (non-metaplastic atrophy) or it may be replaced by glandular structures inappropriate for location (metaplastic atrophy)[26]. Gastric mucosal atrophy and intestinal metaplasia are definite pre-cancerous lesions because they provide the background for subsequent development of dysplasia and intestinal type adenocarcinoma[27,28].
4. ***Metaplasia (IM):*** This is a phenotypic change from normal gastric mucosal epithelium to an intestinal type. There are 2 main varieties of IM: small intestinal or complete type and colonic type or incomplete type. Complete intestinal metaplasia is marked by presence of goblet cells interspersed among absorptive enterocytes with eosinophilic cytoplasm. These cells express the complete set of digestion enzymes such as sucrase and trehalase. The incomplete variant or colonic type is similar to large bowel phenotype in morphology and mucin expression. It is described as incomplete because the set of digestive enzymes disappear partially or completely. Intestinal metaplasia is generally considered a pre-cancerous lesion but the presence of incomplete metaplasia carries a higher cancer risk[29,32].
5. ***Helicobacter pylori:*** Measurement of density of Helicobacter pylori in gastric mucosal biopsies is an important component of the Sydney system. This organism has been found to be the etiologic agent in the vast majority of cases of chronic gastritis. It may be difficult to detect in cases of extensive intestinal metaplasia or during anti-secretory therapy (proton pump inhibitor or PPI) even with special stains. Though Helicobacter pylori density is a graded variable according to the Sydney schema, semi-quantitative score of bacterial density has limited clinical utility. A distinction between positive or negative Helicobacter pylori status is considered adequate.

In Sydney system, 4 biopsies were recommended; 2 from the antrum (from the greater and lesser curvatures) and 2 from the corpus (from the anterior and posterior walls).

The classification system was criticized by American researchers and that led to a revision of the system at Houston in 1994[8] (Updated Sydney system). However, the updated Sydney system differed from the original version in only 2 respects: inclusion of a 5th gastric biopsy from the angulus in order to enhance the detection of atrophy and IM; and the inclusion of a visual analogue scale for the graded variables. This updated version is still referred to as Sydney system

and is used in many parts of the world by pathologists and gastroenterologists. Its use in every day practice has enhanced evidence-based medicine.

Downsides of Sydney System

The Sydney system is not without limitations. One of the most contentious issues at the Houston meeting was the concept of atrophy. There was no consensus on what constitutes normal glandular pattern in the gastric mucosa. The significance of the topographical distribution of atrophy was not captured in the Sydney system[33]. Furthermore, there was no information on any relationship between atrophy and IM. By far the most significant shortcomings of the Sydney system were the lack of a reporting schema that addresses prognostic information, lack of guideline for instituting surveillance against cancer and lack of information on therapy. These drawbacks began to take center stage, more so, in the light of long-term follow-up studies that confirmed that the extent of gastric mucosal atrophy parallels the risk of gastric cancer[34-38].

Researchers began to draw a comparison between chronic hepatitis and chronic gastritis. The former already had a well described terminology for communication among disciplines and stakeholders that had definite information on disease progression and cancer risk[10-12]. There was thus a need for a system that would provide information on gastric cancer risk stratification and surveillance. It was on this premise that the operative link for gastritis assessment (OLGA) was conceived.

Operative Link for Gastritis Assessment (OLGA)

Despite the incontrovertible neoplastic potential of atrophy and intestinal metaplasia, their presence in a biopsy material is not sufficient to clearly inform the clinician about natural history of gastritis, risk of malignant change and what type of surveillance an individual patient might need. Even when a robust system like the Sydney system is used in the histological evaluation, issues of prognosis and subsequent monitoring are not included.

Helicobacter pylori is the major cause of chronic gastritis. In such situations the rational principle of treatment is eradication of the organism. If the pre-cancerous lesions of the stomach (atrophy and IM) have reached the hypothetical "point of no return", the elimination of Helicobacter pylori would not reduce the future risk of cancer. However, if eradication takes place early in the course of gastric inflammation when there are little or no pre-malignant lesions, then the gastric cancer risk will be reduced[39-43]. The hypothetical "point of no return" is not known with exactitude, once Helicobacter pylori is detected, it is promptly eradicated without any further information to the patient as to whether he runs any cancer risk.

In a bid to get round this challenge, a group of pathologists called Operative Link for Gastritis Assessment (OLGA) came together and proposed a new system in 2005[44,45]. Their effort was greatly facilitated by the robust experience that had been accumulated worldwide using the Sydney system. In the OLGA system, a minimum of 5 biopsy samples is recommended: 2 from the distal antrum from the greater and lesser curvatures, 2 from proximal corpus from the anterior and posterior walls and the 5th from the incisura angularis where the earliest atrophic-metaplastic changes tend to occur[46].

The pathologist should be furnished with adequate clinical and relevant laboratory information about the patient. Laboratory data should include Pepsinogen I (PgI) level, Pepsinogen II (PgII) level, Gastrin-17 (G-17) level and possibly results of serological tests for anti-parietal cell antibodies to help in the diagnosis of autoimmune gastritis. Similar to the updated Sydney system, visual analogue scales are employed to ease classification.

Atrophy is the only variable considered in the OLGA schema. It is scored for each biopsy sample on a four-tiered scale (0-3) as follows:

No atrophy, 0%, score=0; Mild atrophy, 1-30%, score=1; Moderate atrophy, 31-60%, score=2; and Severe atrophy, >60%, score=3. The OLGA stage is determined by combining the scores from antral and corporal biopsy samples (table 2).

Table 2: The OLGA system

		CORPUS			
		No atrophy (grade 0)	Mild atrophy (grade 1)	Moderate atrophy (grade 2)	Severe atrophy grade 3)
ANTRUM	No atrophy (grade 0)	STAGE 0	STAGE I	STAGE II	STAGE II
	Mild atrophy (grade 1)	STAGE I	STAGE I	STAGE II	STAGE III
	Moderate atrophy (grade 2)	STAGE II	STAGE II	STAGE III	STAGE IV
	Severe atrophy (grade 3)	STAGE III	STAGE III	STAGE IV	STAGE IV

Stage 0 gastritis: This is a stage in which there is no atrophy in any of the 5 standard biopsies. Typical examples of this stage are antral-predominant non-atrophic gastritis and non-atrophic pangastritis. Most patients with relatively early Helicobacter pylori gastritis will present in this stage.

Stage 1 gastritis: In this stage, there is mild atrophy in the antrum with normal corpus or mild atrophy in the corpus. This is usually due to Helicobacter pylori but the organism may be difficult to demonstrate if the patient had been on PPI treatment. Most dyspeptic patients present with this picture and would require only eradication of the organism as gastric cancer risk is minimal or absent[47].

Stage 2 gastritis: Patients in this stage have mild to moderate atrophy in various gastric areas. Atrophy of antral mucosa tends to be more common than atrophy of corporal mucosa. There is also moderate corporal-predominant atrophy, moderate antrum-restricted atrophy or mild multi-focal atrophic gastritis. This variant is epidemiologically associated with low gastric cancer risk but with risk of development of duodenal ulcer rather than gastric ulcer[48].

Stage 3 gastritis: In this stage, there is moderate to severe atrophy occurring simultaneously in gastric antrum and body. Multi-focal atrophic gastritis or corpus-restricted atrophic gastritis induced by autoimmunity is a strong differential diagnosis of this pattern. This stage has increased gastric cancer risk[49].

Stage 4 gastritis: This stage is marked by severe atrophy in the whole stomach. Gastric carcinogenesis is directly related to the extent of atrophy. Extensive metaplasia may interfere with the capacity to detect Helicobacter pylori histologically. Patients with OLGA stages 3 and 4 are at increased risk of gastric cancer[50]. Accordingly, endoscopic surveillance programs should be directed at OLGA stages 3 and 4[48,50].

Operative Link for Gastric Intestinal Metaplasia (OLGIM) Assessment

Just like OLGA, OLGIM assessment is carried out in the antrum and body of the stomach. It focuses on intestinal metaplasia and is also staged in a similar way to OLGA[45]. Between OLGA and OLGIM, it is still being debated which of the two systems is more

efficient in estimating cancer risk but both clearly identify stages 3 and 4 as being at higher risk for cancer[51-53].

Management of Pre-cancerous Conditions in the Stomach (MAPS)

With the advent of OLGA/OLGIM system and consequent characterization of patients that would require surveillance, it became imperative to develop recommendations on what actions to take when patients present with such pre-cancerous lesions. A consensus guideline from European Society of Gastrointestinal Endoscopy (ESGE), European Helicobacter Study Group (EHSG), European Society of Pathology (ESP) and The Sociedade Portuguesa de Endoscopia Digestiva (SPED) was put together in 2012[54].

A detailed description of the schema for the management of patients with atrophic gastritis, IM, or gastric epithelial dysplasia is beyond the scope of this review.

Non-invasive Detection of Precancerous Lesions

Pepsinogen I & II: Pepsinogens are the precursors of pepsin. Pepsinogen I (PgI) is produced exclusively by the corpus from the chief cells and mucous neck cells. Pepsinogen II (PgII) is produced by cardiac, pyloric and Brunner glands[55]. Pepsinogen levels are reduced in atrophic gastritis but tends to be increased during inflammation. To avoid this confounding when atrophy coexists with Helicobacter pylori infection, the ratio of PgI to PgII is considered a more accurate marker than PgI alone[56-58]. Different test systems are commercially available and are either ELISA-based or utilize latex agglutination.

Results from both types have reasonable correlation but the absolute values may differ. For this reason, test systems should be validated in regions where they are used. Pepsinogen levels are recommended for detection of atrophy (sensitivity of 66.7-84.6%, and specificity of 73.5-87.1%)[59-62], but not for detection of gastric cancer (sensitivity of 36.8-62.3%)[63-65] as this poor performance might result in missing 50% or more of the cases of gastric cancer if used in population-based screening programs.

Gastrin-17 (G-17): This marker is more specific for antral atrophy. It is usually secreted by antral G-cells. In Europe, there is a test kit (GastroPanel)[66] that combines PgI, PgII, G-17 and Helicobacter pylori IgG antibody. Atrophic gastritis is associated with loss of G-cells and low level of G-17 in plasma in either fasting state or following stimulation with the protein drink, gastrin-releasing peptide (Bombesin), or PPI therapy. Low plasma level of G-17 is either due to advanced antral atrophy (OLGA III or IV) or high intragastric acidity. Either situation is an indication for further investigation with upper gastrointestinal endoscopy (which is an invasive test). The former situation has a high risk for cancer while the latter has a high risk for peptic ulcer (duodenal ulcer) from hyperchlorrydria.

Gastrin-17 level in plasma following stimulation with protein-rich food is considered the best way to evaluate the functional capacity of G-cells, but this is practically difficult in screening programs. For this reason, results with G-17 in screening programs have not been consistent[55,67]**.** A normal healthy stomach mucosa (OLGA stage 0) is expected to have no Helicobacter pylori antibodies in plasma, normal plasma PgI, normal ratio of PgI to PgII and normal G-17, provided there is no history of PPI use.

Emerging screening methods which are at various levels of development include microRNAs[68-71], cancer autoantibodies[72] and volatile markers found in exhaled breath[73].

The Perspective of International Classification of Diseases (ICD)

The International Classification of Diseases (ICD) is the international standard for defining and reporting diseases and health conditions. It provides a global template for the identification of health trends and statistics and facilitates the comparison and sharing

of health information using a common language. The tenth revision of ICD (ICD-10)[16] was endorsed by the WHO at the 43rd general assembly in 1990. This was few years after the discovery of Helicobacter pylori. Obviously preliminary work on the document started before the discovery, and the role of the organism in the causation of gastritis was still a subject of intensive research at that time. Consequently, Helicobacter pylori did not feature in the section on gastritis in ICD-10 as the document dwelt more on macroscopic and histomorphological criteria. Only alcohol featured as an etiological agent.

Since 2007, the need to revise ICD-10 received attention and the process of developing ICD-11 is still on-going. Experts and various stakeholders still have the opportunity to make suggestions. The formative version of ICD-11 (foundation component of ICD-11β)[17] utilized etiological factors in classifying gastritis and accordingly, Helicobacter pylori gastritis was a definite disease entity; just like drug-induced gastritis and autoimmune gastritis. However, the document is still undergoing revision and it is not certain what its final form would be when it is eventually released in 2018.

Kyoto Global Consensus Report on Helicobacter Pylori Gastritis

Helicobacter pylori gastritis is not only the predominant type of gastritis but it is also the most relevant etiologic agent from the standpoint of predisposition to severe gastroduodenal complications[74-76]. The Kyoto consensus meeting took place in 2014 (Table 3) and one of its major objectives was to develop global consensus on classiûcation of chronic gastritis and duodenitis[18]. In that meeting an etiology-based classification schema, similar to what was presented in the ICD-11 β was endorsed. Other issues canvassed by the Kyoto consensus include:

1. Categorization of Helicobacter pylori-induced gastritis according to gastric subsites, because the risks of gastric cancer and peptic ulcer are affected by the patterns of gastritis. Antrum-restricted gastritis leads to hypergastrinemia, hyperchlorrhydria and duodenal ulceration while corpus-predominant gastritis or pangastritis results in gastric acid hyposecretion or achlorrhydria and increased risk of gastric cancer[77-79].
2. Categorization of gastritis according to histology, because of the linear relationship between the risk of malignant change and degree of inflammation/atrophy, thus lending credence to the OLGA/OLGIM schema[80-84].
3. Gastric erosions should be reported separately from gastritis. Even though gastric erosions can be detected in the context of Helicobacter pylori infection, they are more commonly caused by intake of mucosal damaging drugs such as aspirin and non-steroidal anti-inûammatory drugs (NSAIDs)[85,86].

Thy Kyoto consensus is therefore a robust document that tries to harmonize all the perspectives of Helicobacter pylori –induced chronic gastritis. However, the recommendations cannot be extrapolated to the few instances where chronic gastritis is due to causes other than Helicobacter pylori.

Another interesting development in Helicobacter-pylori-induced gastritis is its relationship with dyspepsia. The traditional practice of using upper gastrointestinal endoscopy to separate organic dyspepsia from functional dyspepsia (FD) has been challenged. Helicobacter pylori is an infectious disease that leads to chronic gastritis of varying severity in all infected individuals[87]. Eradication of the organism heals the inflammation[88-91]. It is for this reason that it is now recommended that Helicobacter pylori gastritis has to be excluded before a reliable diagnosis of FD can be made[15].

Image-Enhanced Endoscopy

This is a research frontier in endoscopic detection of pre-cancerous lesions. It is highly probable that other classification systems would be developed

Table 3: Chronic gastritis classification systems and guidelines in the post-Sydney era

Classification system/ Guideline and year	Highlights	Reference(s).
Operative link for gastritis assessment (OLGA)/Operative Link for Gastric Intestinal Metaplasia (OLGIM), **2005**	Evaluates atrophy and metaplasia (premalignant lesions) Useful in cancer risk estimation and surveillance.	44, 45
Management of pre-cancerous conditions in the stomach **(**MAPS), **2012**	Stipulates definite steps to take in the management of pre-neoplastic lesions. Designed by professional societies in Europe.	54
Kyoto consensus, **2015**	A consensus guideline. Though robust in content but is restrictive (Addresses Helicobacter pylori gastritis)	18
Maastricht V/Florence consensus, **2017**	Proposes a new definition for functional dyspepsia (Helicobacter pylori gastritis has to be excluded before a reliable diagnosis of FD can be made)	15
ICD-11 (Definitive document expected to be released in **2018**)	Still in its draft form, but is expected to incorporate an etiology-based classification model.	http:// apps.who.int/ classificationsicd11/ browse/f/en

in future that will include the use of image-enhanced endoscopic techniques. These emerging technologies have improved the accuracy and reproducibility of endoscopic diagnosis of premalignant gastric lesions[92-99]. Some of them are now routinely available in Japan and will be increasingly used worldwide with the passage of time.

CONCLUSION

It is evident from the foregoing that chronic gastritis can be likened to the "story of the blind men and the elephant". Each of the blind men had a different perspective of the big animal depending on the part of the animal he felt. Till date, there still exist multiple perspectives of chronic gastritis classification depending on the interests and biases of those proposing them. A single classification that would meet the expectations of all stakeholders remains elusive.

The Sydney system remains a reference schema for the clinical and pathological characterization of the individual patient and is likely to remain in use for a long time to come because of its robustness and patient-centeredness. On the other hand, the OLGA/ OLGIM system is useful in cancer risk stratification and surveillance while the ICD system provides a global template for the reporting of morbidity and mortality information as well as identification of trends in chronic gastritis statistics. It also facilitates the comparison and sharing of information using a common language. The Kyoto consensus, though designed for Helicobacter pylori-induced chronic gastritis, is a comprehensive guideline that encompasses other classification systems but would be difficult to apply in routine patient care because it is more of a consensus guideline than a classification system. It is highly probable that in future other classification systems that utilize image-enhanced endoscopic techniques would emerge.

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