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Chronic Gastritis

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INDEX

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I.

UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS
October 20 - October 25, 1947

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Monday, October 20

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U.H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns' Quarters, U.H.

9:00 - 12:00 Physical Medicine Conference; Report on Other Physical Medicine Centers; Fredric Kottke; Eustis Amphitheater, U.H.

10:00 - 12:00 Neurology Ward Rounds; A. B. Baker and Staff; Station 50, U.H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U.H.

12:00 - 12:50 Physiology Seminar; Butter Yellow Carcinogenesis and Cancer Biology; C. P. Rhoads; 214 M.H.

12:15 - 1:20 Pediatrics Seminar; Influenza Vaccination; R. I. Lienke; 6th Floor Seminar Room; U.H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; M-435, U.H.

12:00 - 12:50 Pathology Seminar; Butter Yellow Carcinogenesis and Cancer Biology; C. P. Rhoads; 214 M.H.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

Tuesday, October 21

8:30 - 10:20 Surgery Reading Conference; Lyle Hay; Small Conference Room, Bldg. I, Veterans' Hospital.

9:00 - 9:50 Roentgenology-Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U.H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Nathanial Lufkin; Veterans' Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I.A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U.H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

5:00 - 5:50 Roentgenology Diagnosis Conference; J. Richards Aurelius and Staff of Ancker Hospital; M-515, U.H.

Wednesday, October 22

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U.H.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Hepatic Coma; E. T. Bell, O. H. Wangensteen, C. J. Watson, and Staff; Todd Amphitheater,

12:00 - 12:50 Physiological Chemistry Seminar; Subject to be Announced; E. G. Frame; 214 M.H.

4:00 - 5:00 Infectious Disease Routes, Todd Amphitheater, General Hospital, Veterans' Hospital.

Thursday, October 23

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and H. M. Stauffer; M-515, U.H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans' Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U.H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U.H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

1:30 - 3:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor West Wing, U.H.

4:00 - 4:50 Bacteriology Seminar; Clostridium Botulinum Studies; J. S. Bever; 214 M.H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U.H.

5:00 - 5:50 Roentgenology Seminar; Calcification of Ascending Aorta as a Roentgen Sign of Syphilitic Aortitis; Robert S. Leighton; M-515, U.H.

7:00 - 8:00 Urology-Roentgenology Conference; H. M. Stauffer and George Eaves; M-515, U.H.

Friday, October 24

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U.H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U.H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U.H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U.H.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Pancreatic Insufficiency Clinical and Metabolic Considerations; Charles D. May; New Powell Hall Amphitheater.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U.H.

1:00 - 2:50 Neurosurgery Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U.H.

5:30 - 6:20 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital.

Saturday, October 25

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U.H.

8:30 - 10:00 Psychiatry and Neurology Grand Rounds; Staff; U.H.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, and Staff; Todd Amphitheater, U.H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; M-515, U.H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U.H.

11:00 - 12:20 Anatomy Seminar; Newer Concepts of Cerebral Localization; A. T. Rasmussen; 226 I.A.

II. CHRONIC GASTRITIS

Robert Hebbel

While the term "gastritis" may be applied to any inflammatory lesion of the stomach it is commonly employed to designate, in a restricted sense, a group of changes, largely of chronic character, mainly limited to the mucosa which are manifested by excess cellular infiltration of the stroma and atrophy and abnormal regeneration of the glands, their crypts and the surface epithelium. This presentation is a brief review of the more important aspects of the pathology of chronic gastritis together with some of the implications concerned.

The more remote historical aspects of the subject, details of which may be found in the papers of Faber,[21,22,25] Konjetzny,[45] and others, may be omitted, One should remember, however, that many of the observations made prior to 1900 may be discarded as unreliable because of the confusion of postmortem with antemortem changes. Attempts to eliminate postmortem changes began with the introduction of fixatives into the stomach or into the abdominal cavity immediately after death. Finally gastric surgery provided another source of well-preserved material.

The Normal Gastric Mucosa.

In addition to longitudinal folds the mucosa presents slightly budging areas from 1 to 5 mm in diameter which, particularly in fixed specimens, give the surface a mammillated appearance--the areae gastricae. These in turn show a much finer relief produced by the gastric pits. There are three types of glands -- cardiac, body and pyloric.

The cardiac glands are compound tubular glands of mucoid character grouped about the gastro-esophageal boundary and occupy a zone of variably estimated width ranging up to 3.4 cm. (Plenk[65], Bensley[3]). They give way to body glands through a narrow transition zone.

The bulk of the mucosa is occupied by the acid and pepsin producing glands named body glands by Schwalbe[73] and also known as fundic glands (Oppel[59]) and chief glands (Lehner[48]). They are closely packed simple tubular glands which present chief, parietal and mucous neck cells. The thickness of the body mucosa ranges around 1 mm. and the crypts occupy 25-30% of the depth of the glands (Berger[4], Paschkis and Orator[64]).

The distal portion of the stomach is occupied by the pyloric glands which are branched tubular glands. The cells are mucoid and resemble those of the cardiac and duodenal glands and the mucous neck cells of the body glands. This portion of the mucosa also ranges around 1 mm. in thickness and the pits here occupy from 50 to 60% of the depth of the glands (Paschkis and Orator[64], Berger[4], Plenk[65]). The site of transition from body to pyloric glands varies but is regularly higher on the lesser curvature than on the greater. On the lesser curvature the transition is at about the angulus and on the greater curvature may be anywhere from pylorus to knee (Paschkis and Orator[64], Berger[4], Miyagawa[57]). The histological antrum, then, occupies a roughly triangular zone with its apex at the angulus and its base toward the pylorus. The source of histological material from the stomach must be selected with regard to these relationships.

Evidences of Gastritis

Gross. It is often quite impossible to recognize grossly the presence or severity of gastritis in either resected or postmortem specimens. Some features may be helpful but their absence does not preclude an altered mucosa. Tenacious, adherent mucus, obvious thinning or exaggerated irregularity of mucosal relief and raised, rounded or elongated, umbilicated foci are reasonably certain signs. Color and focal or more extensive hemorrhage are unreliable. The diagnosis depends on the presence of microscopic changes and should be seldom or never made and certainly never excluded without histo-

logical examination.

Microscopic. The normal histology of the gastric mucosa was based primarily on early studies such as those of Bloch[6], Faber and Lange[26] and Vimtrup[82], who examined stomachs chiefly from infants and young children. In these there was no cellular infiltration of the stroma, the glands were everywhere regularly arranged and intact. Any deviations from this pattern have usually been considered abnormal.

Heavy lymphocytic and plasma cell infiltration of the stroma together with numerous lymphoid follicles is commonly accepted as abnormal. The dividing line between normal and abnormal is necessarily arbitrary. Much of the controversy as to what constitutes gastritis concerns this point. The stroma normally contains a few cells and, in the antrum where the pits are deeper and the stroma somewhat more abundant than in the body the cells are more conspicuous. Hillerbrand[37] stated that a normal number of lymphocytes cannot be estimated. Wanser[83] indicated that anything in excess of a few cells is abnormal. Kalima[42] pointed out that the diagnosis on the basis of scanty infiltration should be made cautiously. Konjetzny[45] believed that the diagnosis must rest not only on cellular infiltration but on changes in the parenchyma and stroma as well. Others have expressed similar views.

Lymph follicles are rare in the stomachs of infants (Lange[47]) and at any age under normal conditions (Konjetzny[45]). When present they tend to be most numerous in the antrum and along the lesser curvature (Hillenbrand[37]). Large numbers are usually accompanied by abnormal degrees of diffuse lymphocytic infiltration. Although a normal number cannot be estimated small numbers of follicles in an otherwise unaltered mucosa are of little significance.

Eosinophils in variable numbers are commonly seen and an absolute normal cannot be estimated. Russell's corpuscles are found in appreciable numbers only in the presence of gastritis (Chuma[13], Konjetzny[45]).

Proliferative changes in the stroma are seen only in the sclerotic stage of atrophic gastritis by Faber and Lange[26] and others. Konjetzny[45] noted a rather constant increase in connective tissue, frequently as wedge-shaped formations piled upon and not sharply separable from the muscularis mucosa.

Thickening of the muscularis mucosa was also described by Konjetzny[45] as evidence of gastritis. Hillenbrand[37] considered it an apparent increase due to loss of glands and a consequent approximation of the muscular twigs which normally pass out between the glands of the antrum.

Parenchymal changes manifest themselves in atrophy and repair and have been thought to result from long-standing, mild inflammatory changes with consequent destruction of the glands accompanied by a relative and sometimes excessive hyperplasia of the epithelium of the pits and surface (Konjetzny[45], Kalima[42]). The glands may completely disappear leaving only a thin membrane covered with surface epithelium. Persistant groups of glands may be left as adenoma-like structures. In the body of the stomach pseudopyloric glands (Stoerk[76]) may be found. Heterotopic glands (extensions into or through the muscularis mucosa) may be found (Hallas[31], Chuma[13], Kalima[42]). Intestinal epithelium occurs as part of the picture of chronic gastritis (Faber[21,22], Konjetzny[45], Magnus[51], Kalima[42]), may be focal or diffuse and occurs in both antrum and body. The glands so formed correspond to the crypts of Lieberkuhn of the small intestine (Lubarsch[50]) and contain Paneth cells (Bloch[6] Jouvenal[40]).

Erosions are variably superficial defects of the mucosa commonly seen in healed or healing stages. Acute erosions range from minute superficial interfoveolar defects to deep clefts approaching the muscularis mucosa. The free surface is covered by exudate and the neighboring portions of the mucosa are infiltrated by an inflammatory cellular exudate. The gross appearance of healed erosions has been referred to. Microscopically there is a depressed area surrounded by a thickened mucosa. Erosions of this kind are seen particularly in the antrum in cases of ulcer

(Konjetzny[45], Faber[24,25]). Erosions seen in the body mucosa at autopsy as little hemorrhagic defects may be inflammatory erosions or agonal changes of no importance. Microscopic examination is required for the diagnosis.

Gastritis in the Absence of Other Gastric Disease.

Changes of the character of those referred to above are said by most observers to be common in the stomachs of adults. In a presumably large autopsy experience Paschkis and Orator[64] stated that over a nine months period they encountered but 10 normal stomachs and that this number was reduced to 4 on detailed examination. Hillenbrand[37] found but 4 of 21 stomachs from individuals past 35 years of age to be free of atrophy and metaphasia and even these showed numerous lymphoid follicles along the lesser curvature. Hamperl[33] expressed the opinion that gastritis changes are so common that one may wonder whether their absence is not the abnormal. Guiss and Stewart[30] noted an increasing incidence of chronic gastritis with advancing age and stated that in old age a normal stomach is rare.

There is no doubt that gastritis changes are common in older individuals but we have little information concerning the severity and extent of the process when it is present. A few focal lesions can hardly have the same significance as a diffuse process; yet, few reports make any distinction. Hillenbrand[37] cut the whole mucosa in serial roll preparations except in those instances where the lesser curvature was free of change but his data concerned a relatively small group of cases. Guiss and Stewart[30] cut sections regularly from multiple areas which could be expected to reflect the condition of the whole stomach. Their report however does not indicate the extent of the process in any given instance. In a previous report I[35] gave the findings in a group of autopsy specimens based on the results of the changes observed in sections from antrum and body respectively which were selected from areas considered most likely to reflect diffuse changes. In a small group of this series the findings in the sections compared favorably with rolls from greater and lesser curvatures. The incidence of significant change was much lower than that reported by others. However, because, in the absence of diffuse alteration, many focal to patchy areas may have been missed the results are open to criticism. We need more information concerning the exact distribution, severity and manner of progression of the changes seen independent of other disease. Such a study is in progress.

Some evidence of the incidence of gastritis is to be found in the data of series of gastroscopic examinations. We do not yet know, however, the degree to which gastroscopic interpretation parallels the histological findings. Few correlations have been made. Swalm and Morrison[77] found disagreement between gastroscopy and biopsy in 24% of 25 cases. Benedict and Mallory[2] compared the gastroscopic and histological findings in a series of 51 resected stomachs and reported complete agreement in 54.9 percent, partial agreement in 33.3 percent and complete disagreement in 11.8 percent of the cases. Magnus and Rodgers[53] noted that histological changes were present when stomachs were gastroscopically normal. It appears that gastroscopic results must be interpreted with at least the reservations that discrepancies such as these may exist.

Large series of gastroscopic examinations show an incidence of gastritis in the neighborhood of 40 percent of the cases (Schindler[72] and others). Such data concern patients. Culter and Walther[15] examined 33 asymptomatic volunteers from 20 to 46 years of age and of these 30 showed no disease. Of 40 asymptomatic individuals 19-32 years of age examined by Fitzgibbon and Long[27] 38 showed no disease. These are largely young individuals. The results parallel the infrequency of gastritic changes in my material[35] in persons under 40 years of age. The examination of a large group of asymptomatics from older age groups would provide useful data. Some evidence in this direction is offered by the experience of Carey, Wetherby and Ylvisaker[11] concerning a group of achlorhydric patients without

gastrointestinal disease in which 25 percent were gastroscopically normal.

Etiology of Chronic Gastritis

There is little factual evidence concerning the etiology of chronic gastritis. Exogenous and endogenous factors (Faber[25]) are recognized. Both are difficult to evaluate. Alcohol is an example of a supposed exogenous irritant which has been alleged to be a factor responsible for gastritis leading to atrophy. There have been no extensive anatomic studies. The few alcoholics represented in my material show no preponderance of gastritis. The best evidence to date is found in gastroscopic studies. Although Henning[36] noted a frequent association of gastritis and alcoholism Gray and Schindler[29] found the stomachs of 55 of a group of 100 chronic alcoholics to be normal or essentially normal. Of the 45 abnormals 30 showed varying degrees of superficial gastritis and 21 showed variably severe atrophic gastritis. In a similar study Berry[5] reported only 35 cases of unequivocal gastritis among 100 alcoholics. Alcohol as well as other substances were used to produce acute mucosal lesions in animals (Ebstein[18] Loesch[49]. Popoff[66]). Thomsen[79] repeatedly introduced alcohol into the stomach of a dog with a fundic pouch and described eventual atrophy. The extent of the process was not noted. It might be mentioned that Overgaard[62] claimed that normal stomachs in dogs are as rare as in man. As another example of irritation gastritis Konjetzny and Puhl[46] described an antral gastritis in calves at the time of weaning and attributed it to the effects of coarse foods on the mucosa. Many other substances have been blamed. Faber[25] summarized the claims as follows: "and after taking strong alcoholic drinks too, the etiology of gastritis is clear enough, but when we have to do with ordinary overeating, bad teeth, coarse or decomposed food, oral sepsis and such things it is conceivable that there is a constant accumulation of less injurious irritants which will act particularly when they come in contact with a stomach that already has gastritic changes." There is certainly much speculation in this comment. Faber too considers an acute hematogenous gastritis to accompany practically all infectious diseases. Gastritis was described in typhoid fever[12], in pneumonia and appendicitis[17] in diphtheria[58] and in acute infections[34,74]. Gastritis similar to that seen in diphtheria was produced in dogs with diphtheria toxin by Enrique and Hallion[19] and by Thomsen[79]. Hurst[38] considers gastritis to frequently follow influenza. My own material includes few infectious diseases but in none of the cases have I found any acute gastritis and among those individuals whose histories recorded infectious diseases was there any preponderance of gastritis. Chronic infections such as tuberculosis are also said to show a high incidence of gastritis (Faber[20]). My material includes few cases of chronic tuberculosis but there is no unusual incidence of gastritis. Another point may be raised. If, as has been claimed, many infectious diseases are accompanied by an acute gastritis which sets the stage for the chronic changes why are lesions of any kind so uncommon in individuals under 40 years of age?

It is obvious that there are many unanswered questions concerning this process which affects the gastric mucosa of many individuals. There is little evidence pointing to specific etiologic factors. All investigators find an increasing incidence and severity with advancing age. While some cases may originate in an acute, florid gastritis or even result from repeated acute attacks the bulk of the histological evidence presents only chronic changes irrespective of severity. One must remember, however, that here, as in other locations, reactions of short duration may exhibit a chronic type of exudate and some of the "chronic" changes may be relatively acute. Once glandular alterations are present one may be certain of chronicity. The process by which glands are transformed and disappear is not easily followed. In my material there is nothing to suggest other than a gradual process. While focal and patchy lesions might well result from exogenous irritants, diffuse changes seem more likely the result of other factors. The gastritis observed in connection with other gastric lesions

must be interpreted with regard to the findings in individuals of the same age otherwise free of disease.

Gastritis With Peptic Ulcer

Most observers are agreed that an abnormal gastric mucosa is found in association with both gastric and duodenal ulcer. Konjetzny[45] in particular made extensive studies of the process. Similar findings are recorded in the papers of Kalima[42], Puhl[68], Orator[60], Magnus and Rodgers[53], Wanser[83] and many others. Gastric and duodenal ulcer must be considered separately for the findings differ.

With duodenal ulcer the gastritis is confined to the antrum and sometimes that portion of the body immediately adjacent to the transition zone. The remainder of the body rarely shows significant change and in the older age groups appears to show change less frequently than is found in "normals".[35] In the antrum the severity of the process varies but the changes exist irrespective of the age and duration of symptoms. Excessive lymphocytic infiltration, numerous lymphoid follicles and variably severe atrophy are present. There are healed and acute erosions. Acute erosions are common according to Konjetzny; they are very rare in my material. Intestinal metaphasia is uncommonly extensive. It may be added here that a similar antral gastritis may exist without demonstrable duodenal or gastric ulcer and has been considered responsible for ulcer symptoms, severe hemorrhage and the like (Anderson[1], Konjetzny[45], Puhl[68], Faber[24], Peaby[63], Roholm[69] and others).

With gastric ulcer there is an antral gastritis of the same character. Additionally there are not uncommonly diffuse gastritic changes in the body mucosa (Orator[60], Borchardt[8], Puchert[67], Simpson[74], Magnus and Rodgers[53], Hebbel[35] and others). While the process is rarely of more than moderate severity extensive atrophy may be found.

Gastritis and Carcinoma of the Stomach

The frequent association of atrophic gastritis with gastric cancer was recognized early and the process has been described and discussed in varying detail by many authors (Rosenheim[70], Matthieu[54], Hammerschlag[32], Boekelman[7], Matti[55], Saltzman[71], Konjetzny[43,44,45], Orator[61], Geissendorfer[28], Borchardt[8], Puchert[67], Steinberg[75], Tuomikoski[81], Simpson[74], Judd[41], Guiss and Stewart[30], Warren and Meissner[84], Magnus[52] and others). This gastritis has been called a pangastritis in contrast to the usually antrum-confined process associated with ulcer. The qualitative features are much the same though more extensive and there is more metaphasia with cancer than with ulcer. Konjetzny considered ulcer- and cancer gastritis as subacute and chronic phases of the same process.

There appear to be three explanations of the association of gastritis with carcinoma of the stomach:
1. Atrophic gastritis precedes and is the basis on which carcinoma develops,
2. Atrophic gastritis precedes but bears only a coincidental relationship to the carcinoma, 3. the gastritis develops because of or in association with the carcinoma.

Mattieu[54] apparently first suggested the possible origin of carcinoma in a gastritic mocosa. Saltzman[71] and Konjetzny[43,44,45], particularly the latter, strongly advanced this concept at about the same time. Konjetzny emphasized the existence of atrophic-hyperplastic changes in gastritis and claimed a demonstrable relationship to this process for about 90 percent of carcinomas. Orator[61] and others also accepted a gastritic basis for carcinoma. Borrmann[9] on the other hand saw no such relationship and concluded that most carcinomas arose without or independent of pre-existing inflammatory changes. Magnus[52] failed to find the hyperplastic changes emphasized by Konjetzny in his material. Wanser emphasized the benignancy of long-standing gastritis associated with ulcer and rightfully questioned Konjetzny's consideration of ulcer-and cancer gastritis as subacute and chronic phases of the same process.

Most tumors are large when examined

and one can no more than infer the charater of the mucosa at the sight of origin. Exact information depends on the examination of very small tumors. Completely satisfactory descriptions and illustrations of small lesions in relation to the adjacent mucosa are not available. In my own material there is one small carcinoma, a prepyloric superficially ulcerating lesion less than 1 cm, in diameter. Even here one cannot tell the character of the destroyed mucosa but, if this carcinoma arose on the basis of gastritis it began in a microscopic patch or focal lesion of which several are scattered through an otherwise normal antrum. The body is entirely normal. It is noteworthy that Magnus[52] reports that of 25 antral carcinomas the body mucosa was normal in 22 instances.

On the other hand there are instances where one may be certain that the tumor arose in an atrophic mucosa, particularly in those cases of pernicious anemia where the carcinoma is in the body of the stomach. That atrophic gastritis is the link between pernicious anemia and the relative frequency of carcinoma in that disease has been maintained by Miller[56], Jenner[39], Thiele[78] and others. There is conflicting opinion in the literature concerning the changes in the stomach in pernicious anemia. Brown[10], for example, included as one case an instance in which there was free acid, no response to liver and described the stomach as normal. This case would seem to be quite certainly not that disease. Faber[21,25] described cases with gastric changes ranging from rather moderate cellular infiltration with well preserved glands to more severe atrophy. Most authors describe complete atrophy. Magnus and Ungley[53a], consider the process in pernicious anemia to be a non-inflammatory atrophy. Faber[25] and Hurst[38] consider the changes to result from a diffuse gastritis leading to atrophy. While some authors describe changes in the antrum the best reports (Magnus and Ungley[53a], Cox[114]) show little or no change in the antral mucosa. If the atrophic mucosa is the basis on which carcinomas develop they should then arise in the body of the stomach. Torgerson[80] attempted to answer this question by reviewing the location in the stomach of 106 carcinomas in patients with pernicious anemia and compared this data with the location of 400 carcinomas in patients without pernicious anemia. The majority of the 400 cases (58 percent) were judged to be antral while among the 106 cases from patients with pernicious anemia 62 percent were judged to arise in the body -- an approximate reversal of the figures. However there remain a considerable number in which the tumor arose, as the best evidence indicates, in a non-involved antrum.

It has been noted above that the noncancerous portions of the mucosa are usually far beyond the starting point of the tumor and, because most carcinomas involve more or less of the antrum, only the body mucosa may be examined in any detail. It has also been noted that most authors describe a pangastritis with carcinoma. If such a process precedes a carcinoma the pattern in the mucosa should parallel that seen with atrophic gastritis alone. Such is admittedly the case in many instances. However, close examination reveals[35] rather frequent inconstant patterns. These range not only from diffuse, severe changes in some specimens to normality in others but also, among some of those which show gastritis a process which varies in severity directly with proximity to the tumor. For example, given a stomach in which the entire antrum is occupied by a carcinoma the body mucosa may show severe changes adjacent to the tumor and milder changes beyond which in turn give way to minimal involvement or even normality in more distant regions.

Possible explanations for these observations are: 1. various stages in an independent spreading gastritis are represented, or 2. the changes develop with and under the influence of the tumor. As concerns the first point it is possible that if a gastritis begins in the lower part of the stomach and spreads upward we may here be observing the differences between cases in which, on the one hand, cancer appeared before the process was complete and, on the other, after it was fully established. However, we do not yet know the exact

manner or possible manners of progression of atrophic gastritis. If it is an upward-spreading process one should occasionally see in postmortem material instances in which the findings sometimes associated with cancer are duplicated. My autopsy material, though the number of pertinent cases is small, does not support this mode of progression. They suggest uniform involvement throughout irrespective of the degree of severity.

As to dependence on the tumor one may immediately raise the question as to why some tumors fail to produce any change. Suggesting a possible influence of the tumor, however, are two cases in the collection of carcinomas of the cardia in which the gastritic process reverses itself in direction and becomes less intense as the distal portions of the body are reached.

It is evident that an appreciable number of carcinomas appear in stomachs which are not the seat of diffuse atrophic gastritis and diffuse atrophy cannot be considered a necessary precurser. Focal lesions are very common and there is here much to suggest a coincidental relationship. The importance of focal and patchy gastritis to the development of carcinoma can be determined only by the examination of small lesions.

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III. GOSSIP

Michael Alpheus Burns, M.D., Milan, Minnesota, was honored with a celebration commemorating 50 years of practice in Milan community. Dr. Burns graduated from the Medical School, University of Minnesota in a distinguished class which included Robert A. Campbell, J. Frank Corbett, Warren A. Dennis (father of Clarence), Robert Earl, Walter R. Ramsey, Harry P. Ritchie (father of Wallie), John A. Thaves and Louis B. Wilson. He took an internship at Minneapolis General Hospital and married one of the nurses which sounds modern. When Dr. Burns went to Milan, he discovered that a majority of his patients were Norwegians. In self defense, he learned to speak the language. Milan Norwegian is idiomatic and cannot be learned from books. During the program they told of many mistakes which Dr. Burns had made in using borderline words. After four years in the community, he decided to go to New York for postgraduate work. He liked it so well he stayed nearly six months. His account of how difficult it was to get out of town was one which depicted a struggle between devotion to duty and a desire to better himself. Just as he was ready to get on the train, someone would call him out to see a patient and he would make the visit. It took him four days to get out of town. When he returned, he had made up his mind to leave for a larger place and he went back to pack his belongings. Just as he got off the train, the people were waiting to ask him to make a call and he has been doing it ever since.

The program was held in a spic and span Lutheran Church. Every seat on the main floor was taken. An overflow audience was in the basement and on the lawn (public address system). A young minister presided and I found him very interesting. I tried to place his accent and finally concluded he must be from Brooklyn. He confided to me at the close of the meeting that he was and he excused himself to go to the house for a minute to see how the "bums" were doing. The old minister who was there when Dr. Burns came told of many experiences that they had had together. He said that Dr. Burns never tried to do anything on them which he was not capable of doing. He also said that he never pretended to know anything which he did not know, and finally, the most important, he had never turned down a call. When other doctors made excuses, Dr. Burns came. This was especially true of night calls. Even today Dr. Smith of Montevideo said, he made night calls without question. He never gave them a brushoff and he never told them to take aspirin. Dr. Burns felt that either his services were needed or that the people were suffering from anxiety, in which case he also was needed. The old minister said that many times doctors were given credit for saving lives when they may not have been responsible. In Dr. Burns' case, however, he felt that his great devotion must have resulted in life-saving especially back in diphtheria days. Children from the school told of their affection for him. Members of the community presented him with a watch. A cash gift was also given by the people. Dr. Burns apparently never paid too much attention to collections and failure to pay his bill was not a reason for denial of service. Dr. Arnold Anderson, one of the many babies he had delivered, sent a wonderful tribute to "our doctor" as he was constantly referred to by each speaker on the program. Dr. Burns waited until Dr. Anderson finished his internship before taking his second vacation. Most amusing part of the program was a quartette with a Gay Nineties Spirit. The tension would become high from the tributes and then the quartette would come out. Someone else would speak and then the quartette would come out. I spoke on the significance of the service Dr. Burns had rendered. Following the formal program there was a reception. I wondered if men of Dr. Burns' type had passed or if times had changed. Certainly the devotion to duty which Dr. Burns had displayed must be present today in some members of the profession (at least in the daytime).. Methods of practice are changing and there may never be future celebrations for physicians who have spent 50 years in one place. It must be a great source of personal satisfaction to know how much their efforts have been appreciated for after all success can be measured only in terms of how well those who know us appreciate what we have done.....