Histopathological Studies on Gastric Carcinoma among Koreans

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ABSTRACT

The materials used in this study consist of 744 gastric resections removed subtotally at the Yonsei University College of Medicine and Severance Hospital during the last 11 years from Jan. 1, 1959 to Dec. 31, 1969. Among these, 446 cases of gastric carcinoma were diagnosed at the Department of Pathology Yonsei University College of Medicine for this same duration. Histopathological studies as well as clinical study on all cases were performed.

Summary:
1. Among 744 gastric resections examined during the last 11 years, gastric carcinoma was found in 446 cases, giving an incidence of 59.95% for subtotally removed stomachs.
2. Male to female ratio was 2.28 to 1. The peak age incidence is between 50 and 59 years and accounted for 172 cases (35.77%).
3. There is no relation between blood group and incidence of gastric carcinoma.
4. Clinical symptoms in order of frequency were as follows: epigastric discomfort and pain (86%), indigestion (86.67%), weight loss (35.67%), nausea and vomiting (35.33%) and palpable epigastric mass (23.33%). The duration of the chief complaints was usually within six months (54.66%).

5. The gastric acidity disclosed that achlorhydria was found in 36.21%, hypochlorhydria in 32.26% and hyperchlorhydria in 2.15%.

6. The location of the tumor was as follows: 44.86% at the pyloric canal, and 26.53% at the antrum. The size of the tumor varied in diameter. The most frequent size was 3—5 cm (43.43%) and 6—10 cm (42.90%).

7. On the macroscopical examination, the classification based on Borrmann's gross types and their incidences were as follows: Type I (9.71%), type II (11.22%), type III (62.18%), and type IV (23.64%).

8. On the histological classification, 70% of the cases were adenocarcinomas. Serosal involvement was found in most cases (86.89%).

9. Metastasis to the regional lymphnodes was found in 68.84%, and direct extension of the omentum was 21.39%.

10. According to the relationship between regional lymphnode metastases and lymphatic permeation, and relationship between lymphatic permeation and histologic type, the degree of lymphatic permeation is more, the more metastases to regional lymphnode is found. And metastasis to regional lymphnode and lymphatic permeation is more common in adenocarcinoma than other types.

11. There is no relationship between intestinal metaplasia and histologic type. But each histologic type was accompanied by intestinal metaplasia frequently.

In conclusion based on the above findings,
it can be stated that gastric carcinoma among Koreans exhibited a far advanced stage, both clinically and morphologically.

INTRODUCTION

In 1858 Virchow(1821—1902), the father of modern pathology, established the concept of fundamentals of morphology of tumors in his great article “Cellular Pathology”, and von Hansemann(1890) classified the degree of malignancy of neoplasms for the first time. Since then cancer has remained a big problem in the medical field and all the scientists in the world have been trying to control the neoplasms. As a result much progress has been made in the field of oncology and the treatment of malignant tumors. Unfortunately, however, the malignant tumor still causes high mortality and remains as an incurable fatal disease.

Among these neoplasms, gastric carcinoma particularly is one of the most common malignant tumors and according to Jemerin and Colp(1952), it once occupied one third to one fifth of all deaths from neoplasms in the United States of America. Since then according to Moore(1962), the incidence of gastric carcinoma has been markedly decreased. But among Koreans, gastric carcinoma is the most common carcinoma next to cervical cancer, and the most common carcinoma in males(Kim et al., 1956; Kim et al., 1962; Lee et al., 1967; Lee et al., 1968).

There is considerable controversy on the etiology of gastric carcinoma and it’s high incidence rate and fatality rate. But the etiology of gastric carcinoma is considered hereditary, environmental, dietary temperature and stimulating factors, endocrinological factor concerning age and sex, constitutional sensitivity, and precancerous diseases, but there are no definite conclusions.

Many efforts have been made to elucidate precancerous disease of gastric carcinoma, and achlorhydria or low acidity, pernicious anemia, gastric polyp, chronic atrophic gastritis, intestinal metaplasia and gastric ulcer are all considered as possible precancerous conditions.

A prolonged state of achlorhydria or low acidity has been reported many times as a precancerous state. Comfort, Kelsey and Berkson(1947) reported the high incidence of gastric carcinoma in low gastric acidity and 68.8 percent of gastric carcinoma patients showed achlorhydria. Berkson, Comfort and Butt(1956) also reported that the incidence of gastric carcinoma is six times higher in patients with achlorhydria and three times higher in people with pernicious anemia(Kaplan and Rigler, 1945; Jørgenson, 1951).

The relationship between chronic atrophic gastritis associated with low acidity and gastric carcinoma, has brought out different opinions. Konjetzny(1921) and Hurst(1933) claimed chronic atrophic gastritis as a precancerous disease and 75 percent of gastric carcinoma arises from chronic atrophic gastritis.Wanser(1939), Hebbel(1943), and Stewart(1925), however, stated that the incidence of chronic atrophic gastritis increases as the age increases and gastric carcinoma might cause chronic atrophic gastritis or worsen preexisting chronic atrophic gastritis, but that there is no relationship between them. But most investigators believed that inflammation, intestinal metaplasia and atrophy of gastric mucosa are precancerous states (Flood and Lattes, 1967).

Adenomatous polyp of the stomach, although it is very rare, seems to cause malignant change and it seems more apparent if they are of multiple origins and large in size (Monaco
et al., 1962).

It is true that some peptic ulcers of the stomach become malignant and it is believed that five to 18 percent of gastric carcinomas originated from a preexisting peptic ulcer of the stomach. Moreover Stewart (1955) made out a pathological criteria for ulcer-carcinoma.

Since Schlatter (1897) had been successful in a gastrectomy for the treatment of gastric carcinoma for the first time, the surgical treatment is the only curable method of treatment. Gilbertsen & Hollenberg (1962) reported recently the survival rate of surgical gastrectomy for the treatment of gastric carcinoma was remarkably increased, and five year-survival rate is 4.5% among all gastric carcinoma patients, 50.8% without regional lymphode metastasis, and 88% in so called early cancer of the stomach (Lawrence & Kay, 1946: Yokoyama et al., 1961).

Since Virchow, pathological study for gastric carcinoma has been made continually to find out the nature of it. The predilection area for gastric carcinoma is in the pyloric region in 50% of the cases (Stewart, 1931: Poscharsky, 1930; Willis, 1967). Trimble and Lynn (1955) analyzed 5,000 cases of gastric carcinoma from literature and reported the most common site for gastric carcinoma as pylorus, lesser curvature, antrum, fundus and greater curvature in that order of frequency. Eker (1951) reported that the ulcerating type is common in the pyloric region and the fungating type increases as it approaches the cardiac portion.

Stout (1953) classifying the gross types of gastric carcinoma emphasized its significance because it depends on the growing method of gastric carcinoma. According to him, it is classified into penetrating or ulcerating type, fungating type, spreading type and no special type and it is in that order of frequency. On the other hand, Borrmann (1926) made the classification of gross type into I, II, III, and IV; type I being fungating type, II ulcerating type, III penetrating as well as ulcerating type, and type IV as a diffusely penetrating type. Recently Willis (1967) concluded that the fungating type in gross has an adenopapillary structure histologically and the spreading type has infiltrating spheroidal cells or a signet-ring cell structure generally.

Histologically gastric carcinoma in most cases arises from glands and the classification does not differ much. The older classification is as follows; (1) simple adenocarcinoma, (2) papillary carcinoma, (3) colloid or gelatinous carcinoma, (4) medullary carcinoma, (5) scirrhous carcinoma, and (6) mixed carcinoma. Mulligan and Rember (1954) put stress on the histogenesis and classified as (1) mucinous cell type, (2) pylorocardiac gland cell type, (3) intestinal cell type. Willis (1967) reviewed many articles and classified as (1) adenocarcinoma or adenopapillary carcinoma, (2) mucoid adenocarcinoma, (3) signet-ring cell carcinoma, (4) infiltrating spheroidal cell carcinoma, (5) metaplastic squamous cell carcinoma, (6) highly cellular anaplastic carcinoma. Ackerman and Del Regato (1954) classified it according to the stage of differentiation as most of gastric carcinoma are adenocarcinoma.

Trial has been increased to differentiate the degree of malignancy of gastric carcinoma by classifying it pathologically. Steiner (1948) studied the morphological factors affecting 5 year-survival after gastrectomy for gastric carcinoma. The relationship between prognosis and morphology of gastric carcinoma also has been studied by many investigators (Mulligan and Rember, 1954; Urban and McNeer, 1959; Brown et al., 1961; McNeer et al., 1958). If the degree of malignancy of gastric carcinoma could be de-
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termined by the histopathology of gastric carcinoma, it would be of great help to determine the prognosis as well as to determine the treatment.

It is character for malignancy to metastasize to a different site. Foulds (1961) lists the following five processes operating in sequence in association with metastasis: (1) liberation of tumor cells or fragments of the primary growth; (2) transportation of the liberated material by one of several routes; (3) deposition of the transported material at a distant site; (4) establishment of the deposited material in its new environment; and (5) growth of the established deposit into a secondary tumor. The gastric carcinoma in the beginning metastasizes through lymphatic channels in the gastric wall. In the duodenum it spreads directly or through the lymphatics in the subserosa to the muscular layer and rarely to mucosa and in esophagus mostly by lymphatics in the submucosa Verbrugghan, 1934; Zinninger and Collins, 1947; Zinniger, 1954). So the method of metastasis could be either by superficial spread along the mucosal layer only or it may involve as far as to muscularis or all layers.

Lymphatic metastasis in gastric carcinoma happens much more frequently than it is thought as gastric carcinoma is usually found sometime after it is far advanced. Although there is some difference in different reports, lymphatic metastasis to greater and lesser curvatures is reported by Stout (1953) to be 70% by gastrectomy and 88% at necropsy. By Marshall (1958) it is 61% after surgical treatment for gastric carcinoma and states that 5 year survival is greatly affected according to the presence or absence of lymphatic metastasis. The direction of the lymphatic metastasis depends entirely upon the site of the tumor mass.

Once it involves the serosa of the stomach it can spread to anywhere in the body through various pathways like any other malignant tumor in a different part of the body. It can spread directly to the liver, transverse colon, and greater omentum through the gastrohepatic or gastrocolic ligament. Direct metastasis occurs frequently to the peritoneum, greater and lesser omenta and examples of transperitoneal dissemination are Blumer's shelf and Krukenberg's tumor.

In the United States of America, the incidence and mortality rate of gastric carcinoma remarkably decreased by dietary improvement, decrease of infection, diminution of exposure to the cancer causing substances, prevention of precancerous diseases, amelioration of social condition, and increase of early detection. However, in Korea it still remains at a high incidence rate.

Gastric carcinoma is at present actively studied in Korea. However, the studies are mostly limited to the clinical and statistical aspects. Therefore in this article the authors are going to present especially the pathological aspects of gastric carcinoma, such as the location and size of gastric carcinoma, the gross and histological classifications, changes in the remaining portion of the stomach (especially types of chronic gastritis and intestinal metaplasia), the degree of the penetration of the gastric wall, lymphatic permeation and regional lymphnode metastasis.

Gastric carcinoma among Koreans will be compared with that of other countries, an effort made to search for etiologic factors, development and progress of gastric carcinoma and to obtain basic information for the early diagnosis and treatment of gastric carcinoma.
MATERIALS AND METHODS

The materials used in this study consists of 744 subtotal gastric resection specimens at the Yonsei University College of Medicine and Severance Hospital during the last 11 years from Jan. 1, 1959 to Dec. 31, 1969. Among these, 473 cases had been diagnosed as malignant tumor by the Department of Pathology Yonsei University College of Medicine. Excluding sarcoma, metastasized choriocarcinoma from the uterus and those pathologically insufficient cases, 446 gastric carcinomas have been chosen for this study.

For the 446 cases whose records were available, sex, age, blood group, chief complaint, duration of the chief complaint and gastric acidity were reviewed.

After each resected stomach and the regional lymphnodes were grossly examined for the location, size, and gross types, more than three sections were obtained from the lesion, two sections from the remaining portion, a small section from the omentum and all regional lymphnodes for histopathological examination.

For the macroscopic examination the classification based on the Borrmann(1926) was used as follows;

Type I: circumscribed, solitary, polypoid carcinomas without important ulceration.

Type II: ulcerated carcinoma with wall-like marginal elevation on sharply defined borders.

Type III: ulcerated carcinoma in part with marginal elevation and in part with diffuse spread.

Type IV: diffuse infiltrating carcinoma.

Paraffin embedded blocks were cut 6µ in thickness and sections were stained by hematoxylin-eosin method. The histopathological study on all cases was performed as follows; (1) histopathological classification, (2) degree of penetration of the gastric wall, (3) degree of lymphatic permeation, (4) regional lymphnodes and omental metastases, (5) changes in the remaining portion, especially types of chronic gastritis and degree of intestinal metaplasia. Willis’ classification was used for the microscopic examination as follows; adenocarcinoma (well differentiated, moderately well differentiated, and poorly differentiated), signet-ring cell carcinoma, mucinous adenocarcinoma, and diffuse anaplastic carcinoma. The penetration of the gastric wall was divided into intramucosal, submucosal, muscular and serosal layers and the chronic gastritis into atrophic, superficial, hypertrophic and follicular types. Intestinal metaplasia and lymphatic permeation were described as mild, moderate, marked and severe.

RESULTS

A. Incidence

1. Incidence of gastric carcinoma among gastrectomy specimens

The total number of gastric resections which

<table>
<thead>
<tr>
<th>Table 1. Incidence of neoplasia among gastrectomy specimens (1959–1969)</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Neoplastic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. Malignant</td>
<td>314</td>
<td>145</td>
<td>459 (61.69%)</td>
</tr>
<tr>
<td>Primary carcinoma</td>
<td>310</td>
<td>136</td>
<td>446 (59.95%)</td>
</tr>
<tr>
<td>Primary sarcoma</td>
<td>4</td>
<td>8</td>
<td>12</td>
</tr>
<tr>
<td>Metastatic malignancy</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>B. Benign</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Polyp</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Leiomyoma</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Neurofibroma</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>II. Non-neoplastic</td>
<td>239</td>
<td>43</td>
<td>282 (38.31%)</td>
</tr>
<tr>
<td>Peptic ulcer</td>
<td>207</td>
<td>36</td>
<td>243 (32.66%)</td>
</tr>
<tr>
<td>Chronic gastritis</td>
<td>14</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>Others</td>
<td>18</td>
<td>4</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>555</td>
<td>189</td>
<td>744</td>
</tr>
</tbody>
</table>
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were investigated during the past 11 years from Jan. 1, 1959 to Dec. 31, 1969 was 744 cases. Gastric carcinoma was found in 446 cases giving an incidence of 60%. Among the non-malignant lesions peptic ulcer was found in 243 cases resulting in 32.66% (Table 1).

2. Sex and age distribution

Of 446 resected gastric carcinomas, 310 cases were male and 136 cases were female with the male to female ratio being 2.28 to 1. The average age of the patients was 50.91 years (50.75 years in male and 51.23 years in females). The youngest patient was 17 year old and the oldest one 80. The peak age is the 50's accounting for 35.77% and the next were 40's and 60's years (Table 2).

Table 2. Sex and age distribution

<table>
<thead>
<tr>
<th>Age</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-19</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>20-24</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>25-29</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>30-34</td>
<td>2(0.65)</td>
<td>5(3.68)</td>
<td>7(2.17)</td>
</tr>
<tr>
<td>35-39</td>
<td>27(8.71)</td>
<td>16(11.76)</td>
<td>43(10.24)</td>
</tr>
<tr>
<td>40-44</td>
<td>26(8.83)</td>
<td>14(10.29)</td>
<td>40(9.34)</td>
</tr>
<tr>
<td>45-49</td>
<td>52(16.77)</td>
<td>21(15.44)</td>
<td>73(16.11)</td>
</tr>
<tr>
<td>50-54</td>
<td>79(25.48)</td>
<td>17(12.50)</td>
<td>96(18.99)</td>
</tr>
<tr>
<td>55-59</td>
<td>54(17.42)</td>
<td>22(16.17)</td>
<td>76(16.78)</td>
</tr>
<tr>
<td>60-64</td>
<td>35(11.29)</td>
<td>25(18.38)</td>
<td>60(14.84)</td>
</tr>
<tr>
<td>65-69</td>
<td>18(5.81)</td>
<td>5(3.68)</td>
<td>23(4.74)</td>
</tr>
<tr>
<td>70-74</td>
<td>8(2.58)</td>
<td>3(2.21)</td>
<td>11(2.39)</td>
</tr>
<tr>
<td>75-79</td>
<td>3(0.97)</td>
<td>2(1.48)</td>
<td>5(1.23)</td>
</tr>
<tr>
<td>over 80</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
</tbody>
</table>

Total 310 136 446

Average : 50.91yr.  M: 50.75yr.  F: 51.23yr.

3. Blood group distribution

In general population blood group A is encountered in 31.15%, B in 28.60%, O in 30.29% and AB in 9.96%. But among the patients of gastric carcinoma, group A was 37.24%, B 29.08%, O 23.93% and AB 9.75%. This seems to show that group A is most susceptible to gastric carcinoma and group O is least susceptible. However, statistically it proved to be not significant (Table 3).

Table 3. Blood group among gastric carcinoma patients

<table>
<thead>
<tr>
<th>Blood group</th>
<th>Male (%)</th>
<th>Female (%)</th>
<th>Total (%)</th>
<th>General population (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>88(39.14)</td>
<td>41(35.35)</td>
<td>129(37.24)</td>
<td>31.15</td>
</tr>
<tr>
<td>B</td>
<td>64(27.12)</td>
<td>36(31.03)</td>
<td>100(29.08)</td>
<td>28.60</td>
</tr>
<tr>
<td>O</td>
<td>65(23.73)</td>
<td>28(24.14)</td>
<td>93(26.93)</td>
<td>30.29</td>
</tr>
<tr>
<td>AB</td>
<td>28(10.61)</td>
<td>11(9.48)</td>
<td>39(11.27)</td>
<td>9.96</td>
</tr>
</tbody>
</table>

B. Clinical findings

1. Chief complaint

Not all the patients complained of all the various clinical symptoms that are found during the course of gastric carcinoma, but in most cases they had the common complaints. Among those symptoms, epigastric discomfort or pain was 86%, indigestion 66.67%, weight loss 35.67%, and 35.33% presented with the chief complain of vomiting. Besides the above they complained of a palpable mass in the epigastric area, anorexia, general malaise of

Table 4. Chief complaints

<table>
<thead>
<tr>
<th>C.C.</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epigastric discomfort and pain</td>
<td>169</td>
<td>89</td>
<td>258(86.00)</td>
</tr>
<tr>
<td>Indigestion</td>
<td>139</td>
<td>61</td>
<td>200(66.67)</td>
</tr>
<tr>
<td>Weight loss</td>
<td>68</td>
<td>39</td>
<td>107(35.67)</td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td>67</td>
<td>39</td>
<td>106(35.33)</td>
</tr>
<tr>
<td>Epigastric palpable mass</td>
<td>43</td>
<td>27</td>
<td>70(23.33)</td>
</tr>
<tr>
<td>Anorexia</td>
<td>38</td>
<td>21</td>
<td>59(19.67)</td>
</tr>
<tr>
<td>General weakness</td>
<td>22</td>
<td>18</td>
<td>40(13.33)</td>
</tr>
<tr>
<td>Epigastric fulling sense</td>
<td>21</td>
<td>9</td>
<td>30(10.00)</td>
</tr>
<tr>
<td>Acid belching</td>
<td>19</td>
<td>10</td>
<td>29(13.00)</td>
</tr>
<tr>
<td>Constipation</td>
<td>16</td>
<td>7</td>
<td>23(7.67)</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>8</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Hematemesis</td>
<td>10</td>
<td>3</td>
<td>13</td>
</tr>
<tr>
<td>Tarry stool(melena)</td>
<td>14</td>
<td>3</td>
<td>17</td>
</tr>
<tr>
<td>Abdominal distension</td>
<td>11</td>
<td>7</td>
<td>18</td>
</tr>
<tr>
<td>Heart burning sense</td>
<td>9</td>
<td>4</td>
<td>13</td>
</tr>
<tr>
<td>Dysphagia(swallowing difficulty)</td>
<td>9</td>
<td>1</td>
<td>10</td>
</tr>
</tbody>
</table>
fullness of epigastrium (Table 4).

2. Duration of chief complaints
As for the duration of chief complaints when hospitalized, symptoms for three months were present in 103 cases (27.46%) symptoms for three to six months in 102 or 27.20%. Therefore within six months 54.66% were involved and 24 cases or 6.4% had the history for longer than five years (Table 5).

Table 5. Duration of chief complaints

<table>
<thead>
<tr>
<th>Duration</th>
<th>Male</th>
<th>Female</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>under 3M.</td>
<td>78</td>
<td>25</td>
<td>103(27.46)</td>
</tr>
<tr>
<td>4—6M.</td>
<td>71</td>
<td>31</td>
<td>102(27.20)</td>
</tr>
<tr>
<td>7—12M.</td>
<td>46</td>
<td>21</td>
<td>67(17.86)</td>
</tr>
<tr>
<td>1—2Y.</td>
<td>20</td>
<td>14</td>
<td>34( 9.09)</td>
</tr>
<tr>
<td>3—5Y.</td>
<td>26</td>
<td>19</td>
<td>45(12.00)</td>
</tr>
<tr>
<td>over 5Y.</td>
<td>16</td>
<td>8</td>
<td>24( 6.40)</td>
</tr>
</tbody>
</table>

C. Acidity of gastric juice
The analysis of gastric acidity (free acidity) in the patients with gastric carcinoma according to the standards of Vanzant et al (1933) and Wells and Halsted (1969) showed that achlorhydria was found in 101 cases or 36.32% and hypoacidity in 93 cases or 32.26%, therefore 68% of the cases fell into the group of achlorhydria or hypoacidity. Hyperacidity was present in only six cases or 2.15% but five of these were gastric carcinoma combined with peptic ulcer (Table 6).

Table 6. Gastric acidity (free acid)

<table>
<thead>
<tr>
<th>Type</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achlorhydria(0 mEq/l)</td>
<td>101</td>
<td>36.21%</td>
<td></td>
</tr>
<tr>
<td>Hypoacidity</td>
<td>99</td>
<td>32.26%</td>
<td></td>
</tr>
<tr>
<td>Normoacidity (25—50mEq/l)</td>
<td>73</td>
<td>29.38%</td>
<td></td>
</tr>
<tr>
<td>Hyperacidity</td>
<td>6</td>
<td>2.15%</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>279</td>
<td>100.00%</td>
<td></td>
</tr>
</tbody>
</table>

D. Histopathological findings

1. Location of the lesions
The most frequent site of gastric carcinoma is the pyloric canal being 44.86% and the next is the antrum being 26.53% of the cases. Tumor develops 3.7 times more often in the lesser curvature than in the greater curvature (Table 7).

Table 7. Location of the Lesions

<table>
<thead>
<tr>
<th>Location</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pylorus</td>
<td>140(46.97)</td>
<td>56(42.75)</td>
<td>196(44.86)</td>
</tr>
<tr>
<td>Antrum</td>
<td>74(24.83)</td>
<td>37(28.24)</td>
<td>111(26.53)</td>
</tr>
<tr>
<td>Body</td>
<td>22(7.38)</td>
<td>12(9.16)</td>
<td>34(8.27)</td>
</tr>
<tr>
<td>Cardia</td>
<td>21(7.07)</td>
<td>6(4.88)</td>
<td>27(5.83)</td>
</tr>
<tr>
<td>Uncertain</td>
<td>41(13.75)</td>
<td>20(15.27)</td>
<td>61(14.51)</td>
</tr>
</tbody>
</table>

Total       | 238     | 131       | 469       |

Lesser       | 165(76.77) | 79(79.79)  | 244(78.28) |
Greater      | 50(23.23)  | 20(20.21)  | 70(21.72)  |

2. Size of the lesions
The diameter of the tumor mass exceeds three to five cm. in 43.48% and six to ten cm. in 42.90%. Therefore in almost four fifths of the total cases the size fell between three to ten cm. in diameter. Less than two cm. was 7.23% and more than 10 cm. 6.44% (Table 8).

Table 8. Size of the lesions

<table>
<thead>
<tr>
<th>Size</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 2cm.</td>
<td>21(7.42)</td>
<td>9(7.68)</td>
<td>30(7.23)</td>
</tr>
<tr>
<td>3—5cm.</td>
<td>125(43.11)</td>
<td>56(43.75)</td>
<td>181(43.43)</td>
</tr>
<tr>
<td>6—10cm.</td>
<td>119(42.05)</td>
<td>56(43.75)</td>
<td>175(42.90)</td>
</tr>
<tr>
<td>Over 10cm.</td>
<td>21(7.42)</td>
<td>7(5.47)</td>
<td>28(6.44)</td>
</tr>
<tr>
<td>Total</td>
<td>283</td>
<td>128</td>
<td>411</td>
</tr>
</tbody>
</table>

Table 9. Gross type (Bormann's classification)

<table>
<thead>
<tr>
<th>Gross type</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1</td>
<td>18(5.90)</td>
<td>2(1.51)</td>
<td>20(3.71)</td>
</tr>
<tr>
<td>Type II</td>
<td>35(11.15)</td>
<td>15(11.28)</td>
<td>49(11.22)</td>
</tr>
<tr>
<td>Type III</td>
<td>189(61.96)</td>
<td>83(62.41)</td>
<td>272(62.18)</td>
</tr>
<tr>
<td>Type IV</td>
<td>64(20.98)</td>
<td>33(26.31)</td>
<td>97(23.64)</td>
</tr>
<tr>
<td>Total</td>
<td>305</td>
<td>133</td>
<td>438</td>
</tr>
</tbody>
</table>
3. Gross types of gastric carcinoma

The macroscopical examination of gastric carcinoma according to the Borrmann's classification was utilized. The most common type was type III being 62.18% being two thirds of the total cases and the next is type IV 23. 64%. Type I was 3.71% (Table 9).

4. Histopathological findings of gastric carcinoma

(a) Histopathological types and classification

1) Gastric carcinoma, uniform

The 228 cases revealed uniform histological pattern (50.8%) and among these 158 cases were adenocarcinoma or 65.77%, diffuse anaplastic carcinoma 28.15%, mucinous adenocarcinoma 4.94% and signet-ring cell carcinoma 1.15% (Table 10-A).

Table 10-A. Histopathological classification

<table>
<thead>
<tr>
<th>Histologic type</th>
<th>Male (%)</th>
<th>Female (%)</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma, well diff.</td>
<td>123(56)</td>
<td>35(38.33)</td>
<td>158(68.77)</td>
</tr>
<tr>
<td>mod. well diff. adenocarcina.</td>
<td>20</td>
<td>6</td>
<td>26</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>49</td>
<td>9</td>
<td>58</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>54</td>
<td>20</td>
<td>74</td>
</tr>
<tr>
<td>Signet-ring cell ca.</td>
<td>3(4.94)</td>
<td>2(3.33)</td>
<td>11(6.54)</td>
</tr>
<tr>
<td>Diffuse anaplastic ca.</td>
<td>0(0.6)</td>
<td>1(1.17)</td>
<td>1(0.6)</td>
</tr>
<tr>
<td>with fibrosis</td>
<td>27</td>
<td>19</td>
<td>46</td>
</tr>
<tr>
<td>without fibrosis (solid)</td>
<td>6</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Subtotal</td>
<td>168</td>
<td>60</td>
<td>228(100)</td>
</tr>
</tbody>
</table>

2) Gastric carcinoma, combined

Histologically 211 cases (49.22%) showed combined gastric carcinoma, that is more than one type of gastric carcinoma were found in one individual. These gastric carcinomas were classified by the predominating type. In this combined type of gastric carcinoma, the undi-

Table 10-B. Histopathological classification

<table>
<thead>
<tr>
<th>Histologic type</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma, well diff., plus</td>
<td>7</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>muc. well diff. adenocarcina.</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>signet-ring cell ca.</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>diffuse anaplastic ca.</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Adenocarcina., mod. well diff.,</td>
<td>10</td>
<td>6</td>
<td>16</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>signet-ring cell ca.</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>diffuse anaplastic ca.</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>Adenocarcina., poorly diff., plus</td>
<td>34</td>
<td>17</td>
<td>51</td>
</tr>
<tr>
<td>mod. well diff. adenocarcina.</td>
<td>6</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>signet-ring cell ca.</td>
<td>6</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>5</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>diffuse anaplastic ca.</td>
<td>17</td>
<td>7</td>
<td>24</td>
</tr>
<tr>
<td>Signet-ring cell ca., plus</td>
<td>19</td>
<td>8</td>
<td>27</td>
</tr>
<tr>
<td>well diff. adenocarcina.</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>3</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>6</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>diffuse anaplastic ca.</td>
<td>8</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Mucinous adenocarcina, plus</td>
<td>11</td>
<td>21</td>
<td>32</td>
</tr>
<tr>
<td>mod. well diff. adenocarcina.</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>1</td>
<td>8</td>
<td>9</td>
</tr>
<tr>
<td>signet-ring cell ca.</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>diffuse anaplastic ca.</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Diffuse anaplastic ca., plus</td>
<td>41</td>
<td>35</td>
<td>76</td>
</tr>
<tr>
<td>well diff. adenocarcina.</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>mod. well diff. adenocarcina.</td>
<td>2</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>poorly diff. adenocarcina.</td>
<td>14</td>
<td>13</td>
<td>27</td>
</tr>
<tr>
<td>signet-ring cell ca.</td>
<td>22</td>
<td>18</td>
<td>40</td>
</tr>
<tr>
<td>mucinous adenocarcina.</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Adenocanthoma</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Subtotal</td>
<td>123</td>
<td>90</td>
<td>213</td>
</tr>
</tbody>
</table>

differentiated carcinoma are encountered frequently (Table 10-B).

(b) Relationship between Gross type and Histopathological classification of gastric carcinoma.

The gross type III or IV reveals more anaplastic type in histopathological features than gross type I or II (Table 11).
Table 11. Relationship between gross type and histopathological classification of gastric carcinoma
(uniform carcinoma)

<table>
<thead>
<tr>
<th></th>
<th>Type I (%)</th>
<th>Type II (%)</th>
<th>Type III (%)</th>
<th>Type IV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenocarcinoma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>well differentiated</td>
<td>9(60)</td>
<td>2(6)</td>
<td>14(10)</td>
<td>0</td>
</tr>
<tr>
<td>moderately well diff.</td>
<td>5(33)</td>
<td>13(38)</td>
<td>32(23)</td>
<td>5(13)</td>
</tr>
<tr>
<td>poorly differentiated</td>
<td>1(7)</td>
<td>13(38)</td>
<td>51(37)</td>
<td>13(36)</td>
</tr>
<tr>
<td>Mucinous adenocarcinoma</td>
<td>0</td>
<td>0</td>
<td>7(5)</td>
<td>6(16)</td>
</tr>
<tr>
<td>Signet-ring cell carcinoma</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1(3)</td>
</tr>
<tr>
<td>Diffuse anaplastic type</td>
<td>0</td>
<td>4(12)</td>
<td>33(24)</td>
<td>11(30)</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>34</td>
<td>137</td>
<td>36</td>
</tr>
</tbody>
</table>

(c) Lymphatic permeation

The investigation of 438 cases of gastric carcinoma revealed 62% of the cases to have remarkable lymphatic permeation and there was no lymphatic permeation in 1.08%. It was uncertain in 27.26% (Table 12).

(d) Relationship between lymphatic permeation and histologic type

According to the relationship between histologic type and lymphatic permeation, lymphatic permeation is more commonly found in adenocarcinoma than any other type and its severity was more so (Table 13).

(e) Penetration of the gastric wall

Among 446 cases of gastric carcinoma, 86.89% showed serosal involvement and it was only less than 5% that showed penetration of the gastric wall limited to the mucosa or submucosa so called superficial or early cancer (Table 14).

Table 12. Lymphatic permeation

<table>
<thead>
<tr>
<th>Degree</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent</td>
<td>2(0.67)</td>
<td>2(1.48)</td>
<td>4(1.08)</td>
</tr>
<tr>
<td>Uncertain</td>
<td>72(23.92)</td>
<td>41(30.60)</td>
<td>113(27.26)</td>
</tr>
<tr>
<td>Mild</td>
<td>144(47.84)</td>
<td>57(42.54)</td>
<td>201(45.19)</td>
</tr>
<tr>
<td>Moderate</td>
<td>54(17.94)</td>
<td>23(17.6)</td>
<td>77(17.55)</td>
</tr>
<tr>
<td>Marked</td>
<td>22(7.31)</td>
<td>5(3.73)</td>
<td>27(5.52)</td>
</tr>
<tr>
<td>Severe</td>
<td>7(2.32)</td>
<td>6(4.49)</td>
<td>13(3.31)</td>
</tr>
<tr>
<td>Total</td>
<td>301</td>
<td>134</td>
<td>435(100.00)</td>
</tr>
</tbody>
</table>

Table 13. Relationship between lymphatic permeation and histologic type (carcinoma, uniform)

<table>
<thead>
<tr>
<th>Degree of lymphatic permeation</th>
<th>Histologic type</th>
<th>Adenocarcinoma</th>
<th>Signet-ring cell ca.</th>
<th>Mucinous adenocarcinoma</th>
<th>Diffuse anaplastic ca.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Adenocarcinoma</td>
<td>Signet-ring</td>
<td>Mucinous adenocarcinoma</td>
<td>Diffuse anaplastic ca.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>W. M. P. %</td>
<td>cell ca. %</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>Mild</td>
<td>57.7 55.7 57.1</td>
<td>0</td>
<td>23.5</td>
<td>30.0</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>3.7   30.1 16.2</td>
<td>0</td>
<td>15.4</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Marked</td>
<td>3.7   1.4   8.4</td>
<td>0</td>
<td>7.6</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Severe</td>
<td>0     0     0</td>
<td>0</td>
<td>0</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>Uncertain</td>
<td>35.0 12.8 14.7</td>
<td>0</td>
<td>53.5</td>
<td>58.0</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>
Histopathological Studies on Gastric Carcinoma among Koreans

Table 14. Penetration of the gastric wall

<table>
<thead>
<tr>
<th>Degree</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transmural</td>
<td>276(86.61)</td>
<td>116(84.18)</td>
<td>392(86.89)</td>
</tr>
<tr>
<td>Muscular</td>
<td>23(7.47)</td>
<td>13(9.35)</td>
<td>36(8.41)</td>
</tr>
<tr>
<td>Submucosal</td>
<td>7(2.27)</td>
<td>4(2.87)</td>
<td>11(2.57)</td>
</tr>
<tr>
<td>Mucosal (superficial)</td>
<td>2(0.65)</td>
<td>5(3.60)</td>
<td>7(1.23)</td>
</tr>
<tr>
<td>Total</td>
<td>308</td>
<td>138</td>
<td>446</td>
</tr>
</tbody>
</table>

5. Metastasis

(a) Regional lymphnode and omental metastasis

Among 429 gastric carcinomas, 68.84% showed regional lymphnode metastasis and 21.39% direct dissemination to the omentum (Table 15).

Table 15. Regional lymphnodes and omental metastases

<table>
<thead>
<tr>
<th>No. of metastatic lymphnode</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>78(26.60)</td>
<td>33(25.38)</td>
<td>111(25.69)</td>
</tr>
<tr>
<td>1–4</td>
<td>113(37.67)</td>
<td>43(33.07)</td>
<td>156(35.37)</td>
</tr>
<tr>
<td>5–9</td>
<td>51(17.00)</td>
<td>27(20.77)</td>
<td>78(17.77)</td>
</tr>
<tr>
<td>10–14</td>
<td>22(7.33)</td>
<td>14(10.77)</td>
<td>36(8.05)</td>
</tr>
<tr>
<td>Over 15</td>
<td>17(5.67)</td>
<td>9(6.92)</td>
<td>26(6.29)</td>
</tr>
<tr>
<td>No regional lymphnode</td>
<td>19(6.33)</td>
<td>3(2.29)</td>
<td>22(4.31)</td>
</tr>
<tr>
<td>Omentum</td>
<td>62</td>
<td>30</td>
<td>92(21.39)</td>
</tr>
</tbody>
</table>

(b) Relationship between regional lymphnode metastasis and histologic type of gastric carcinoma

In general metastasis to regional lymphnode is more common in adenocarcinoma than other types and in combined carcinoma than in uniform carcinoma (Table 16).

(c) Relationship between regional lymphnode metastasis and lymphatic permeation.

It seems to be that the more the degree of lymphatic permeation is, the more metastasis to regional lymphnode occurs (Table 17).

6. The changes of the remaining stomach.

Chronic gastritis and intestinal metaplasia of stomach were observed to occur for the remaining tissue of the stomach. Chronic atrophic gastritis was found in 46 cases being 10.89%. In most of the patients with gastric carcinoma, the remaining portion of the stomach was accompanied with some degree of intestinal metaplasia and 80% cases showed intestinal metaplasia up to a moderate degree and in 20% it was marked (Table 18).

7. Relationship between intestinal metaplasia and histologic type of gastric carcinoma.

There is no relationship between intestinal metaplasia and the histologic type as intestinal metaplasia is frequently present in all histologic types of gastric carcinoma (Table 19).

Table 16. Relationship between regional lymphnode metastases and histologic type

<table>
<thead>
<tr>
<th>Histologic type</th>
<th>Regional lymphnode metastases (%)</th>
<th>Metastases (%)</th>
<th>No regional lymphnode</th>
<th>Number of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uniform</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenocarcinoma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>well diff.</td>
<td>12(46.1)</td>
<td>13(50.0)</td>
<td>1</td>
<td>26</td>
</tr>
<tr>
<td>mod. well diff.</td>
<td>15(26.2)</td>
<td>39(73.2)</td>
<td>2</td>
<td>56</td>
</tr>
<tr>
<td>poorly diff.</td>
<td>14(19.3)</td>
<td>56(80.6)</td>
<td>6</td>
<td>76</td>
</tr>
<tr>
<td>Signet-ring cell ca.</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Mucinous adenocarc.</td>
<td>5(38.5)</td>
<td>7(63.8)</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td>Diffuse anaplastic ca.</td>
<td>22(44.0)</td>
<td>25(50.0)</td>
<td>3</td>
<td>50</td>
</tr>
</tbody>
</table>
Combined

Adenocarcinoma

Well diff. 0 7 (100) 0 7

Mod. well diff. 3(15.0) 16(80.0) 1 20

Poorly diff. 15(23.8) 48(71.6) 4 67

Signet-ring cell ca. 6(24.0) 18(72.0) 1 25

Mucinous adenoc. 7(22.6) 23(77.4) 1 31

Diffuse anaplastic ca. 15(23.8) 59(76.0) 3 77

Sang Ho Cho, Yoo Bock Lee and Dong Sik Kim

(continued Table 16.)

<table>
<thead>
<tr>
<th>No. of regional lymphnode metastasis</th>
<th>Degree of lymphatic permeation</th>
<th>Uniform (%)</th>
<th>Severe (%)</th>
<th>Uncertain (%)</th>
<th>Combined (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Mild</td>
<td>27 (25.12)</td>
<td>3 (8.82)</td>
<td>0 (57.81)</td>
<td>37 (25.81)</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>15 (34.58)</td>
<td>7 (44.12)</td>
<td>1 (17)</td>
<td>32 (24.41)</td>
</tr>
<tr>
<td>1–4</td>
<td>Marked</td>
<td>27 (27)</td>
<td>7 (2)</td>
<td>2 (3)</td>
<td>3 (19)</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>8 (8)</td>
<td>4 (4)</td>
<td>1 (3)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>5–9</td>
<td>Uncertain</td>
<td>5 (5)</td>
<td>2 (2)</td>
<td>0 (6)</td>
<td>0 (4)</td>
</tr>
<tr>
<td>10–14</td>
<td>Total</td>
<td>107</td>
<td>34</td>
<td>10</td>
<td>6</td>
</tr>
<tr>
<td>over 15</td>
<td></td>
<td>64</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 17. Relationship between regional lymphnode metastases and lymphatic permeation

Table 18. Changes in remaining portion (intestinal metastasis)

<table>
<thead>
<tr>
<th>Degree</th>
<th>Male(%)</th>
<th>Female(%)</th>
<th>Total(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>102(35.05)</td>
<td>60(46.51)</td>
<td>162(40.78)</td>
</tr>
<tr>
<td>Moderate</td>
<td>122(41.92)</td>
<td>48(37.21)</td>
<td>170(39.57)</td>
</tr>
<tr>
<td>Marked</td>
<td>55(18.93)</td>
<td>21(16.28)</td>
<td>76(17.61)</td>
</tr>
<tr>
<td>Severe</td>
<td>12(4.12)</td>
<td>0</td>
<td>12(2.66)</td>
</tr>
</tbody>
</table>

DISCUSSION

With the constant research to conquer gastric carcinoma it has become possible to reduce its incidence rate and mortality rate by improving the methods for early detection, prevention of precancerous diseases, and early treatment.

The incidence rate of gastric carcinoma has been reported to be dependent upon the geographical and racial status obviously, though accurate statistics are not available.

By Kim et al. (1956), Kim et. al. (1962), and Lee et. al. (1967), the gastric carcinoma is the most frequent carcinoma in Korea excluding uterine cervical cancer. Lee et. al. (1968) investigating gastric carcinoma among Koreans during 10 years from 1958—1967 reported it as the second most common carcinoma in Korea. According to Suh (1963), gastric carcinoma was 1.2% of all hospitalized patients in Severance Hospital Yonsei University during 8 years since 1955. In 1967 Weill-Bousson and Rousselot reported that 38.6% of gastrectomies from 1925 to 1965 were for gastric carcinoma.

In this investigation 60% of 744 gastrectomies were due to gastric carcinoma which means that in Korea the incidence of gastric
Table 19, Relationship between intestinal metaplasia and histologic type

<table>
<thead>
<tr>
<th>Histologic type</th>
<th>Degree of intestinal metaplasia</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
<th>Uncertain</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uniform</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenocarcinoma</td>
<td>well diff.</td>
<td>14</td>
<td>7</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>mod. well diff.</td>
<td>17</td>
<td>22</td>
<td>12</td>
<td>2</td>
<td>3</td>
<td>56</td>
</tr>
<tr>
<td></td>
<td>poorly diff.</td>
<td>25</td>
<td>31</td>
<td>19</td>
<td>0</td>
<td>1</td>
<td>76</td>
</tr>
<tr>
<td></td>
<td>Signet-ring cell ca.</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Mucinous adenoca.</td>
<td>5</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>Diffuse anaplastic ca.</td>
<td>22</td>
<td>17</td>
<td>5</td>
<td>2</td>
<td>4</td>
<td>50</td>
</tr>
<tr>
<td>Combined</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenocarcinoma</td>
<td>well diff.</td>
<td>5</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>mod. well diff.</td>
<td>5</td>
<td>6</td>
<td>2</td>
<td>0</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>poorly diff.</td>
<td>20</td>
<td>27</td>
<td>10</td>
<td>3</td>
<td>0</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>Signet-ring cell ca.</td>
<td>9</td>
<td>8</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td></td>
<td>Mucinous adenoca.</td>
<td>10</td>
<td>15</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>Diffuse anaplastic ca.</td>
<td>35</td>
<td>22</td>
<td>13</td>
<td>2</td>
<td>3</td>
<td>75</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>167</td>
<td>163</td>
<td>76</td>
<td>12</td>
<td>18</td>
<td>436</td>
</tr>
</tbody>
</table>

carcinoma has not been reduced yet, contrary to the report of its marked decrease in the United States of America and Europe by Ochsner(1962) and Moore(1962).

The incidence rate of gastric carcinoma by the age group was at its peak in the 50's accounting 35.77%, and the next was 40's, 60's, and 30's in that order of frequency. This is consistent with the report of Lee et. al. (1968) in their statistics of malignant tumors among Koreans, Jemerin and Colp(1952), and Mitty et. al. (1960). Yamagata et. al.(1961) and Deguchi(1967) also reported 50's as the most common age group. Shahon et. al.(1966) and Ochsner(1962) reported 60's, 50's, and 70's in that order of frequency. The average age on our article is at age 51. In comparison with the statistics from other countries, the peak age group is the same 50's, but the age for development of carcinoma seems to be somewhat younger. However, comparing with the 40's reported previously as the peak age group by Suh(1963) and Kim(1961), it appears that the age of incidence has been somewhat delayed. Therefore we realized the age of patients of gastric carcinoma is a little delayed with the improvement of various environmental factors. But this is a different story from the report of Yokoyama et. al.(1961) who say the age for early gastric carcinoma is about 10 years earlier and it is considered to be due to extension of life span, improvement of diet and amelioration of the social status.

In the sex distribution of gastric carcinoma, the male and female ratio reveals 2.28 to 1 and this is consistent with the results of Harvey et al.(1951), 2 : 1, Jemerin and Colp(1952), 2.3 : 1, Mitty et. al.(1960), 2 : 1, Ikeuchi et. al.(1961), 2.5 : 1, Ochsner(1962), 2.1 : 1, Donhauser(1962), 2.1 : 1, Kim et. al.(1964), 3.5 : 1, in that male is predominantly susceptible to gastric carcinoma. According to our
statistics, the male and female ratio for all kinds of malignant tumors is 1 to 1.1 and comparing with this it is obvious that the ratio of 3.28 : 1 for gastric carcinoma is of its own finding not affected by the ratio of male and female of hospital population.

The blood group has been raised as one of the genetic factors among many possible etiologic components for gastric carcinoma. McConnell(1960) and Glass(1965) reported gastric carcinoma and pernicious anemia are more commonly in the blood group A. In this study, the percentage of group A in gastric carcinoma is 37.24% and O 23.93%, while in general population group A is present in 31.15%, B in 28.60%, O in 30.20% (Lee,1969) and AB in be more common in group A and less common in O, however this difference proved not to be significant.

There are no specific clinical symptoms in the early stage of gastric carcinoma and the patients usually seek medical aid when carcinoma is somewhat advanced. Among the main subjective complaints described by the patients when first seen by the doctor, although they are a little different according to the location of the lesion, in most cases they complained of upper abdominal pain and/or discomfort(86%) and indigestion(67%). In literature, upper abdominal pain or discomfort is the main complaint in 71.8% by Jemerin and Colp(1952), 56.1% by Abrahamson and Hinton(1947), 81.3% by Ochsner (1962). Shimizu(1967) and Yamagata et. al. (1967), in their reports for early gastric carcinoma, also described upper abdominal pain as the most common symptom. Weight loss(35.7%), vomiting(35%), palpable mass in upper abdomen, loss of appetite, general malaise, sense of fullness of the upper abdomen and dysphagia are the other common symptoms.

As for the duration of chief complain, the patients first seen by the doctor within the first three months of symptoms was 35.1% in our survey and 12% by Harvey et al. (1961), 14% by Jemerin and Colp(1952), 29.2% by Shahon et al. (1956) in their reports. 54.7% had complaints for six months, while by Jemerin and Colp (1952) 31% and by Shahon et al. (1956) 31%. The duration of chief complain in the authors’ survey is shorter than that from Europe and United States of America, but the gastrectomy rate in our hospital is lower than that of other countries(Suh, 1963). The reason seems that the patients present in the later stage of the disease with the severe symptoms having neglected those light symptoms such as indigestion and discomfort etc. Though test of acidity of the gastric juice is not specific for gastric carcinoma, it has been strongly suggested that achlorhydria precedes gastric carcinoma, therefore it plays an important role in the diagnosis of gastric carcinoma.

Approximately half of the patients with gastric carcinoma fail to secrete gastric acid to histamine. 18% of these cases with achlorhydria proved to be carcinoma (Grossman, Kirsner and Gillespie, 1963; Fishermand and Koster, 1962). According to Jemerin and Colp (1952), the cases of achlorhydria are 62.5% and hypochlorhydria 19.0%. Such decrease of gastric acid secretion in one of the significant findings in gastric carcinoma. Fortunately Comfort, Kelsey and Berkson (1947) realized in the early days that hypochlorhydria is much more common(47.8%) in a group of patients who two years later turned out to have gastric carcinoma and at the time of diagnosis 68.8% of those proved to be achlorhydric. Moreover, Berkson, Comfort and Butt (1956) recently reported the six times higher incidence rate
of gastric carcinoma in 800 cases of achlorhydria not associated with pernicious anemia.

The most common location for gastric carcinoma seems to be the pyloric area in the authors' study being 45—76%, and is consistent with the findings of Stewart(1931), Poschariansky(1930), Boyd(1946), Bellavia and Mascolo(1956), Willis(1957), Trimble and Lynn (1955), Harvey et. al(1951), Ikeuchi et. al. (1962). Eker(1951) reported that most of the ulcerocarcinoma develop in the pylorus while polyoid and expansive type in fundus. Since the majority of gastric carcinoma are present in the lower half of the stomach, Ochsner(1962) felt that radical subtotal gastrectomy leads to good results in most cases of gastric carcinoma.

The diameter of the tumor mass measured 3—5cm. in 43.11% and 6—10cm. in 42.65% thus the majority were between 3—10cm. Mulligan and Rember(1954) had the same result, their group being 70%. Urban et. al.(1955) in the evaluation of the relationship between the size of tumor and postoperative survival rate, reported that 53% of long term survival patients who survived more longer than five years had the tumor of less than five cm in diameter and not a single patient survived more than five years whose tumor size was larger than 15cm in diameter and they emphasized that the degree of tumor invasion plays an important role in the prognosis.

The macroscopical examination is based on Borrmann's classification. According to Schindler et. al.(1941) and Brown et. al.(1961), most of the cases of five year survival belong to type I and II. McNeer et. al.(1958) reported that type III was the most frequent type at the time of operation and five year survival rate is 50% in type I and II. In the authors' study, type III was 62%, type IV 21% and type I and II 14.9% and considering the infiltrating type of carcinoma has worse prognosis than localized one, the prognosis of these patients is expected to be discouraging. The reason for this seems to be that in Korea the operation for gastric carcinoma is performed in the full blown stage.

As precancerous diseases, achlorhydria, atrophic gastritis, intestinal metaplasia of the stomach, and peptic ulcer are considered to be important.

Since Konjetzny(1921) at first suggested the pathological correlation of chronic gastritis and gastric carcinoma, Hurst(1933) reemphasized the probability of chronic gastritis to give rise to malignant change to gastric carcinoma. Recently some pathologists put significant on chronic atrophic gastritis characterized by atrophy of mucosal layer, infiltration of the inflammatory cells and intestinal metaplasia of the stomach (Guiss and Stewart, 1943; Morson, 1956). In spite of that, it is true to observe gastric carcinoma to arise more frequently when there is severe atrophic changes in the gastric mucosa. Nakamura et al.(1965) emphasized the importance of intestinal metaplasia of the stomach reporting that 84.8% of gastric carcinoma develops from intestinal type epithelium and 12.1% from gastric mucosa.

Though there are different opinion of the frequency, it seems to be that some gastric cancer develops from chronic peptic ulcer. Stewart(1955) has established the pathological criteria of gastric carcinoma arising from benign gastric ulcer as that it should take a chronic course and there should be destruction of muscular layer, formation of scar, obstructive endarteritis at the surrounding area of the ulcer and organized thrombus of the vein, and cancer cells should be present at the margin of the ulcer. According to the recent pathological
reports, 5–18% of gastric carcinoma arises from the chronic benign gastric ulcer (Harnett, 1947; Gomori, 1933; Waugh and Charendoff, 1952; Mallory, 1940; Nakamura et al. 1967). Among these cases some are confined to the mucosal layer spreading superficially and others to the muscular layer having the cancer cells as the base of the ulcer. According to Stewart (1955) more than 88% of these ulcer cancer patients have clinical symptoms referring to gastrointestinal tract for more than two years and 57% for five years and free gastric acid was found in 89%.

The authors of this article observed nine cases of such ulcer-cancer (2.1%). All of the lesions were located at the lesser curvature of the pylorus and the size was within the range of 3–5 cm in diameter. Five cases showed hyperacidity and the remainder were normal in its acidity. The clinical symptoms persisted over a period of one year and in the longest one as long as six years.

Achlorhydria, chronic atrophic gastritis, benign peptic ulcer, all of these are considered to give damage to the gastric mucosa and the damaged mucosa is the good environment for cancer to develop. The injury to the mucosa gives rise to the mutation of cells and facilitates the contact with carcinogen. The atrophy of the epithelial cell is in another word maturation defect and having lost its function as a mucus barrier it is much easier for carcinogens to penetrate in through gastric mucosa. Morson (1956), Heinkel et al. (1960) reported the intestinal metaplasia of stomach as an important finding in atrophic gastritis. According to Teir and Räsänen (1961) the cell division rate in the mucosa of the gastric pylorus having intestinal metaplasia it is twice higher than that in normal. In other words the cellular turnover is very rapid. Teir and Räsänen (1961), Nagoya et al. (1965) in their recent pathological investigations reported that severe intestinal metaplasia is usually found in the well-differentiated carcinoma and not in the undifferentiated one. By Nieburgs and Glass (1963), the patient of gastric carcinoma has maturation arrest in the gastric mucosa and finally atrophic gastritis brings about the decrease in secretion of the gastric juice.

In recent days vigorous investigations have made in the fields of histochemistry, particularly in those of enzymes and mucous substance. In those study it seems that aminopeptidase doesn’t exist in normal gastric mucosa, however, it increases in gastric carcinoma tissue and in the area of intestinal metaplasia of stomach as it does in small bowel (Wattenberg, 1965; Planteydt and Willighagen, 1968; Planteydt, 1964; Ming et al., 1967). The active study has been carried out in order to extract certain enzyme histochemical method from the tissue of gastric carcinoma and intestinal metaplasia of the stomach as well as in chronic atrophic gastritis, and it will be worth while to do it.

Whether there is a metastases to the regional lymphnodes or not is the most important finding to determine the prognosis and 5-year survival rate is largely depend upon it. The frequency of metastasis to the regional lymphnodes at the time of surgery is reported to be 77.5% by Coller et al. (1941), 70.3% by Shahon (1956), 55% by Lehmann et al. (1958), 61% by Marshall (1958) and they all show similar findings. Without regional lymphnodes metastases, the 5-year survival rate is 44% by Shahon et al. (1956), 44% by McNeer et al. (1958), 34.8% by Marshall (1958), and 49.5% by Ransom (1933). On the contrast with metastases to regional lymphnodes, it is 11.4%, 14.8%, 7.2%, and 28.0%, respectively. The relation between gross type of cancer and metastases to regional lymph-
phnodes according to the literature is as follows;

<table>
<thead>
<tr>
<th>Polypoid</th>
<th>Ulcerative</th>
<th>Ulcerative &amp; infiltrating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coller (1941)</td>
<td>60%</td>
<td>66.6%</td>
</tr>
<tr>
<td>Eker (1951)</td>
<td>70%</td>
<td>90%</td>
</tr>
<tr>
<td>Fly (1956)</td>
<td>11.1%</td>
<td>34.7%</td>
</tr>
</tbody>
</table>

If there is no infiltration of tumor cells in the serosa, the prognosis is favorable. McNeer et al. (1958) reported the 5-year survival rate as 20.8% in the case having serosal infiltration and 34.1% of cancer is limited to the mucosal and muscular layers.

As for the histopathological classification of gastric carcinoma as Stout (1953) has pointed out, all gastric carcinoma belongs to adenocarcinoma. As the differential degree is variable, the histopathological classification was made based on the amount of differentiation. However few cases belong to those category which are completely differentiated and forms the glands, or those which are not differentiated at all, but majorities are the between cases of two extreme and a certain type of cancer secretes mucin intra or extracellularly. The relationship between this histopathological pattern and the prognosis has been also actively investigated. According to the report of Steiner et al. (1948) referring to the morphological factors of 5-year survival after gastrectomy due to gastric carcinoma, the prognosis is good in Borrmann's type I, and also in rather poorly differentiated carcinoma which has abundant stromal tissue of tumor mass and accompanied by chronic inflammatory reaction and also in the case where there is degeneration and atrophy of the tumor and not having regional lymphnodes metastases. Brown et al. (1961) stated that to be in 5-year survival it should be adenocarcinoma either ulcerative or polypoid in type. According to Urban and McNeer (1959), carcinoma has favorable outlook, when the tumor mass is less than 5 cm. in diameter, confined to the stomach wall and there is no regional lymphnode metastases and that has few signet-ring cell in it. The metastases to the regional lymphnodes and involvement of serosa were found four times more frequently in those group who expired within a year, and those which has undifferentiated glands and abundant signet-ring cells were three times higher in its frequency. Fisher and Hoerr (1955) in their study on the relation between morphology of gastric carcinoma and the metastases revealed that gastric carcinoma of any histopathological type is capable of metastases and there is no certain relationship between them but adenocarcinoma and medullary carcinoma generally brings about wide spread. As for the relationship between histopathological type and the degree of infiltration, superficial spread is most frequently observed in adenocarcinoma, none of scirrhous or mixed carcinoma showed superficial spread and the degree of infiltration is not affected its histological classification. The adenocarcinoma in general is most malignant and the next is medullary carcinoma, carcinoma simplex, and scirrhous carcinoma in that order. Mulligan and Rember (1954) made the histopathological classification of gastric carcinoma as mucous cell carcinoma, pyloric gland cell carcinoma and intestinal cell carcinoma. The mucous cell carcinoma is composed of mucous secreting signet-ring cell and undifferentiated cells. It is frequent before the age of 40 and it is large size, flat in shape and it often accompanies diffuse infiltration and metastases. On the other hand, pyloric gland cell carcinoma has well differentiated glandular structure and frequent in male after the age of 40. It is usually limited to antrum.
and cardia and either fungating or ulcerating in shape and less prone to metastasize than mucous cell carcinoma. The intestinal cell carcinoma is usually composed of both well and poorly differentiated adenocarcinoma, frequent in female, after age of 40. It develops commonly in the body of stomach and it is polypoid in type and seldom metastasizes. The mortality rate is 98% in mucous cell carcinoma, 75% in pylorocardiac gland cell carcinoma and 60% in the intestinal cell carcinoma. The relationship between the histopathological classification and their prognosis does not much differ from one article to another. The authors were observed adenocarcinoma as the most frequent type of gastric carcinoma and many of the remainder are those mixed with more than two different kinds of carcinomas, or mixture of undifferentiated carcinomas. Regional lymphnodes metastases was most common in adenocarcinoma and metastases was frequently in the combined type of gastric carcinoma. The lymphatic permeation was also frequently encountered in adenocarcinoma and the more the lymphatic infiltration it has, the more the metastases occurs.

The histopathological pattern and the degree of differentiation of gastric carcinoma and the intestinal metaplasia seemed to be well associated together without having any special relationship each other.

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Fig. 1. Well differentiated adenocarcinoma. H-E stain. 100X.

Fig. 2. Well differentiated adenocarcinoma. H-E stain 100X.

Fig. 3. Moderately well-differentiated adenocarcinoma. H-E stain. 100X.
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**Fig. 4.** Poorly differentiated adenocarcinoma. H-E stain. 100×.

**Fig. 5.** Mucinous adenocarcinoma. H-E stain 100×.

**Fig. 6.** Signet-ring cell carcinoma. H-E stain. 100×.
Fig. 7. Diffuse anaplastic carcinoma. H&E stain. 100X.

Fig. 8. Diffuse anaplastic carcinoma. H&E stain. 100X.

Fig. 9. Severe lymphatic permeation in the submucosal lymphatics. H&E stain. 100X.
Fig. 10. Chronic superficial gastritis. H-E stain. 100X.

Fig. 11. Chronic atrophic gastritis. H-E stain. 40X.

Fig. 12. Severe intestinal metaplasia of the gastric mucosa. H-E stain. 100X.