CARCINOMA

OF THE

STOMACH

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CONTENTS

"Of all the diseases which affect mankind the cancer is the most grievous and rebellious and is generally incurable....."

Richard Wiseman, 1676.

"All knowledge is of itself of some value. There is nothing so minute or inconsiderable that I would not rather know it than not."

Dr. Samuel Johnson.

A review of the clinical and pathological features exhibited by 2,000 consecutive patients with carcinoma of the stomach has been made in an endeavour to enquire into the natural history of the disease and to determine those factors which may be of prognostic value.

These 2,000 patients represent the total cancer clinic experience of carcinoma of the stomach in the Province of Saskatchewan during the years 1932-55, inclusive.

INTRODUCTION

Saskatchewan is a large Canadian province which covers an area approximately two and a half times that of the entire United Kingdom, but boasts a population considerably less than that of the city of Montreal.

In its half century of existence as a self-governing province many changes have occurred affecting the welfare of its population. Not the least beneficial of these was the institution of the Saskatchewan Cancer Commission which was created by the provincial government, at the request of the medical profession, in 1930, to advise on the measures which should be adopted to ensure notification, diagnosis and treatment of cancer.

As a result of the recommendations made, the provincial government provided two well equipped cancer clinics, one in Regina in the south, and the other in Saskatoon in the

northern part of the province. All physicians practising within the province and all hospitals receiving government financial aid were required, by the Cancer Control Act, to notify all cases of cancer coming within their responsibility. An additional source of confirmatory information from which the incidence of cancer in the province could be computed was the legislation providing for the compulsory examination by competent pathologists of all tissue removed at operation. These pathological records are available for survey.

From a modest but ambitious beginning the cancer organisation developed, and in 1944 attained some degree of maturity when the Provincial Government introduced and passed legislation to make the cancer service entirely free to residents of the province. With the establishment of free diagnostic and treatment facilities and free hospitalization, all financial barriers to the early diagnosis and treatment of cancer were removed.

The clinics form a separate and distinct unit in the large general hospitals in which they are situated, and are staffed by full time salaried specialists, including internists, radiotherapists and a surgeon.

No patient is admitted to the clinics unless he has been referred by his private physician, and then only when a diagnosis of cancer has been made or is suspected. This provision was made to ensure that the efficiency of the clinics would not be diluted by those members of the public who wished to avail themselves of a free medical service, but in whom there was little symptomatic evidence to support a diagnosis of cancer. Moreover, admission to the clinics without referral would have defeated the wish of the staff of the clinics to co-operate rather than compete with their colleagues in private practice. The cancer organisation is based on the goodwill of the profession as a whole, on its effort to control and improve the diagnosis and treatment of cancer, and on the belief that the general practitioner is an integral part of the service and its first line in cancer detection.

When each patient has been fully investigated in the clinics, a complete record of his history and the results of his clinical and ancillary examinations are presented at a daily staff conference where the diagnoses and the best forms of treatment are discussed.

Every patient, whether he has received treatment or not, is expected to return to the clinics for examination at regular intervals until death. An effort is made to obtain autopsy examination in as many cases as possible. The completeness of the review system adopted may be measured by

the fact that, in the present survey on gastric cancer, up-to-date records were obtained from the clinic charts in all but two patients, representing a follow-up failure of only one tenth of one per cent.

When patients arrive at a terminal phase of their disease and are unfit to come to the clinics for review examination, their private physicians assume responsibility for their care and in return for their services and for submitting regular medical reports, are paid by the Cancer Commission on a fee for service basis.

The cost of the cancer services in Saskatchewan has risen steadily each year, and while its total may appear formidable, it has never exceeded $1.82 per head of the population.

The data presented in this review have been drawn from the records of the Saskatchewan Cancer Commission.

CHAPTER I

THE INCIDENCE OF GASTRIC CANCER

In the voluminous literature relative to the many problems associated with the study of gastric cancer, there is a marked paucity of authoritative data on the incidence of the disease. The comparative absence of information does not reflect a lack of interest, but is an expression of the difficulty in obtaining data which would satisfy statistical demands and which would allow for the calculation of incidence rates in terms of specific population groups exposed to the disease. Studies of incidence based on the experience of large hospitals and clinics are unreliable because the rates obtained cannot be related to the population as a whole. Similarly, mortality records are not a valid source of acceptable data because they document only those cases in which treatment has been unsuccessful, and give no indication of the number of new cases which arise in any one year within a population

of known size and whose age and sex distributions have been determined. Moreover, mortality data are suspect because the recorded cause of death in many cases is unsupported by microscopic or other proof and occasionally may be based only upon supposition.

The only method whereby a reasonably accurate incidence of gastric cancer can be ascertained is by the study, over many years, of the frequency with which the disease arises in a definite and defined population group. While this is the obvious approach, it is not readily attainable because cancer, for the most part, is not a notifiable disease, and few cancer organizations are equipped to discover all of the cases of cancer which occur within their province. As a result, information on actual incidence tends to be fragmentary. It is therefore important to measure the degree in which the case finding programme, within a known population group, is successful.

In 1948, Watson conducted a painstaking survey of the incidence of cancer in Saskatchewan and found that, by examining all hospital records and pathological records in the province for that year, eighty-five per cent of patients with cancer had been referred to the cancer clinics. There was substantial evidence that of those cases not referred to the Cancer Commission, the major proportion was of the less lethal varieties of the disease. It has been estimated that since 1948, more than ninety percent of patients with cancer in the province, each year, have been investigated within the

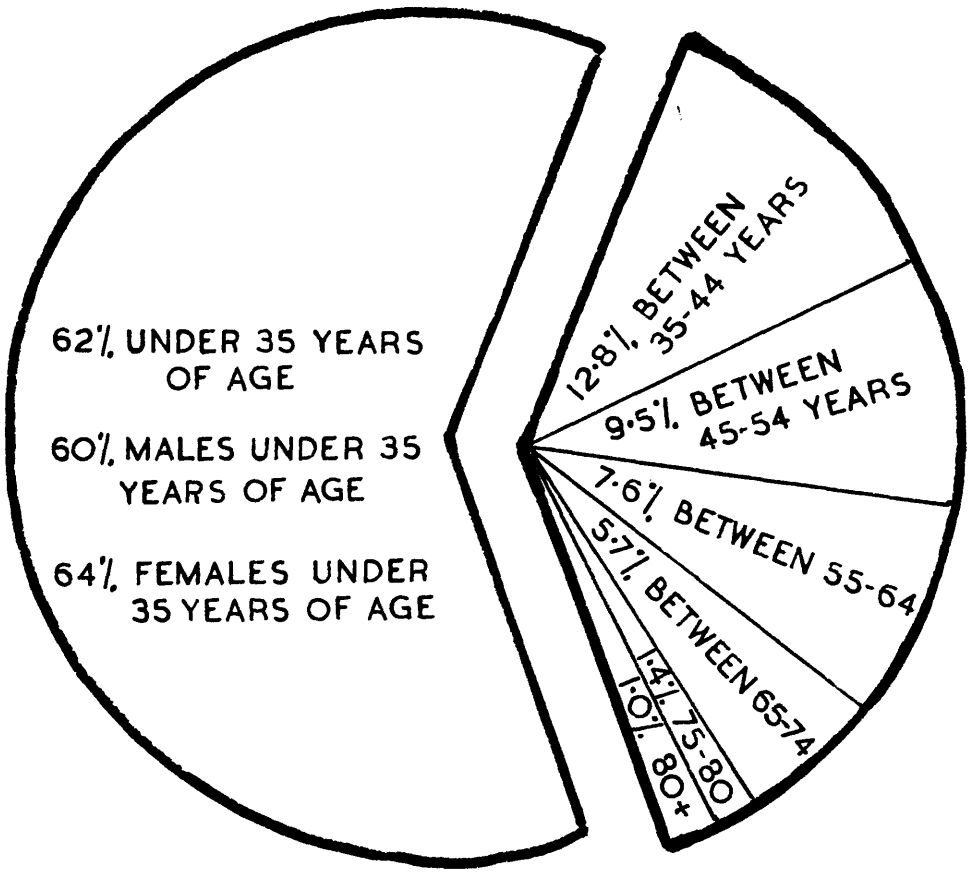
cancer organization. It would appear that with access to the well documented records of these patients a reasonable estimate of the incidence of gastric cancer occurring in Saskatchewan can be made.

For purposes of accuracy the period following the introduction of the comprehensive and free service has been taken to form the basis of the calculation of incidence. Because statistical studies of incidence must necessarily be limited to the collection of data concerning diagnosed cases, only those patients investigated at the clinics will be considered in the analysis.

THE POPULATION IN SASKATCHEWAN

The average population in Saskatchewan during the period between 1945 and 1955 was 845,000. The proportion of males to females was 1.1 to one. Sixty-two percent of the total population was under the age of thirty-five years, with sixty percent of the males and sixty-four percent of the females occupying the younger age group (Fig. 1). There was very little alteration in the age pattern between the years 1945 and 1955, the only notable change being a slight increase in the proportion of the population over the age of sixty years, from 10.4 percent to 12.2 percent. It is apparent from this age distribution that the population of Saskatchewan is predominantly young and a marked minority is in the age group in which cancer occurs most commonly. As a direct result of this, it would be

AVERAGE POPULATION 845,000



$$\frac{\text{MALE}}{\text{FEMALE}} \quad \frac{1\cdot1}{1}$$

FIGURE 1

expected that the incidence of cancer in general, and of gastric cancer in particular, would be much lower than that reported for the population in other geographic regions in which the age distribution curve exhibits a preponderance in the older age groups.

CANCER INCIDENCE IN SASKATCHEWAN

During the years between 1945 and 1955 there was a moderate increase in the total incidence of cancer in the province of Saskatchewan. The average incidence over the last five years of the survey was 213 per 100,000 of the population, with the respective incidences in males and females being 242 and 181. The total incidence corresponds with the seven year average incidence of 207.8 per 100,000 reported by Macdonald of Connecticut in 1948, but is distinctly less than that reported by Dorn (1944) who gave an incidence rate of 230 new cases annually per 100,000 of the population. It is of interest to note that in the present series the incidence in males is much higher than in females (Table 2). Both Macdonald and Dorn report the incidence among females to be much higher than in males. In the Connecticut series, which represents the experience of cancer incidence in a population of approximately three quarters of a million people and which corresponds in number to the population of Saskatchewan, the incidence of cancer of all

AVERAGE INCIDENCE OF ALL PRIMARY

CANCER 1951-55 IN SASKATCHEWAN

213 PER 100,000

MALES 242 PER 100,000

FEMALES 181 PER 100,000

TABLE I.

YEAR	INCIDENCE PER 100,000 BOTH SEXES (CRUDE)	INCIDENCE PER 100,000 MALES (CRUDE)	INCIDENCE PER 100,000 FEMALES (CRUDE)
1945	124	139	109
1946	143	159	126
1947	163	181	142
1948	169	193	143
1949	184	212	154
1950	192	216	167
1951	197	212	182
1952	219	251	186
1953	209	244	170
1954	215	244	183
1955	224	259	186

INCIDENCE PER 100,000 ALL CANCER

SASKATCHEWAN 1945-54

TABLE 2

sites in males was 194.4 per 100,000 and in females 221.2 per 100,000. The only reason which can be offered for the difference in sex distribution of cancer between the Saskatchewan and Connecticut studies is the marked preponderance of young females in the general population of the former region.

THE DISTRIBUTION OF CANCER ACCORDING TO SITE IN SASKATCHEWAN

Examination of the records of the Cancer Commission reveals that, in the twenty-four year interval between 1932 and 1955 the skin was the commonest site of cancer. Of 23,984 primary cancers in 22,438 patients 21.3 per cent of the lesions were located in the skin. The gastro-intestinal tract was primarily involved in 20.5 per cent of the cases, constituting the second most frequently encountered site of the disease. (Table 3).

A detailed examination of the relative incidence of cancer involving the digestive system at various levels reveals that the stomach is the site of cancer in more than two-fifths of all gastro-intestinal lesions. (Table 4).

THE YEAR TO YEAR INCIDENCE OF GASTRIC CANCER, WITH COMPARISON OF CANCER INVOLVING CERTAIN OTHER SITES (Fig. 4, 5 and 6).

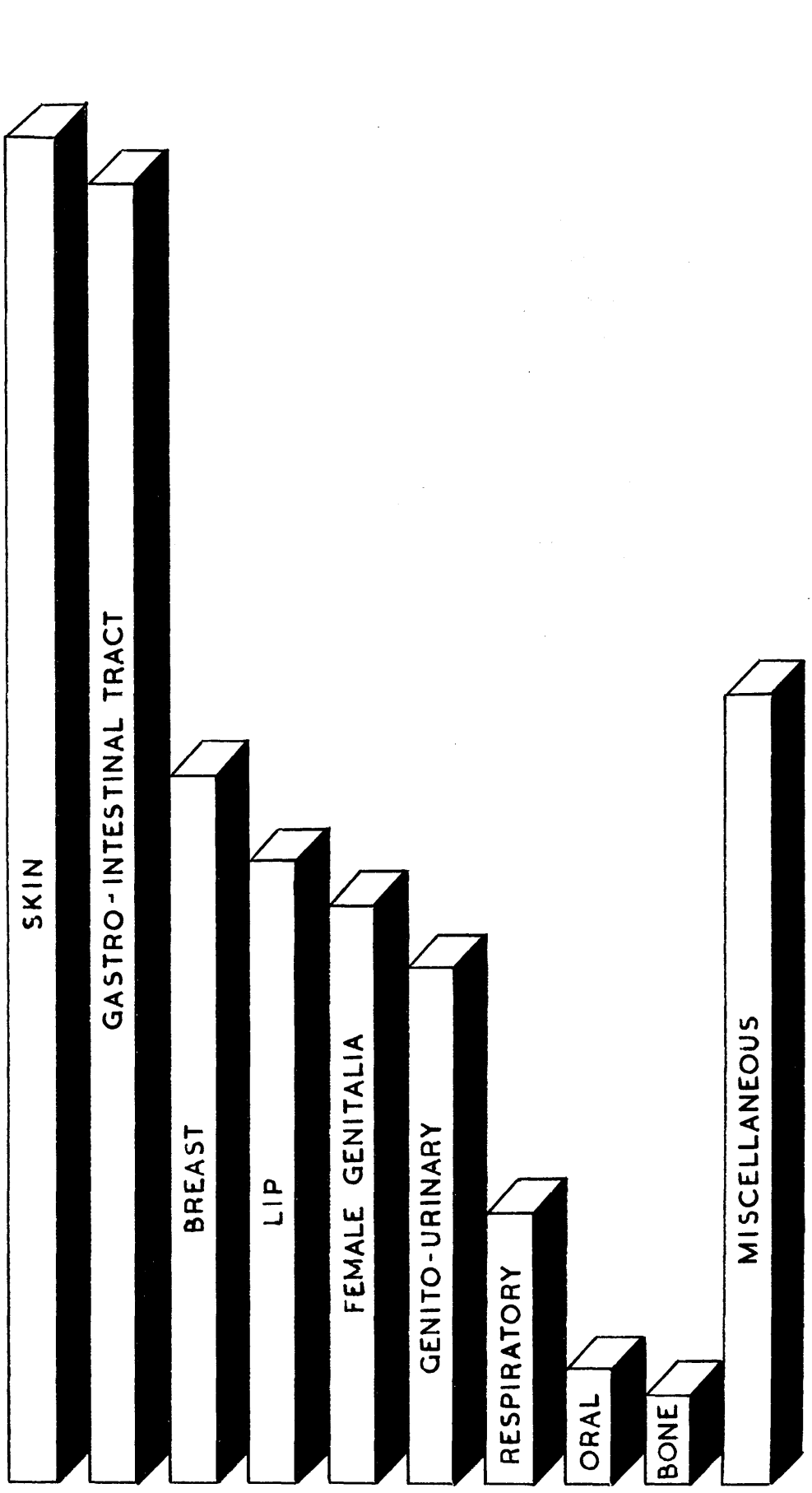
Study of the crude rates of incidence of cancer in various sites relative to that of gastric cancer reveals the

SITE	NUMBER OF CASES	PER CENT OF TOTAL
Skin	5097	21.3
Gastro-intestinal	4917	20.5
Breast	2699	11.3
Lip	2349	9.8
Female Genitalia	2195	9.2
Genitourinary	1956	8.1
Respiratory	1025	4.3
Oral	453	1.9
Bone	349	1.4
Miscellaneous	2944	12.2
Total	23,984	100.0

DISTRIBUTION OF 23,984 PRIMARY CANCERS IN 22,438 PATIENTS

SASKATCHEWAN 1932-55

TABLE 3



DISTRIBUTION OF 23,984 PRIMARY CANCERS
IN 22,455 CONSECUTIVE PATIENTS.
SASKATCHEWAN 1932-1955.

FIGURE 2

SITE	NUMBER OF CASES	PER CENT OF TOTAL
Stomach	2,000	40.7
Colon	1,078	21.9
Rectum and Anus	958	19.5
Pancreas	363	7.4
Gall Bladder, Bile Ducts and Liver	293	5.9
Esophagus	201	4.1
Small Intestine	24	0.5
Total	4,917	100.0

DISTRIBUTION OF 4917 SEPARATE PRIMARY CANCERS OF THE GASTROINTESTINAL TRACT

SASKATCHEWAN 1932-55

TABLE 4

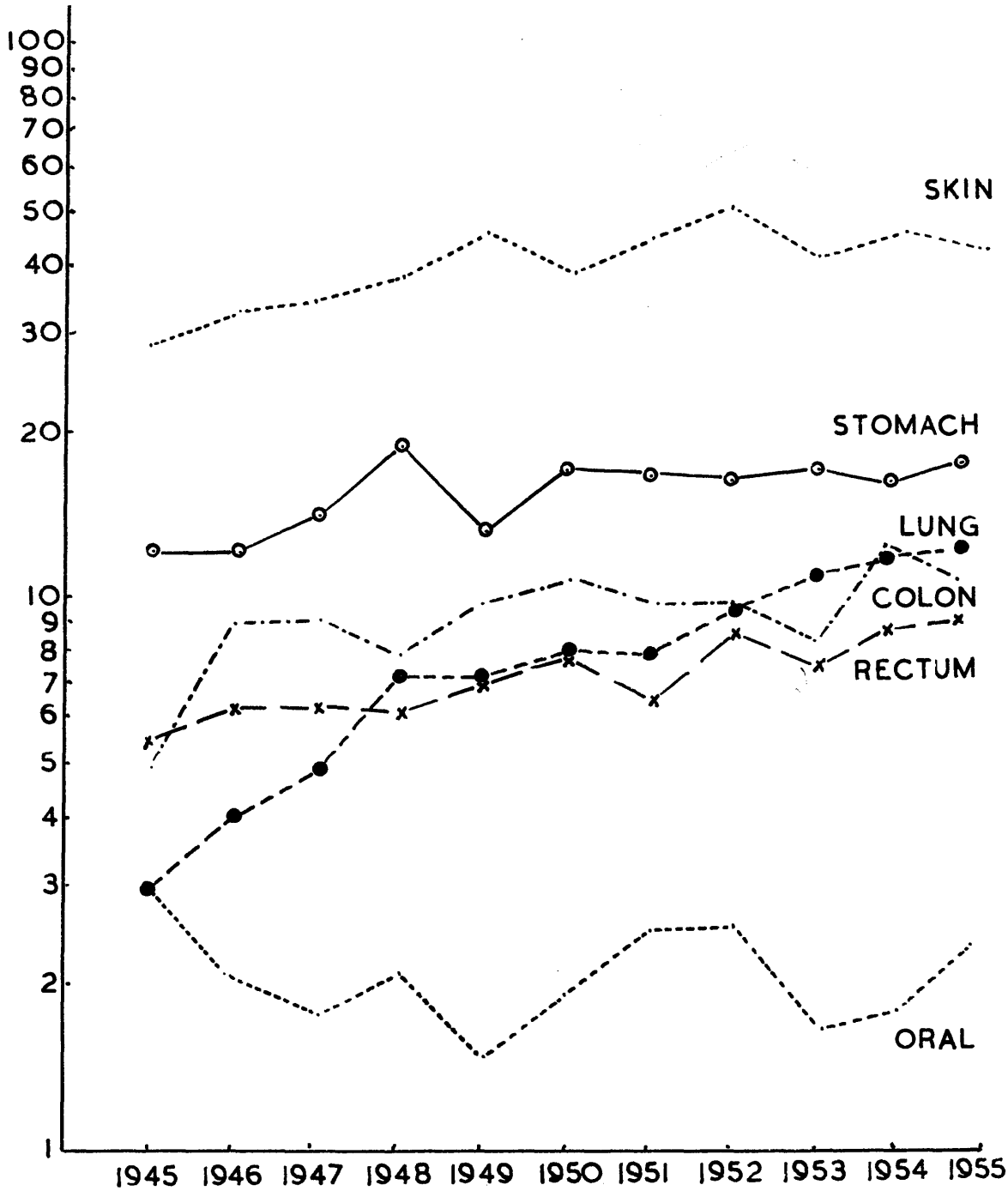
DISTRIBUTION OF 4917 PRIMARY CANCERS OF THE GASTRO-INTESTINAL TRACT. SASK. 1932-55

FIGURE 3

interesting finding that, from year to year, the incidence of each regional cancer is similar with the exception of the disease as it affects the bronchi and lung parenchyma. A pronounced increase in the incidence of respiratory cancer is evident during the eleven year period between 1945 to 1955 with the incidence, especially in males, approaching that of gastric carcinoma in the last year of the survey. It is probable that this increase each year, is due in greater part to increased vigilance by the general medical practitioners and improved diagnosis, rather than to an actual dramatic increase in the frequency of the disease over the period.

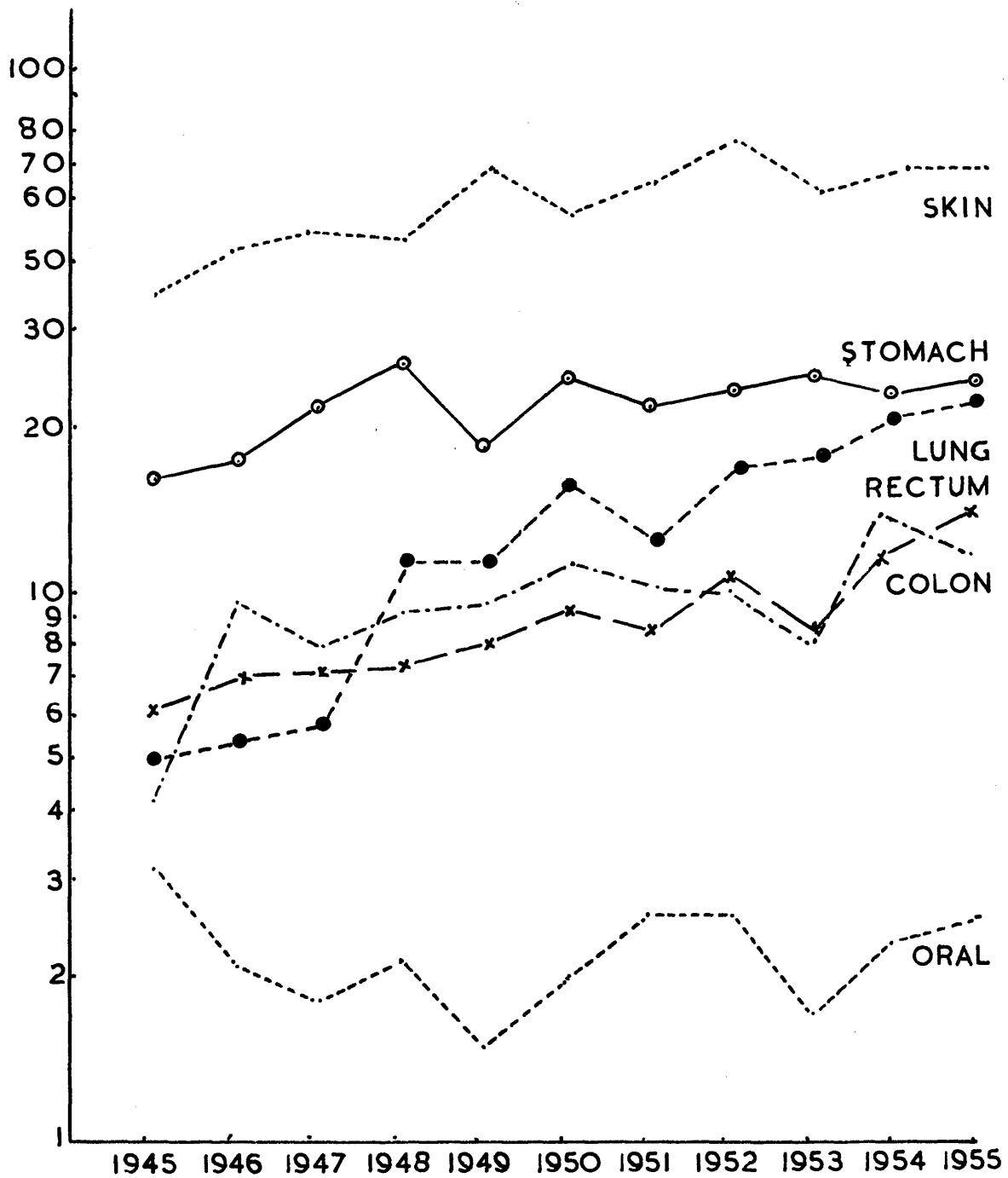
THE SEX INCIDENCE OF GASTRIC CANCER

It is generally recognised that cancer of the stomach occurs more frequently in males than in females, but there is considerable disparity between the various estimates of the comparable incidence. In the Saskatchewan series males were affected approximately three times more frequently than females. Calculated on the sex rates of incidence of the disease, the ratio of male to female involvement was 2.5 to one. In her survey in Connecticut, Macdonald (1948) found the sex ratio to be 1.9 to one, with the incidence in males predominating. One explanation for the difference between the sex incidence of the disease in Connecticut and Saskatchewan may be the fact that in the former area females, and especially those in the older age groups, are greater in number in the general population, while in the latter



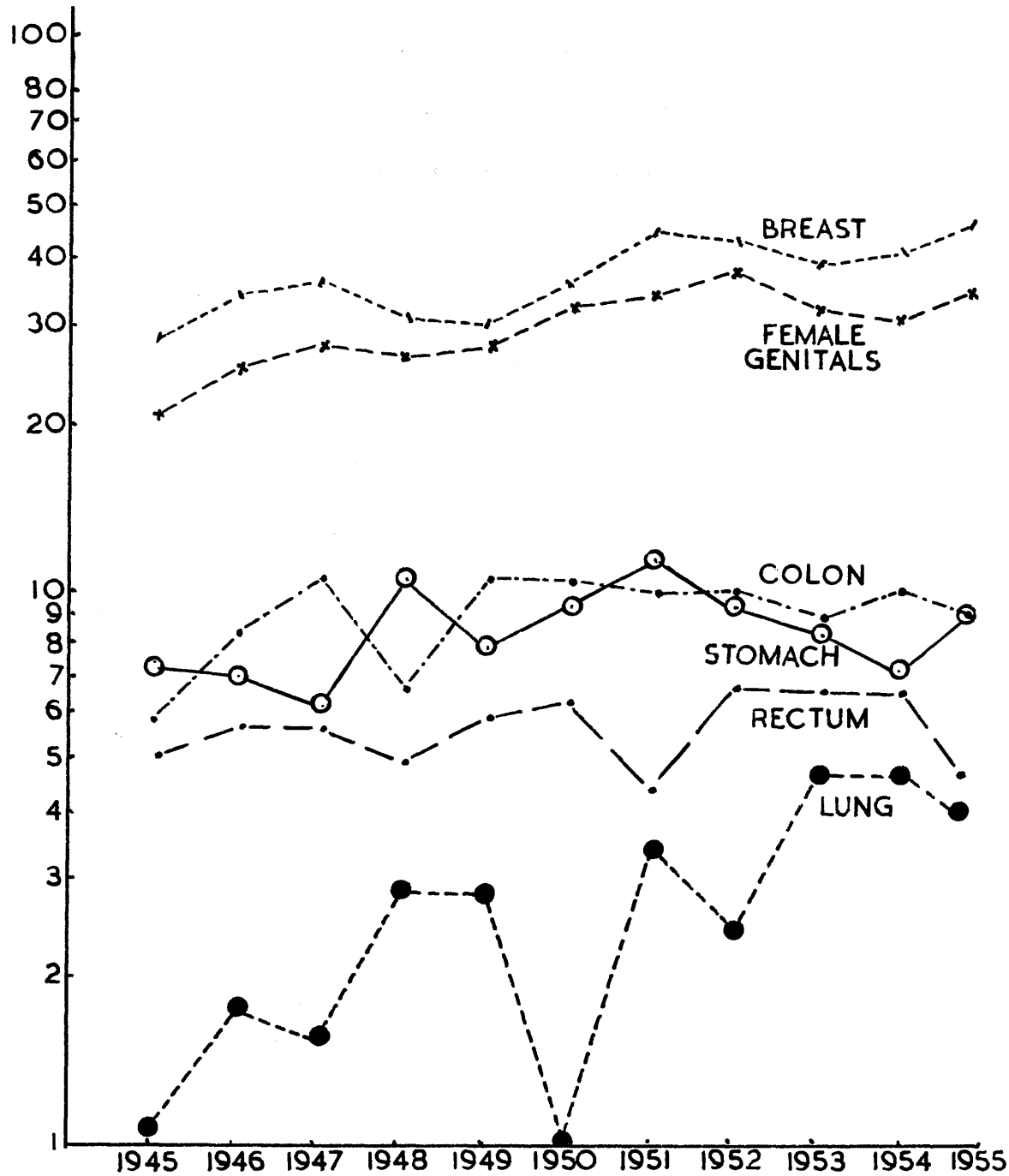
SEMI-LOGARITHMIC GRAPH DEMONSTRATING CRUDE RATE OF INCIDENCE OF CANCERS IN VARIOUS SITES PER 100,000 OF THE POPULATION. SASK. 1945-1955

FIGURE 4



SEMI-LOGARITHMIC GRAPH DEMONSTRATING CRUDE RATE OF INCIDENCE, IN MALES, OF CANCERS IN VARIOUS SITES, PER 100,000 OF MALE POPULATION. SASKATCHEWAN 1945-1955

FIGURE 5



SEMI-LOGARITHMIC GRAPH DEMONSTRATING CRUDE RATE OF INCIDENCE, IN FEMALES, OF CANCERS IN VARIOUS SITES, PER 100,000 OF FEMALE POPULATION. SASKATCHEWAN 1945-1955

FIGURE 6

region, males predominate in number in all age groups.

THE RELATIONSHIP BETWEEN AGE AND INCIDENCE OF GASTRIC CARCINOMA

Gastric cancer is primarily a disease of middle age and senescence and occurs with greatest frequency in those communities in which the life expectancy exceeds the sixth decade. In Saskatchewan, 87.6 per cent of the patients with gastric cancer were fifty years of age or more, 64.6 per cent were sixty years or more and thirty per cent were over seventy years of age. The incidence of the disease for the various age and sex groups is shown in Tables 5 and 6. It is apparent that the risk of developing gastric cancer increases with age. For males the chance of developing carcinoma of the stomach between the ages of fifty-five and sixty-four years is one in 1,430, between sixty-five and sixty-nine years, one in 833 and for those of seventy years of age and over it is one in 588. In females, the risk in each of these respective age groups is one in 3,330, one in 2,000 and one in 1,250 (Table 7). In a community in which there is such a preponderance of young people, the incidence rate for those of 35 years of age and over is of interest (Table 8).

The standardized rates of incidence of gastric carcinoma in Saskatchewan are presented in Table 9. A comparison between these and the age-adjusted rates published by Macdonald (1948) and Boles and Baum (1955) is possible. The average of the

	AGE GROUPING						
	Total	25-34	35-44	45-54	55-64	65-69	70 and over
Total Rate	15.7	1.7	6.8	16.8	52.6	90.8	133.7
Male Rate	21.9	1.6	8.4	21.5	67.9	116.4	171.5
Female Rate	8.9	1.8	5.0	11.3	31.4	52.6	82.2

GASTRIC CANCER: RATE OF INCIDENCE PER 100,000 OF POPULATION IN VARIOUS AGE GROUPS

SASKATCHEWAN 1945-55

TABLE 5

Age & sex	1945	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955
BOTH SEXES	11.6	13.2	15.5	15.8	16.8	16.1	17.3	17.2	16.9	16.6	16.2
MALE	16.4	18.7	22.0	22.4	23.3	21.9	23.6	23.8	24.2	23.8	22.9
25-34	1.5	1.5	1.0	1.0	1.6	2.1	1.1	1.6	1.6	1.6	1.6
35-44	9.1	8.3	7.6	6.2	8.0	8.5	12.0	8.9	8.6	5.7	4.3
45-54	14.5	15.7	18.3	20.7	23.9	22.6	26.2	25.0	26.3	23.8	22.6
55-64	55.9	61.5	68.1	71.3	73.3	67.7	66.4	69.7	74.8	61.1	66.3
65-69	96.8	114.1	135.4	143.2	128.1	103.4	102.7	107.5	119.9	117.1	115.7
70 and over	131.5	152.4	188.9	172.9	174.8	166.7	184.3	183.0	170.0	175.6	178.4
FEMALE	6.1	7.0	8.2	8.5	9.6	9.9	10.4	10.0	8.9	8.6	8.7
25-34	2.8	2.2	2.4	1.6	2.2	1.6	1.6	1.1	0.8	0.8	0.8
35-44	3.7	4.4	2.8	3.5	3.7	5.3	4.5	7.0	7.4	3.1	1.8
45-54	8.8	8.9	8.1	8.1	11.7	13.6	13.5	13.4	11.7	7.3	5.1
55-64	26.9	26.9	33.8	34.9	38.3	32.5	31.3	29.0	26.7	34.1	37.9
65-69	32.1	48.3	56.5	47.9	49.2	53.4	69.2	59.2	49.8	46.9	45.5
70 and over	49.6	62.8	79.0	86.3	91.1	93.6	95.5	88.8	74.9	82.9	86.9

GASTRIC CANCER: THREE YEAR MOVING AVERAGE OF INCIDENCE RATES PER 100,000 BY AGE AND SEX

SASKATCHEWAN 1945-55

TABLE 6

SEX	AGE GROUP (YEARS)	INCIDENCE
Males	55 - 64	1 in 1,430
	65 - 69	1 in 833
	over 70	1 in 588
Females	55 - 64	1 in 3,330
	65 - 69	1 in 2,000
	over 70	1 in 1,250

THE RISK OF DEVELOPING GASTRIC CANCER INCREASES WITH AGE

TABLE 7

YEAR	INCIDENCE RATE OVER 35 YEARS OF AGE	MALE INCIDENCE	FEMALE INCIDENCE
1945	32.3	41.4	20.1
1946	33.1	33.1	19.9
1947	39.6	57.1	17.5
1948	50.3	66.8	29.4
1949	35.1	45.3	22.6
1950	45.1	61.5	25.1
1951	43.6	53.7	31.5
1952	43.5	58.0	26.4
1953	41.6	56.6	23.8
1954	39.2	54.7	21.3
1955	42.5	57.3	25.2

GASTRIC CANCER: INCIDENCE PER 100,000 (CRUDE RATE) IN POPULATION OVER 35 YEARS

SASKATCHEWAN 1945-55

TABLE 8.

	YEAR										
	1945	1946	1947	1948	1949	1950	1951	1952	1953	1954	1955
TOTAL	10.9	11.0	12.6	15.9	11.4	14.7	13.4	13.3	14.2	13.1	14.1
MALE	14.6	15.0	19.0	21.8	15.5	20.7	17.4	18.8	20.6	18.6	20.4
FEMALE	6.8	6.3	5.3	9.1	6.7	7.8	8.7	7.1	6.9	7.3	7.0

GASTRIC CANCER: STANDARDIZED RATES OF INCIDENCE

SASKATCHEWAN 1945-55

TABLE 9.

age-adjusted incidence over the last five years of the Saskatchewan study is 13.6, the seven year average in the Macdonald survey is 15.6 and the average of the regional investigations conducted by the National Cancer Institute of the United States reported by Boles and Baum (1955) 34.7 and 26.4 respectively in the years 1937 and 1947. In Connecticut the age-adjusted incidence rates for females is considerably greater than that in Saskatchewan (10.7 in comparison with 7.4) but the rates for males 20.5 and 19.2 respectively, are reasonably comparable. The greater overall incidence of the disease in Connecticut as compared with that in Saskatchewan appears to be due mainly to this greater incidence among females and may reflect the greater proportion of females within the cancer age group in the general population in that State. The marked overall greater incidence in the ten separate surveys conducted by the American National Cancer Institute and the greater male and female incidence is not so readily explained. The disparity in the figures in the latter surveys may be related to the method of case reporting. Complete reliance was placed on the voluntary notification by physicians in, and the hospital records of, the ten metropolitan areas studied, and although a high proportion of the cases was proved histologically, many were accepted only on clinical grounds. Even taking into consideration the possible differences in incidence of gastric carcinoma in the various races represented in their cities, the range of incidence of

14.9 to 30.5 per 100,000 in the separate areas surveyed, appears to be too wide to be readily acceptable as true incidence rates. Boles and Baum (1955) are conscious of the difficulty of explaining these differences, but erroneously draw the analogy of the review by Stocks (1953) in which the comparative mortality figures are given for cancer of the stomach in aggregates of rural districts of North Wales and parts of England during the period between 1921 and 1949. Mortality and incidence rates are not comparable.

CHAPTER 2

DELAY IN DIAGNOSIS OF GASTRIC CANCER

The delay in diagnosis has proved to be a convenient hook on which to hang the responsibility for the poor results of surgical therapy for carcinoma of the stomach. Medical literature abounds with repeated pleas for earlier diagnosis, but despite all efforts to impress upon the public the importance of seeking medical advice when the first symptom arises, there is little evidence that patients are consulting their medical advisers at an earlier stage. Harvey et al (1951) following an analysis of 34 years' experience of cancer of the stomach at the Presbyterian Hospital, New York, were disturbed by the fact that the duration of symptoms, before medical consultation, was as long in the later years of the study as in the earlier years. Welch and Allen (1948) commented on the same lack of improvement in two consecutive ten year studies from the Massachusetts General Hospital.

The responsibility for delay in diagnosis does not lie only with the patient. Harvey et al (1951) commented on the culpability of the medical profession. They found that in half of all their cases the physician was responsible for a longer period of delay than was the patient. Boyce (1953) reported a similar experience in three consecutive series of cases.

In Saskatchewan, in the interval between 1932 and 1951 there was little evidence that the total duration of time between the onset of the first symptom and the time of treatment was materially lessened. An average delay of between nine and ten months was the rule, the median duration being six months. Between 1952 and 1955 the average period of delay was just less than eight months, the median being five months (Table 10). The greater culpability for this delay was that of the patient in all the periods. The doctors of the province were responsible for a median delay of little more than one month.

Incrimination of the patient and the doctor in respect of delay in diagnosis does not cover the whole field of responsibility. Some account of the role played by the disease itself has to be considered. Carcinoma of the stomach does not produce any characteristic syndrome in its early stages of development. Frequently the symptoms may be referable to other parts of the gastro-intestinal tract and not to the stomach itself. (Weiner 1933, Jordan and Hill 1932, Wilbur 1935). Although the significance of a change in symptomatology in a patient who

PERIOD	DELAY IN DIAGNOSIS AND TREATMENT					
	Duration in months from onset of the first symptom to treatment		Duration in months from onset of the first symptom to the first consultation with a doctor		Duration in months from the first medical consultation to treatment	
	Average	Median	Average	Median	Average	Median
1932-36	8.9	6.0	6.0	5.0	2.8	1.0
1937-41	9.0	6.0	5.1	3.0	5.3	1.0
1942-46	10.2	6.0	6.3	3.0	4.1	2.0
1947-51	10.0	6.0	5.4	2.0	4.0	2.0
1952-55	7.6	5.0	4.4	3.0	3.2	1.0

GASTRIC CANCER: THE PERIODS OF DELAY BETWEEN THE ONSET OF THE FIRST SYMPTOM AND TREATMENT IN VARIOUS YEAR GROUPS

SASKATCHEWAN 1932-55

TABLE 10

has a long history of duodenal ulcer is recognised by most clinicians, (Jordan 1938), the fact that a carcinoma of the stomach may develop silently in such a patient, without any alteration of symptoms, is perhaps less widely appreciated. No longer can we assume that a patient with duodenal ulcer has comparative immunity to gastric cancer. From time to time patients who have had complete freedom from gastro-intestinal complaints are found at autopsy to have large gastric cancers which have already metastasised. In these cases, even the recommendation given by Bayle in 1833 and re-emphasised by Collins (1938) to make the diagnosis when the patient is asymptomatic, might not result in an early enough diagnosis to permit cure. Wangensteen (1951) has estimated that a period of twenty months may elapse between the beginning of the disease and the onset of its first symptom.

Ketch (1937) described three phases of gastric cancer. The first of these is the period of absolute latency, the second the period of relative latency and the third the period of apparent disease. Over the past two decades there has been a considerable effort to diagnose the condition during the period of absolute latency. Several workers have attempted to demonstrate asymptomatic gastric cancer by screening large numbers of the public in a manner similar to that of mass photofluorography of the chest. Dailey (1947) found no individual with gastric neoplasm in a radiological survey of 500 asymptomatic men

over the age of forty-five years. In the studies by St. John et al (1944) there was an incidence of only 1.24 gastric cancers per thousand patients examined. Roach et al (1952) conducted a similar survey of patients attending the out-patient department of the Johns Hopkins Hospital. Of 9,072 cases only one gastric cancer was found for every 476 examinations made. Only one of the patients found to have gastric cancer was asymptomatic. These studies, while laudable in their intent, proved that mass screening of the stomach in a search for neoplasm was not practicable because of the cost and the extremely small yield.

Cancer detection centres have been set up in various parts of the United States of America to investigate, thoroughly, large numbers of asymptomatic persons at six monthly intervals in a search for early cancer. In the University of Minnesota, where a cancer detection centre has been established since 1948, a total of 7,074 supposedly well people have been examined and tested for gastric acidity. Most of these people have been re-examined at yearly intervals with aspiration of gastric juice for testing at each examination. By this method nineteen patients were found to have gastric cancer. At the time of discovery of their disease twelve were still asymptomatic, three had developed mild gastric symptoms and four had marked symptoms. One patient had inoperable gastric cancer. Of the twelve asymptomatic patients eight had tumour confined to the stomach, with no

metastases. Two of the three patients with mild symptoms had no extra gastric spread. All of the frankly symptomatic patients had tumour involved regional lymphatic glands.

In the light of this evidence, it is possible that a more rational approach to the problem of the detection of gastric cancer in the early and asymptomatic phase would be the repeated investigation of patients who are known to have conditions considered to be precancerous.

CHAPTER 3

PRECURSORS OF GASTRIC CANCER

The incidence of gastric carcinoma in individuals with pernicious anaemia and achlorhydria is higher than in persons of similar age and sex distribution who exhibit no evidence of these two pathological states. The evidence supporting the claim that gastritis and gastric polypi are also gastric cancer precursor conditions is less definite.

PERNICIOUS ANAEMIA

Although the clinical features of pernicious anaemia were first described by Thomas Addison in 1885, Combe, three years earlier published a report on several patients who undoubtedly had pernicious anaemia and in whom, at autopsy, he observed changes in the digestive tract which he believed to be significant. In 1860, Austin Flint also remarked on the signs of degenerative disease of the gastric mucosa and on the

achlorhydria which accompanied, or was part of, the pernicious anaemia syndrome. Jones (1855) described a case of "extreme wasting of the secretory structure of the stomach coinciding with like wasting of the blood, without any apparent cause."

The relationship between pernicious anaemia and gastric mucosal changes was established by Samuel Fenwick (1870, 1877). He described the pale smooth mucosa, the atrophy of the glands and the infiltration of the involved area with lymphocytes. Similar changes were described by Quinke (1877) and Nothnagel (1879). Further studies of these changes were made by Magnus (1938) and Cox (1943). Hyperplasia was observed by Olsen (1945) to be present in eighty-five per cent of the mucous secreting cells described by Cox. The term hyperplasia included such changes as irregularity of the mucous cells, irregularity of size and position of the nuclei, hyperchromatism of the nuclei and the presence of occasional mitotic figures. These changes were considered by these authors to be related to the development of cancer.

Quinke was the first to demonstrate an apparent relationship between pernicious anaemia and gastric carcinoma, and described the first recorded case in which the two conditions co-existed. Since then numerous reports have appeared in the medical literature in which individual cases exhibiting both diseases simultaneously have been described. These reports have been reviewed by Cotte (1938), Kaplan (1945), Washburn (1938) and Jenner (1939). These authors are convinced that an association between the two diseases has been established.

Jenner (1939) was perhaps the first to demonstrate that the association between the two conditions was not accidental. In a series of 181 patients with well established pernicious anaemia he found eight patients (4.4 per cent) who also had carcinoma of the stomach. He calculated that this incidence was twelve times as frequent as in the rest of the living population of the same age and sex.

Doehring (1942) found the co-existence of carcinoma of the stomach in 1,014 cases of pernicious anaemia to occur in 1.6 per cent, thus representing an incidence five times greater than that occurring in the general population over the age of forty-five years (Collins et al 1941). Various reports from the Mayo Clinic by Griffin (1923) Connor (1933) Washburn (1938) and Doehring (1942) reveal a steady increase in the incidence of cancer of the stomach occurring in patients also suffering from the Addisonian type of anaemia. The great increase in the life expectancy of patients with pernicious anaemia since the introduction of liver therapy in 1926 is generally offered as the explanation for this apparent increase in gastric cancer morbidity among them. Before the introduction of liver therapy, most patients afflicted were dead within three years. This short survival did not permit the development of many complications, gastric cancer possibly being one of them.

Rigler et al (1945) published a report of their radiological investigation of 211 patients who had well established

pernicious anaemia. Most of these patients had twice yearly x-ray studies of their stomachs and during the period of investigation seventeen were found to have developed cancer of the stomach. This is an incidence which, calculated by the method of Collins (1941) is approximately twenty-seven times greater than that occurring in a comparable group of individuals without pernicious anaemia. It is of interest to contrast this result with that of the mass radiological study of St. John et al (1944) in which the incidence of gastric cancer in asymptomatic individuals was 1.24 per one thousand persons examined. Hitchcock (1955) reports that the incidence of gastric cancer in those patients with pernicious anaemia followed at the University of Minnesota Cancer Detection Centre was 18.3 times greater than the expected national incidence.

Cotte (1938) compiled all the published data concerning the association between pernicious anaemia and cancer in all sites. He found that the stomach was the primary site in ninety-three of one hundred and seven cases, or eighty-six per cent of the entire series. Because he estimated that cancer of the stomach represented only twenty to forty per cent of all cancers in the general population, the marked predilection for this organ in patients with pernicious anaemia was considered to represent a link in the chain of corroborative evidence of the association between the two diseases.

Kaplin et al (1945) studied all the cases of carcinoma

of the stomach, colon and rectum which they found in a series of 43,021 consecutive autopsies performed in the University of Minnesota Hospitals. Of 293 patients who had pernicious anaemia, they found thirty-eight had co-existing carcinoma of the stomach, seven had gastric polypi and only four had cancer of the colon or rectum. They stated that statistical analysis revealed that only twelve cases of gastric cancer would have been expected and that the probability that the observed number was due to chance alone was less than one in a billion.

The careful studies by Mosbech (1953, 1954) and Videbaek and Mosbech (1954) indicate that pernicious anaemia may be an inherited defect which is localized in the gastric mucosa. From their data it would appear reasonable to conclude that gastric cancers complicating pernicious anaemia may also be genotypic, depending on a common inherited predisposition to the two diseases. The gastric mucosal atrophy which accompanies pernicious anaemia is irreversible and is unaffected by liver or vitamin B_{12} therapy [Dorg (1950), Fink (1953), Magnus (1938), Meulengracht (1939), Cox (1943), Feyrter (1952), Palmer (1953)] and constitutes a long term threat to the patient's life by providing a fertile bed in which precancerous polypi and overt malignancies are prone to develop. (Rubin 1955)

ACHLORHYDRIA

From 1879, when Van der Velden first recognized the association between achlorhydria and gastric cancer, until recently,

it was assumed that the anacidity arose as a direct result of the tumour. Comfort (1937, 1947, and 1948) however, in reviewing a group of patients upon whom at least one gastric analysis had been done two or more years before a diagnosis of cancer of the stomach had been made, demonstrated that the reverse of this sequence was nearer the truth and that the achlorhydria was probably a forerunner of the gastric neoplasm.

This and other similar evidence led Hitchcock (1955) and his associates at the University of Minnesota Hospitals to test the value of achlorhydria and hypochlorhydria as a screening procedure for persons likely to develop gastric cancer. By observing a large number of achlorhydric persons of fifty years of age and more, over a number of years, they found that the incidence of cancer of the stomach in that selected group was 3.2 times greater than the expected incidence. In the hypochlorhydric patients the incidence was 2.6 times greater than that expected. Of nineteen gastric cancers found, twelve were asymptomatic. In eight of these (sixty-six per cent) the gastric neoplasm was confined to the stomach, whereas in the seven patients, who had associated symptoms, the tumour had extended to the regional lymphatic glands in five.

These studies would indicate that a reduced level of free hydrochloric acid in the stomach may constitute a continuing threat to the well-being of an individual and increase his chance of developing gastric cancer. Moreover, they would suggest

that by frequent observation of patients of this type, it might be possible to detect early neoplastic changes within the stomach and by instituting adequate treatment before the patient became symptomatic increase the salvage rate from the disease.

ATROPHIC GASTRITIS AND GASTRIC POLYPI

The theory of the role played by chronic atrophic gastritis in the aetiology of cancer of the stomach has been based upon the high coincidence of the two conditions in surgically resected stomachs and in autopsy material. The initial evidence presented by Orator (1925) and Hurst (1929) was supported by Konjetzny (1942) whose photographic demonstration of atrophic gastritis with pronounced polypoid changes merging into carcinoma made it difficult to disregard completely the possibility of the association of the two conditions. On the other hand, studies by Guiss et al (1943), Wanser (1939) and Hebbel (1943), did not confirm the relationship and Stout (1943 and 1945), while he did not deny that a possible association did exist, could not demonstrate any actual progression from altered mucosal glands to frank carcinoma.

Chronic atrophic gastritis is found as a concomitant not only of gastric cancer (Schindler 1923 and 1941), but also of gastric polypi (Hay 1953) and in patients with pernicious anaemia. The incidence of gastric polypi in patients with pernicious anaemia [Saltzman (1931), Rhoads (1941) and Brown (1934)] and in those with gastric cancer (Hay 1956) is high.

With the knowledge that a common factor in all of these four conditions is an abnormally low level of free gastric hydrochloric acid and that the incidence of gastric cancer is high in the presence of achlorhydria and hypochlorhydria, it would appear that more than a coincidental relationship between all of the conditions exists and that possibly atrophic gastritis and gastric polypi, whether they arise as part of the pernicious anaemia syndrome or not, play a role in the developmental pattern of gastric cancer.

THE RELATIONSHIP BETWEEN ABO BLOOD GROUPS AND GASTRIC CANCER

The suggestion that there might be a relationship between the ABO blood groups and certain diseases has recurred in the medical literature since 1921 [Buchanan (1921), Alexander (1921), Johannsen (1925), Goldfeder (1937) and Ivey (1946)] , but, until Struthers (1951) reported on an excess of Group A among children dying with bronchopneumonia, no significant association was demonstrated. Aird and his associates, in 1953, were the first to show statistical significance in the association between gastric cancer and blood group A. Confirmation of this relationship has been reported by Clarke (1955), Buckwalter (1956) and Canonico (1955). In 1956 Aird and Creger presented evidence to support a relationship between blood group A and pernicious anaemia. This observation

combined with that on the blood group association with gastric cancer adds further to the evidence supporting an association between pernicious anaemia and carcinoma of the stomach. It is possible that both conditions are related directly to the occurrence of group A and only indirectly to each other. Peebles Brown (1956) and Aird (1954) demonstrated that there was a higher incidence of group O in patients with peptic ulcer than in the general population.

Whereas Aird, Peebles Brown and others have assumed that it was the pathological type of the upper gastro-intestinal disease which was related to the ABO blood group, Jennings (1956), Balme (1957) and Billington (1956 (a) and 1956 (b)) presented evidence to support the theory that the association was between the site of the lesion within the stomach and the blood group. They believed that the pathological type of the lesion, whether benign or malignant, was irrelevant. They found that in carcinoma of the body of the stomach group O was predominant and that group A was more commonly associated with cancer of the antrum and prepyloric areas. Benign gastric ulcer behaved in a similar way, with simple ulcers in the antrum and prepylorus tending to be associated with group A and simple ulcers proximal to the angulus with group O. The overall apparent relationship between gastric cancer and group A, and between benign ulcer and group O resulted from the fact that most cancers

have been found to occur in the distal part of the stomach (in lesions of which group A predominates) and most benign ulcers in the body of the stomach in which group O is predominant.

The investigation into the relationship between blood groups and gastric disease is still in its early phase, but it gives promise of yielding a new basis to the understanding of the etiology of gastric cancer and peptic ulcer. Whatever the co-relationship may be, it is unlikely to be a remote one. Although the term "blood group" designates a certain property of blood, it is not a property which is peculiar to blood alone. It has been shown that the fixed tissue cells of the entire body exhibit the same group specific characteristics as the blood cells [Witebsky (1927), Witebsky and Okabe (1927), Kritschewsky and Schwarzmann (1927)] but that the concentration of the group specific substances differs considerably in various organs. The brain contains very little. The pancreas and gastric and intestinal mucosa are rich in them and indeed may have a concentration greater than the blood cells. Saliva and gastric juice are characterised by a high content of the group specific substances in about eighty per cent of all human beings, but approximately twenty per cent of individuals fail to secrete them [Putkonen (1930), Lehrs (1930) and Friedenreich (1938)]. Secretion and non secretion are constant and inherited properties and the gene of secretion is dominant over that of non-secretion (Schiff 1932).

The blood group substances are muco-polysaccharides. It seems unlikely that they are either carcinogenic or ulcerogenic compounds. It would be much more in character for muco-polysaccharides to be concerned with protection against disease rather than with its causation, and that, while all the blood group substances would protect against gastric cancer and peptic ulcer, groups A and B protect more effectively against ulcer and groups O and B against cancer of the stomach.

The interesting question arises whether there is any relationship between the ability of the stomach and intestine to secrete blood group specific substances and the development of pathological conditions of these organs. Research into this facet of the problem would be of value.

Using the clinical material available in the Regina clinic of the Cancer Commission and the records of the Grey Nuns' Hospital, a survey of ABO blood groups and their relationship to cancer in various sites within the body was made. The distribution of blood groups within the province was obtained from the records of the Red Cross Blood Transfusion service. Statistical analysis demonstrates significance in the relationship between gastric cancer and group A, and there is borderline significance between group O and peptic ulceration. No significance was discovered between ABO blood groups and the other specified conditions.

ABO Blood Grouping in General Population							
O		A		B		AB	
Number	Per cent	Number	Per cent	Number	Per cent	Number	Percent
10,070	44.53	9,280	41.04	2,332	10.31	933	4.12

THE DISTRIBUTION OF ABO BLOOD GROUPS IN THE GENERAL POPULATION OF THE SOUTHERN PART OF SASKATCHEWAN

TABLE 11

Site	ABO Blood Groups in Patients with Cancer in Various Sites							
	O		A		B		AB	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Stomach (577 patients)	234	40.6	277	48.0	47	8.1	19	3.3
Colon (281 patients)	124	44.1	114	40.6	26	9.3	17	6.0
Rectum (152 patients)	54	35.5	74	48.7	16	10.5	8	5.3
Breast (294 patients)	120	40.8	131	44.6	31	10.5	12	4.1
Female Genitals (146 patients)	59	40.5	57	39.1	23	15.7	7	4.7
Male Genito-urinary (288 patients)	113	39.2	124	43.1	39	13.6	12	4.1
Other miscellaneous cancers (413 patients)	157	38.1	171	41.4	59	14.3	26	6.2

THE ABO BLOOD GROUP DISTRIBUTION IN CANCERS OF VARIOUS SITES

TABLE 12

	ABO Blood Groups in Patients Without Cancer							
	O		A		B		AB	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Duodenal ulcer (566 patients)	293	51.7	207	36.6	40	7.1	26	4.6
Gastric ulcer (145 patients)	70	48.3	55	37.9	14	9.6	6	4.2
Miscellaneous non malignant conditions (3,477 patients)	1,452	41.8	1,457	41.9	400	11.5	168	4.8

THE ABO BLOOD GROUP DISTRIBUTION IN PEPTIC ULCER AND IN A CONSECUTIVE SERIES OF HOSPITAL PATIENTS WITHOUT CANCER

TABLE 13

CHAPTER 4

DIAGNOSIS OF GASTRIC CANCER

The great need for the recognition of gastric cancer at a stage when effective surgical treatment can be given is obvious. The difficulties of its detection at an early stage in development, and the advantages of anticipating the onset of the condition, in groups of patients who appear to have a predisposition to it have been discussed.

There are many symptoms which may be associated with gastric cancer, but none of them is peculiar to that disease alone, and none by itself allows confident differentiation from benign conditions of the digestive tract or from diseases of certain other systems. Reviews of large groups of patients with gastric cancer have been conducted by various investigators and all of them have found the symptom pattern of the disease to be similar, [Jemerin (1952), Harris (1936), Marshall (1937), Levitt (1938), Harnett (1947) and Walters (1943)]

The patients in the Saskatchewan series do not differ materially in this respect. Pain was the most frequent first symptom (Table 14), arising as a first warning of abnormality in 47.5 per cent, an incidence similar to the 47.9 per cent reported by Harnett (1947). Anorexia and the sensation of postprandial fullness each occurred as the first symptom in approximately one tenth of the patients.

It is of interest to assess the impact, or alarm index, of the various first symptoms upon the patients in terms of causing them to seek medical advice (Table 15). Almost three fifths of all the patients in whom pain was the presenting symptom sought medical advice within three months of the onset and more than three-quarters of them within the first six months. Less than thirteen per cent tolerated pain for more than one year without consulting their medical advisor. The other leading first symptoms of anorexia, postprandial fullness, weakness and vomiting gave rise substantially to no greater or lesser degree of concern on the part of the patient. Haematemesis and gross melaena were regarded by the patient as being sufficiently dramatic to warrant immediate attention. These latter symptoms occurred as the initial warning in only one per cent of the patients.

Few patients presented with only one symptom. An analysis of the incidence of the most frequently occurring groups of symptoms is given in Table 16. Anorexia, weakness and loss of weight was the most commonly occurring triad of symptoms.

FIRST SYMPTOM	FREQUENCY (PER CENT)
No Symptoms	0.2
Pain	47.5
Anorexia	11.6
Postprandial fullness	9.7
Weakness	7.5
Vomiting	6.6
Dysphagia	3.0
Loss of Weight	2.6
Constipation	2.0
Nausea	1.6
Diarrhoea	0.9
Haematemesis	0.8
Palpable Mass	0.6
Regurgitation	0.4
Melaena	0.2
Other	0.6
Unstated	4.2

GASTRIC CANCER: THE FIRST SYMPTOM IN GASTRIC CANCER

SASKATCHEWAN 1932-55

TABLE 14

FIRST SYMPTOM	TOTAL PER CENT	DISTRIBUTION OF PATIENTS (PER CENT)				
		DURATION OF SYMPTOMS FROM ONSET TO TREATMENT (MONTHS)				
		Less than 1 month	1 - 3	4 - 6	7 - 12	13 and over
Pain	100.0	27.4	30.8	17.7	11.5	12.6
Anorexia	100.0	22.1	39.2	19.1	11.1	8.5
Postprandial fullness	100.0	23.9	35.3	16.2	12.6	12.0
Weakness	100.0	25.0	27.3	18.0	15.6	14.1
Vomiting	100.0	30.1	36.3	15.0	13.3	5.3
Haematemesis	100.0	92.9	-	-	-	7.1
Palpable mass	100.0	20.0	30.0	10.0	40.0	-
Melaena	100.0	100.0	-	-	-	-
All others	100.0	45.3	25.1	13.5	13.1	3.0

GASTRIC CANCER: FIRST SYMPTOM RELATED TO THE INTERVAL BETWEEN THE ONSET OF THE SYMPTOM AND FIRST REPORTING FOR TREATMENT

SASKATCHEWAN 1932-55

TABLE 15

SYMPTOM		FREQUENCY (PER CENT)
Anorexia Loss of weight	Weakness Pain	15.7
Anorexia Loss of weight	Weakness Constipation	14.1
Anorexia Loss of weight	Pain Palpable mass	8.9
Anorexia Loss of weight	Weakness Vomiting	6.0
Anorexia Loss of weight	Weakness Postprandial fullness	5.7
Vomiting Postprandial fullness	Nausea Pain	3.9
Anorexia Loss of weight	Vomiting Postprandial fullness	3.8
Anorexia Loss of weight	Vomiting Nausea	3.7
Weakness Loss of weight	Pain Palpable mass	3.7
Vomiting Postprandial fullness	Nausea Constipation	3.2
All other combinations		31.3

GASTRIC CANCER: FREQUENCY OF FOUR-FOLD SYMPTOM GROUPING

SASKATCHEWAN 1932-55

TABLE 16

It is possible that anorexia, with the resultant reduced nutrition of the patient gave rise to the other members of the triad, weakness and loss of weight. Associated anaemia may also have contributed towards the weakness of the patient. More than ninety per cent of all the patients in the series had lost weight. In thirty per cent the loss was no greater than one-tenth, and in just over two fifths the loss was between ten and twenty per cent of the normal body weight, (Table 17).

The incidence or degree of the weight loss had little relationship to the site of the primary tumour within the stomach (Table 18), or to the presence or absence of extra gastric spread of the disease (Table 19). A similar lack of relationship existed between the degree of weight loss and the level of free hydrochloric acid in the stomach at the time of the patient's first examination (Table 20).

Data on the haemoglobin levels in 92.2 per cent of the patients were available. In more than fifty per cent of the known cases the anaemia was of a significant order (Table 21).

The haemoglobin level appeared to be uninfluenced by the site of the lesion (Table 22). The type of the lesion however, did have an influence (Table 23). When ulcerative, polypoid and infiltrative lesions were compared, it became evident that ulcerative and polypoid cancers were more commonly associated with gross anaemia than infiltrative lesions. A minority of the patients with infiltrative disease had significant anaemia. The

WEIGHT LOSS (PER CENT OF NORMAL BODY WEIGHT)	DISTRIBUTION OF PATIENTS BY WEIGHT LOSS (PER CENT)
None	7.3
1 - 10	30.3
11 - 20	41.6
21 - 30	17.3
30 and greater	3.5

GASTRIC CANCER: THE INCIDENCE AND DEGREE OF WEIGHT LOSS. THE DATA WERE AVAILABLE IN SEVENTY PER CENT OF PATIENTS

1400 CASES

SASKATCHEWAN 1932-55

TABLE 17

Site	Weight loss (Per cent of normal body weight)				
	No weight loss	1 - 10	11 - 20	21 - 30	30 and greater
All sites	7.3	30.3	41.6	17.3	3.5
Prepylorus	8.3	30.4	40.8	16.9	3.6
Antrum	5.2	26.5	47.1	18.1	3.1
Lesser curvature	8.0	34.5	38.7	16.5	2.3
Greater curvature and fundus	13.6	28.4	44.3	11.3	2.4
Body	3.9	35.1	44.2	10.4	6.4
Cardia	3.6	27.9	37.8	27.1	3.6
Whole stomach	3.0	19.4	43.3	29.8	4.5

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE GASTRIC NEOPLASM AND THE WEIGHT LOSS, CALCULATED AS A PERCENTAGE OF THE PATIENTS' NORMAL WEIGHTS. THE DATA WERE AVAILABLE IN 1159 (58.8 PER CENT OF TOTAL NUMBER OF CASES)

SASKATCHEWAN 1932-55

TABLE 18

	Weight loss (Per cent of Normal Body Weight)				
	No weight loss	1 - 10	11 - 20	21 - 30	30 and greater
All patients	7.3	30.3	41.6	17.3	3.5
No metastases	9.7	36.9	41.7	8.3	3.4
Positive nodes only	8.0	35.2	41.8	12.4	2.6
Liver metastases only	7.7	25.8	43.9	20.6	2.0
Positive glands and other metastases	6.9	29.8	35.8	23.0	4.5
Multiple metastases (unspecified)	6.0	34.4	27.7	27.6	4.3
Other	7.5	29.8	40.3	19.4	3.0

GASTRIC CANCER: THE RELATIONSHIP BETWEEN METASTASES AND WEIGHT LOSS, THE LATTER CALCULATED AS PER CENT OF THE PATIENTS' NORMAL WEIGHTS. THE DATA WERE AVAILABLE IN 1061 PATIENTS (53.0 PER CENT OF TOTAL GROUP)

SASKATCHEWAN 1932-55

TABLE 19

Weight loss calculated as percent of normal body weight	All cases	Free hydrochloric acid levels in stomach				
		Not stated per cent	Achlor-hydria with histamine	hypochlor-hydria with histamine	Normal acidity with histamine per cent	Hyper-acidity with histamine per cent
Total	100.0	100.0	100.0	100.0	100.0	100.0
None or voluntary loss	5.0	4.3	5.4	5.5	7.5	3.3
0 - 5	7.0	3.3	9.3	6.2	13.4	10.0
5 - 10	14.0	9.6	16.8	18.5	14.9	13.3
10 - 15	16.0	12.7	18.1	15.1	22.4	20.0
15 - 20	13.0	10.0	14.8	16.4	14.9	6.7
20 - 25	8.1	7.3	9.6	4.8	4.5	6.7
25 - 30	4.6	4.5	4.6	4.8	4.5	-
30 - 35	1.4	1.5	1.2	2.7	-	-
More than 30	0.9	0.7	0.9	0.7	3.0	3.3
Unstated	30.0	46.1	19.1	25.3	14.9	36.7

GASTRIC CANCER: RELATIONSHIP BETWEEN GASTRIC FREE HYDROCHLORIC ACID AND WEIGHT LOSS. THE DATA AVAILABLE IN 923 PATIENTS (46.1 PER CENT OF TOTAL NUMBER OF CASES)

SASKATCHEWAN 1932-55

TABLE 20

	HAEMOGLOBIN LEVELS PERCENT OF NORMAL TAKEN AS 15.6 GM.							
	Less than 30	30-39	40-49	50-59	60-69	70-79	80-89	90-100
Number	24	63	108	170	274	346	312	547
Per Cent	1.3	3.4	5.4	9.3	14.9	18.3	17.6	29.8

GASTRIC CANCER: DISTRIBUTION OF HAEMOGLOBIN LEVELS AT THE TIME OF ADMISSION TO CLINIC OBSERVATION IN 1844 CASES

SASKATCHEWAN 1932-55

TABLE 21

SITE OF LESION	HAEMOGLOBIN LEVELS PERCENT OF NORMAL TAKEN AS 15.6 gm.							
	Less Than 30	30-39	40-49	50-59	60-69	70-79	80-89	90-100
All Cases	1.3	3.4	5.4	9.3	14.9	18.3	17.6	29.8
Prepylorus	1.3	4.5	7.8	10.9	12.2	17.7	16.8	28.8
Antrum	1.6	5.8	6.3	9.5	17.4	16.8	11.0	31.6
Lesser Curvature	1.6	4.0	4.2	8.2	16.0	19.2	16.0	30.8
Greater Curvature & Fundus	0.7	-	5.2	4.6	19.3	21.3	20.6	28.3
Body	-	0.7	5.7	14.9	15.6	17.0	17.0	29.1
Cardia	1.1	2.6	4.8	9.6	12.2	21.2	20.5	28.0
Whole Stomach	1.9	1.9	6.7	6.7	13.4	15.2	19.0	35.2

GASTRIC CANCER: DISTRIBUTION OF HAEMOGLOBIN LEVELS ACCORDING TO THE SITE OF THE PRIMARY TUMOUR. DATA AVAILABLE IN 1723 PATIENTS (86.2 PERCENT OF TOTAL NUMBER OF CASES).

SASKATCHEWAN 1932-55

TABLE 22

TYPE OF TUMOUR	HAEMOGLOBIN LEVELS PERCENT OF NORMAL TAKEN AS 15.6 gms.							
	Less Than 30	30-39	40-49	50-59	60-69	70-79	80-89	90-100
Polypoid	0.7	1.4	4.9	13.9	20.3	17.5	14.7	26.6
Ulcerating	1.3	3.4	5.6	10.2	14.5	17.1	13.8	34.1
Infiltrating	-	0.9	4.5	4.5	11.5	16.1	22.2	40.3
Linitis	2.9	8.8	2.9	2.9	8.8	11.8	11.8	50.1
Cancer or Benign Ulcer	-	6.7	-	6.7	13.3	-	40.0	33.3

GASTRIC CANCER: HAEMOGLOBIN LEVELS RELATED TO THE TYPE OF LESION. DATA WERE AVAILABLE IN 972 CASES (48.6 PER CENT OF THE TOTAL NUMBER OF CASES)

SASKATCHEWAN 1932-55

TABLE 23

number of patients with linitis plastica for whom data on haemoglobin levels were available, was too small for comparative study. No correlation between the degree of anaemia and the presence or absence of occult blood in the stool could be made because an insufficient number of the patients had their faeces tested for blood.

Table 24 demonstrates the relationship between haemoglobin levels and the presence or absence of metastases. More than two-thirds of the patients who had no metastatic disease and more than half of those in whom the metastases were to regional lymphatic glands only, did not have significant anaemia. Patients with liver secondaries had the greatest degree of anaemia.

While a large number of patients complained of indigestion and heartburn, there appeared to be no constancy of definition of these terms. To some the former meant slight to moderate discomfort after meals, often associated with the sensation of postprandial fullness and accompanied by belching. To others indigestion meant actual pain of moderate or greater severity, frequently of gnawing type and relieved by vomiting. Heartburn to most of them meant retrosternal discomfort with belching, regurgitation and sometimes nausea.

Pain was the commonest individual symptom. Its site was usually epigastric, but sometimes it occurred in the right or left upper abdominal quadrants, or peri-umbilical region.

	HAEMOGLOBIN LEVELS PER CENT OF NORMAL TAKEN AS 15.6 gm.							
	Less Than 30	30-39	40-49	50-59	60-69	70-79	80-89	90-100
No Metastases	0.3	2.6	2.3	8.2	15.5	17.8	17.9	35.4
Involved Regional Glands Only	1.1	4.0	5.8	9.1	12.5	14.6	19.0	34.0
No Glandular Involvement But Other Metastases Present	-	1.1	1.1	7.0	17.3	18.4	12.6	42.5
Involved Glands Plus Other Metastases	1.0	2.6	5.6	8.6	11.6	24.6	17.8	28.2
Lower Metastases Only	0.9	2.3	7.5	7.9	20.6	21.1	16.8	21.9

GASTRIC CANCER: HAEMOGLOBIN LEVELS RELATED TO METASTASES. DATA AVAILABLE ON 1577 PATIENTS (78.9 PER CENT OF THE TOTAL NUMBER)

SASKATCHEWAN 1932-55

TABLE 24

In others the only pain complained of was more remote as in the lumbo-dorsal region or anterior chest. It varied in intensity from patient to patient and also in the time of its occurrence in relation to food. In only 5.1 per cent of the 1,489 patients in whom the relative information was available, was there no relationship between the onset of pain and the taking of food (Table 25). In ten per cent the pain occurred before and in 36.5 per cent after taking food. In almost half of the patients there was no constant relationship. The data on the relief of pain were inadequate and no conclusions could be drawn from them (Table 26).

Constipation was a very common complaint. It occurred second in frequency to pain as a concomitant symptom with the triad of anorexia, weakness and loss of weight. Many patients described their stool as "chunky" or "jack-rabbit" in form. The significance of this observation is not apparent.

In almost two thirds of the patients, the level of gastric free hydrochloric acid was determined at the time of their first examination. In three-quarters of these patients no free hydrochloric acid was found even with histamine stimulation. In approximately ten per cent the level was either within normal range or above it (Table 27).

The data presented in Table 28 would suggest that there may be a relationship between the gross type of cancer and the level of free hydrochloric acid in the stomach. Those with

PAIN - RELATIONSHIP BETWEEN ITS ONSET AND THE TAKING OF FOOD (PER CENT)			
BEFORE	AFTER	INCONSTANT	NONE
10.1	36.5	48.3	5.1

GASTRIC CANCER: RELATIONSHIP BETWEEN THE ONSET OF PAIN AND TAKING FOOD. OBSERVATION FROM 1489 PATIENTS

SASKATCHEWAN 1932 - 55

TABLE 25

RELIEF OF PAIN					
By Food (Per Cent)	By Alkali (Per Cent)	By Vomiting (Per Cent)	By Food and Alkali (Per Cent)	By Food and Vomit (Per Cent)	Not Relieved (Per Cent)
26.9	30.0	10.6	9.9	1.5	21.1

GASTRIC CANCER: DATA ON RELIEF OF PAIN IN 331 PATIENTS ON WHOM THE RELATIVE DATA WERE AVAILABLE

SASKATCHEWAN 1932-55

TABLE 26

	Total Cases	Achlor-hydria	Hypo-chlor-hydria	Normal HCl Level	Hyper-chlor-hydria
Number	1,299	985	176	93	45
Per Cent	100.0	75.8	13.5	7.2	3.5

GASTRIC CANCER: THE LEVEL OF FREE HYDROCHLORIC ACID IN THE STOMACH OF 1299 (65.0 PER CENT) OF THE TOTAL NUMBER OF PATIENTS

SASKATCHEWAN 1932-55

TABLE 27

	Achlorhydria		Hypo-chlorhydria		Normal HCl and Liver		Hyper-chlorhydria	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Ulcerating	299	69.5	67	15.6	39	9.1	25	5.8
Infiltrating	126	72.0	25	14.3	19	10.8	5	2.7
Polypoid	90	91.8	6	6.2	1	1.0	1	1.0
Linitis	18	75.0	2	8.3	4	16.7	-	-
Cancer or Benign Ulcer	2	20.0	3	30.0	2	20.0	3	30.0

GASTRIC CANCER: LEVELS OF FREE HYDROCHLORIC ACID IN THE VARIOUS TYPES OF CANCER. DATA AVAILABLE IN 737 PATIENTS (36.9 PER CENT OF TOTAL NUMBER OF CASES)

SASKATCHEWAN 1932-55

TABLE 28

ulcerative lesions had the lowest incidence of achlorhydria and the highest incidence of hyperchlorhydria. Polypoid lesions were associated with the highest incidence of achlorhydria and the lowest incidence of hyperchlorhydria. In the distribution of acid levels the infiltrative lesions were intermediate between the ulcerating and polypoid types. The numbers of patients in each of the other groups were comparatively small and any conclusions drawn regarding the relationship between them and free hydrochloric levels would probably be erroneous.

It is of interest to observe the remarkable sameness in the incidence of hyperchlorhydria and the various sites of the primary tumour (Table 29). With the exception of anterior wall lesions of which there was a very small number, prepyloric lesions were associated with the greatest incidence of normal and greater than normal levels of free hydrochloric acid.

Fourteen per cent of all the patients in the series gave a history of prolonged digestive disturbance with more recent change in the character or degree of the old symptoms or the addition of new symptoms. In more than half of these patients the duration of the dyspeptic history was greater than five years (Table 30), and in more than three quarters of them was greater than three years. Two fifths of them had a history longer than ten years.

Ninety-eight per cent of these patients had received repeated and prolonged medical treatment for their gastric complaint before the onset of the significant change in their

SITE OF TUMOUR	DISTRIBUTION OF PATIENTS (PERCENT) IN EACH LEVEL OF FREE HCL.			
	Achlor-hydria	Hypochlor-hydria	Normal Acidity	Hyperchlor hydria
Prepylorus	67.9	18.0	10.6	3.5
Antrum	82.8	11.0	2.7	3.5
Lesser Curvature	76.2	13.7	6.7	3.4
Greater Curvature	82.1	8.5	5.1	4.3
Body	82.7	9.6	4.8	2.9
Cardia	78.0	16.0	3.0	3.0
Anterior Wall	58.9	5.8	29.5	5.8
Posterior Wall	85.0	10.0	5.0	-
Whole Stomach	78.5	9.2	10.8	1.5
All Cases	75.3	13.9	7.5	3.3

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE FREE HYDROCHLORIC ACID LEVEL AND THE SITE OF THE TUMOUR IN THE STOMACH. OBSERVATIONS IN 1,312 PATIENTS (65.6 PERCENT OF TOTAL NUMBER).

SASKATCHEWAN 1932-55

TABLE 29

Patients With Previous Gastric Symptoms	Duration of Previous Gastric Symptoms (Years)						
	All Cases	Less Than 3	3 - 5	6 - 9	10 - 19	20 or more	Not stated
Number	278	61	51	54	69	38	5
Per cent	100.0	22.0	18.3	19.4	24.8	13.7	1.8

GASTRIC CANCER: THE DURATION OF PROLONGED PREVIOUS GASTRIC SYMPTOMS

SASKATCHEWAN 1932-55

TABLE 30

symptomatology (Table 31).

In only two per cent of the whole group of 2,000 patients and in fourteen per cent of those with prolonged gastric symptoms, was there confirmation of a diagnosis of a previous or concomitant duodenal ulcer (Table 32). In thirty-six per cent, the ulcer history extended back for ten years or more (Table 33). An almost similar proportion of those with prolonged histories, but in whom no evidence of duodenal ulcer was apparent, had dyspepsia for ten or more years (Table 34).

A comparison between the levels of free hydrochloric acid in the gastric juice of these patients who had and had not duodenal ulceration is given in Table 35. It will be noted that, with approximately a similar proportion in each group in whom no data on acidity are available, there is a smaller proportion of patients with achlorhydria and a greater proportion with normal and greater than normal free hydrochloric acid in the group who also exhibited duodenal ulcer when compared with the patients without concomitant or previous ulcer. There does not appear to be a significant relationship between the levels of free hydrochloric acid and the duration of the previous prolonged gastric history (Table 36).

It may not be a statistically correct procedure, but it is of interest to withdraw those patients who had a long history of dyspepsia from the total number of patients and compare certain features of that group with those of the whole series. There would appear to be a greater incidence of patients in whom the regional

Patients With Previous Gastric Symptoms	All Cases	Had Prolonged Treatment	Had No Prolonged Treatment
Number	278	273	5
Per cent	100.0	98.2	1.8

GASTRIC CANCER: THE PROPORTION OF PATIENTS WHO RECEIVED PROLONGED MEDICAL TREATMENT FOR THE PREVIOUS DYSPEPTIC SYMPTOMS

SASKATCHEWAN 1932-55

TABLE 31.

Patients With History of Prolonged Gastric Symptoms	All Cases	History of Previous Duodenal Ulcer	No History of Duodenal Ulcer	Unstated
Number	278	39	228	11
Percent	100.0	14.0	82.0	4.0

GASTRIC CANCER: THE PROPORTION OF PATIENTS WITH PREVIOUS PROLONGED GASTRIC HISTORY AND THE PRESENCE OF DUODENAL ULCER

SASKATCHEWAN 1932-55

TABLE 32

Patients With Duodenal Ulcer	Duration of Ulcer History (Years)						
	All Cases	Less Than 3 Years	3 - 5	6 - 9	10-19	20 or more	Prolonged History But Exact Duration Unstated
Number	39	7	9	4	4	10	5
Percent	100.0	18.0	23.0	10.3	10.3	25.6	12.8

GASTRIC CANCER: THE DURATION OF THE PREVIOUS GASTRIC SYMPTOMS AMONG THE PATIENTS IN WHOM A DIAGNOSIS OF DUODENAL ULCER WAS ALSO CONFIRMED

SASKATCHEWAN 1932-55

TABLE 33

Patients Without Duodenal Ulcer or Unstated	Duration of Previous Gastric Symptoms (Years)						
	All Cases	Less Than 3 Years	3 - 5	6 - 9	10 - 19	20 or more	Long History But Exact Date Unstated
Number	239	54	42	50	65	28	0
Percent	100.0	22.6	17.6	20.9	27.2	11.7	0

GASTRIC CANCER: THE DURATION OF GASTRIC SYMPTOMS IN THOSE PATIENTS WHO EXHIBITED NO EVIDENCE OF HAVING HAD DUODENAL ULCER

SASKATCHEWAN 1932-55.

TABLE 34

Level of Free HCl.	Patients with Prolonged Gastric History (Per-cent)		
	All Cases	Those With Duodenal Ulcer	Those Without Duodenal Ulcer or Not Stated
Achlorhydria	45.0	39.5	46.4
Hypo-chlorhydria	10.4	14.0	9.5
Normal Level	8.1	11.6	7.2
Hyper-chlorhydria	4.3	7.0	3.6
Not stated	32.3	27.9	33.3

GASTRIC CANCER: THE LEVELS OF FREE HYDROCHLORIC ACID, THE GASTRIC JUICE OF THE PATIENTS WHO GAVE A LONG PREVIOUS HISTORY OF DYSPEPSIA
DATA AVAILABLE IN 188 PATIENTS (67.7% OF RELEVANT GROUP)
SASKATCHEWAN 1932-55

TABLE 35

Level of Free Hydrochloric Acid	Duration in Years of Symptoms Related to Free Hydrochloric Acid (Per cent)					
	Less Than 3 Years	3 - 5	6 - 9	10-19	20 or more	Those with long history but exact duration unstated
Achlorhydria	29.2	27.8	32.5	26.2	31.1	25.0
Hypochlorhydria	50.0	41.7	44.2	52.5	43.2	50.0
Normal Acidity	8.3	12.5	2.3	8.2	12.2	-
Hyperacidity	4.2	13.9	14.0	13.1	5.4	-
Not Stated	8.3	4.1	7.0	-	8.1	25.0
Total	100.0	100.0	100.0	100.0	100.0	100.0

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE LEVEL OF FREE HYDROCHLORIC ACID IN THE GASTRIC JUICE OF PATIENTS AND THE DURATION OF THE PROLONGED HISTORY

SASKATCHEWAN 1932-55

TABLE 36

lymphatic glands were uninvolved in the group with the long dyspeptic history (Tables 37 and 38). A somewhat similar finding is evident when the incidence of metastasis is considered (Tables 39 and 40).

It will be observed that there is little relationship between the long dyspeptic history and the incidence of regional glandular metastases (Table 41 and 42), and the incidence of extra glandular metastases (Table 43 and 44).

The influence of the long duration of symptoms upon operability and resectability is seen in Table 45. Although the operability rate was greater in those with the prolonged history as compared with that of the patients in the whole series, the resectability rate was markedly lower. This finding appears to be inconsistent with the evidence that the incidence of regional lymphatic glandular involvement and metastases was lower in those patients with the prolonged history.

	Proportion of Patients with Involved Glands (Per Cent)	Proportion of Patients with Uninvolved Glands (Per Cent)	Proportion of Patients in whom Glandular Status was Unstated (Per Cent)
Whole Group of Patients	44.7	11.5	43.8
Patients with Prolonged History	37.9	19.3	42.8

GASTRIC CANCER: COMPARISON BETWEEN THE INCIDENCE OF INVOLVED LYMPHATIC GLANDS IN THE WHOLE SERIES AND THE GROUP OF PATIENTS WHO HAD LONG DYSPEPTIC HISTORIES

SASKATCHEWAN 1932-55

TABLE 37

	Proportion of Patients with Involved Glands (Per cent)	Proportion of Patients with Uninvolved Glands (Per cent)
Whole Group of Patients	79.5	20.5
Patients with Long Dyspeptic Histories	66.3	33.7

GASTRIC CANCER: THE INCIDENCE OF INVOLVED REGIONAL LYMPHATIC GLANDS IN THE PATIENTS OF THE WHOLE SERIES AND THAT OF THE PATIENTS IN THE GROUP WHO GAVE LONG DYSPEPTIC HISTORIES, USING ONLY THOSE PATIENTS IN WHICH THE GLANDULAR STATUS HAD BEEN DETERMINED. IN 159 PATIENTS IN THE PROLONGED GROUP THE RELEVANT DATA WERE AVAILABLE

SASKATCHEWAN 1932-55

TABLE 38

	Proportion of Patients with Metastases (Per Cent)	Proportion of Patients with No Metastases (Per Cent)	Proportion of Patients in Whom Glandular Status was Not Determined
Whole Group of Patients	29.0	24.2	46.8
Patients with Long Dyspeptic Histories	22.1	34.8	43.1

GASTRIC CANCER: COMPARISON BETWEEN THE INCIDENCE OF METASTASES IN PATIENTS OF THE WHOLE SERIES AND IN THE GROUP OF PATIENTS WHO GAVE LONG HISTORIES OF DYSPEPSIA

SASKATCHEWAN 1932-55

TABLE 39

	Proportion of Patients with Metastases (Per cent)	Proportion of Patients without Metastases (Per cent)
Whole Group of Patients	54.6	45.4
Patients with Long Dyspeptic Histories	38.9	61.1

GASTRIC CANCER: THE INCIDENCE OF METASTASES IN THE PATIENTS OF THE WHOLE SERIES AND THAT OF THE PATIENTS WHO GAVE LONG DYSPEPTIC HISTORIES, USING ONLY THOSE PATIENTS IN WHOM THE DATA ON METASTASES WERE AVAILABLE. DATA WERE AVAILABLE IN 158 PATIENTS IN THE PROLONGED HISTORY GROUP

SASKATCHEWAN 1932-55

TABLE 40

Duration (Years)	Patients with Involved Nodes (Per Cent)	Patients with Uninvolved Nodes (Per cent)	Patients in Whom Nodal Pathology was Unknown (Per Cent)
1 - 3	50	33.3	16.7
3 - 5	41.9	14.9	43.2
6 - 9	22.7	13.7	63.6
10 - 19	40.9	23.0	36.1
20 or more	36.4	16.9	46.7
Actual Prolonged Duration Unstated	40.0	60.0	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE DURATION OF SYMPTOMS IN THOSE PATIENTS WHO GAVE LONG HISTORY OF DYSPEPSIA AND THE INCIDENCE OF INVOLVED REGIONAL LYMPHATIC GLANDS

SASKATCHEWAN 1932-55

TABLE 41

Duration (Years)	Patients with Involved Nodes (Per cent)	Patients with Uninvolved Nodes (Per cent)
1 - 3	60.0	40.0
3 - 5	73.8	26.2
6 - 9	62.5	37.5
10 - 19	64.0	36.0
20 or more	68.0	31.2
Actual prolonged duration unstated	40.0	60.0

GASTRIC CANCER: THE COMPARATIVE INCIDENCE OF INVOLVED AND UNINVOLVED REGIONAL LYMPHATIC GLANDS IN THE PATIENTS WHO GAVE LONG DYSPEPTIC HISTORIES AND IN WHOM GLANDULAR STATUS HAD BEEN DETERMINED IN TERMS OF DURATION OF SYMPTOMS. DATA WERE AVAILABLE IN 157 PATIENTS IN THE GROUP WITH PROLONGED HISTORY

SASKATCHEWAN 1932-55

TABLE 42

Duration (Years)	Patients with Metastases (Per Cent)	Patients with No Metastases (Per Cent)	Patients in Whom Unknown Whether Metastases Present
1 - 3	28.0	44.0	28.0
3 - 5	20.3	33.8	45.9
6 - 9	15.9	31.8	52.3
10 - 19	22.9	37.7	39.4
20 or more	26.3	31.6	42.1
Actual Prolonged Duration Unstated	-	40.0	60.0

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE DURATION OF SYMPTOMS IN THOSE PATIENTS WHO GAVE LONG HISTORIES OF DYSPEPSIA AND THE INCIDENCE OF EXTRA GLANDULAR METASTASES

SASKATCHEWAN 1932-55

TABLE 43

Duration (Years)	Patients with metastases (Per cent)	Patients with no metastases (Per cent)
1 - 3	38.9	61.1
3 - 5	37.5	62.5
6 - 9	33.3	66.7
10 - 19	37.8	62.6
20 or more	45.5	54.5

GASTRIC CANCER: THE COMPARATIVE INCIDENCE OF METASTASES IN THOSE PATIENTS WHO GAVE LONG DYSPEPTIC HISTORIES AND IN WHOM THE PRESENCE OR ABSENCE OF METASTASES WAS DETERMINED IN TERMS OF DURATION OF SYMPTOMS. DATA AVAILABLE IN 154 IN THE PROLONGED HISTORY GROUP

SASKATCHEWAN 1932-55

TABLE 44

	Patients with Prolonged Histories (Per cent)	Whole Group of Patients (Per cent)
Operability	63.3	59.5
Resectability	37.0	48.6

GASTRIC CANCER: COMPARISON OF THE OPERABILITY AND RESECTABILITY RATES IN THOSE PATIENTS WITH PROLONGED HISTORIES, WITH THOSE OF THE WHOLE SERIES

SASKATCHEWAN 1932-55

TABLE 45

CHAPTER 5

BENIGN AND MALIGNANT GASTRIC ULCERATION

Since Baillie (1818) and Cruveilhier (1829, 1835) first made the distinction between chronic ulcer and cancer of the stomach, the diagnostic differentiation between the two conditions has presented an absorbing problem to the physician, surgeon, radiologist and general practitioner alike. There are few experienced clinicians who have not, on some occasion, been doubtful about the pathological character of a gastric ulcer even after the patient has been thoroughly investigated. He is indeed a fortunate physician who has consistently avoided the pitfall of assuming the benignity of an ulcer because of an apparently classical clinical picture presented by the patient. Even the surgeon, with the patient's abdomen open and the involved portion of stomach in his hands, not infrequently feels uncertain whether he is dealing with a benign or a neoplastic ulcer. The

tragedy of permitting a small carcinomatous ulcer of the stomach to progress to the stage of non-resectability because of the acceptance of an unproven diagnosis of benign ulcer is not uncommon. Moynihan emphasised this in his aphorism "One cause of death in cancer of the stomach is the unsuccessful medical treatment of the malignant lesion that masquerades as a benign ulcer."

THE INCIDENCE OF DIAGNOSTIC ERROR

Reports from some of the larger American medical centres indicate that the incidence of error in distinguishing between benign and malignant gastric ulcers is not small and confirm that the neoplastic ulcer may simulate the simple one in a relatively high proportion of cases. Eusterman (1940) found that at least five per cent of the lesions unequivocally diagnosed as benign ulcer by the radiologists at the Mayo Clinic were found to be malignant. Other reports (Table 46) demonstrate that, even with the greatest care and the employment of all investigative measures, certain ulcers, short of histological examination, defy correct diagnosis.

There is no single clinical criterion by which an accurate differentiation between benign and malignant gastric ulcer can be made. On the basis of repeated clinical and pathological observations certain factors of relative diagnostic value have been evolved. These factors, which include the age of the patient, the site of the lesion, the duration and character

Author	Year	Number of Cases of Presumed Benign Ulcer	Diagnostic Error (Per Cent)
Allen and Welch	1941	277	14.0
Smith and Jordan	1948	600	9.8
Marshall and Welch	1948	131	20.0
Welch and Allen	1949	295	10.8
Lampert et al	1950	550	13.0
Johnsson et al	1950	219	11.0

DIAGNOSTIC ERROR IN ULCERATIVE

LESIONS OF THE STOMACH

TABLE 46

of the symptoms, the degree of acidity, the size of the ulcer and the response by the ulcer to medical treatment, may contribute towards differentiating between the two types of gastric ulcers but none of them applied singly, nor all of them collectively, will necessarily provide the correct diagnosis in all cases.

To examine the problem a comparative study was made of those patients who were referred to the clinics of the Saskatchewan Cancer Commission between the years 1932 and 1953 because of the presence of gastric ulcers of undetermined type. Those with lesions which subsequently proved to be benign (284 cases) were compared with the patients in the gastric cancer series who presented with carcinomatous ulcers (489 cases). The group of benign ulcers studied may not be representative of those which are found in every day general practice in that they had given rise to especial diagnostic difficulty, but they may serve better, despite this special selection, to illustrate the similarity in the clinical presentation of the two pathologically very different types of lesion.

The malignant lesions studied are those with true carcinomatous ulcers in contradistinction to ulceration in a gastric cancer. This distinction is necessary since, for example, ulceration of a polypoid lesion may be dissimilar in behaviour and presentation to an ulcer in the stomach wall which is found to be malignant in type.

SEX INCIDENCE OF BENIGN AND MALIGNANT ULCERATION

Cancer of the stomach occurs more commonly in males than in females. The same preponderance in males is evident when only the ulcerative form of the disease is considered. Table 47 demonstrates that, in the comparative series of benign ulcers, approximately the same sex incidence occurred as that in malignant ulcers.

AGE AND SEX DISTRIBUTION IN BENIGN AND MALIGNANT GASTRIC ULCERATION

Table 48 demonstrates the age distribution of the patients with each type of lesion. In each, and in both sexes, the greatest incidence is in the 55 - 64 years age group. Approximately one quarter of the benign lesions occur in patients older than sixty-five years, while 38.9 per cent of the malignant lesions are present in the same age group. The findings of this survey compare with those recorded by Comfort (1937). He found that the relative incidence of benign ulcer was somewhat greater than that of ulcerating cancer in all decades with the exception of the eighth. In this decade ulcerating cancer was found to be almost twice as frequent as benign gastric ulcer. This evidence is in contrast to that reported by Alvarez (1931) who found that in the sixth, seventh and eighth decades, benign ulcer occurred almost as frequently as malignant ulcer, and Boudreau (1951) who found the ratio of benign to malignant ulcers

Type of Ulcer	Total Number of Patients	Number of Males	Number of Females	Ratio Between Male and Female Incidence
Malignant	489	367	122	3.0/1
Benign	284	220	64	3.4/1

BENIGN AND MALIGNANT ULCERATION

RELATIONSHIP BETWEEN SEX INCIDENCE

SASKATCHEWAN 1932-53

TABLE 47

Type of Ulcer	Sex	All Cases	AGE DISTRIBUTION PER CENT OF TOTAL CASES IN EACH GROUP						
			0 - 24	25-34	35-44	45-54	55-64	65-69	70 & over
Malignant	Total	100.0	-	2.3	7.4	16.1	35.3	13.7	25.2
	Male	100.0	-	1.9	7.4	16.1	34.9	14.3	25.4
	Female	100.0	-	3.3	7.4	15.6	36.9	12.3	24.6
Benign	Total	100.0	0.4	1.8	9.2	25.3	37.3	13.7	11.9
	Male	100.0	-	1.3	8.6	25.5	38.2	14.6	11.8
	Female	100.0	1.6	3.2	10.9	25.0	35.9	10.9	12.5

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE RELATIONSHIP BETWEEN AGE AND SEX INCIDENCE

SASKATCHEWAN 1932-53

TABLE 48

in the same age group to be more than two to one.

THE SITE OF BENIGN AND MALIGNANT GASTRIC ULCERATION

There is no part of the stomach which is immune from either benign or malignant ulceration. Although there are certain sites which appear to exhibit a predilection for one or other type, there is a sufficient degree of overlap to make the decision regarding benignancy or malignancy a difficult one when an individual case is under consideration.

Holmes and Hampton (1932) believed that benign ulceration within the prepyloric area was of such infrequent occurrence that all ulcers in this site should be considered malignant until proven otherwise. Their analysis of cases seen at the Massachusetts General Hospital showed that carcinomatous ulcers outnumbered benign ones by twelve to one. Sampson and Sosman (1939) studied 545 cases of gastric ulcer from the Peter Bent Brigham Hospital in Boston. Of the 57 prepyloric ulcers found, 46 were carcinomatous and eleven were benign. Allen (1945) reported that 65 per cent of ulcers occurring within the two centimetres immediately proximal to the pylorus were malignant. Boudreau et al (1951) found 58 per cent of all malignant and 56 per cent of all benign gastric ulcers occurred within the prepyloric area. Benedict (1950) reported a similar incidence of benign and malignant lesions at the prepylorus and Singleton (1936) found benign prepyloric ulcers to be twice as frequent as

the malignant variety in his series from the Toronto General Hospital.

Boyce (1953) made a general statement that the commoner sites of benign ulcer and carcinomatous lesions do not coincide. He found that the commonest site for benign ulcer was the lesser curvature and that only 12 per cent of gastric carcinomata occurred in this region. In Allen's (1945) experience, only ten per cent of lesser curvature ulcers were malignant. Benedict (1950), believed that 75 per cent of lesser curvature ulcers were benign. Sussman (1950) agrees that most lesser curvature ulcers are benign, but states that an ulcer high on that curvature is more likely to be malignant than one situated distally.

There appears to be unanimity of opinion in respect of the serious import of the appearance of an ulcer on the greater curvature of the stomach. Benedict (1950) reported that only rarely was such an ulcer benign. This view was supported by Sampson (1939) and Allen (1945). Boudreau (1951) on the other hand found that the incidence of benign ulcer on the greater curvature was as great as that of malignant ulcer.

Table 49 summarizes the data obtained in the Saskatchewan study. Eighty per cent of all the benign lesions occurred on the lesser curvature, the remaining twenty per cent being distributed over the prepyloric, antral, greater curvature and fundal, posterior wall and cardiac regions of the stomach.

Type of Ulcer	All Cases	SITE OF ULCER NUMBER OF PATIENTS									
		Unstated	Prepylorus	Antrum	Lesser Curvature	Greater Curvature and Fundus	Anterior Wall	Posterior Wall	Body	Cardia	Large Ulcer Involving More Than One Site
Malignant	489	3	177	60	133	30	4	10	30	38	4
Benign	284	73	11	12	170	7	-	4	-	7	-
		SITE OF ULCER PER CENT OF STATED CASES IN EACH GROUP									
Malignant	486	-	36.3	12.4	27.3	6.2	0.9	2.1	6.2	7.8	0.8
Benign	211	-	5.2	5.7	80.6	3.3	-	1.9	-	3.3	-

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE SITE OF THE LESION IN EACH TYPE OF ULCER

SASKATCHEWAN 1932-53

TABLE 49

Carcinomatous ulceration occurred most frequently at the prepylorus, but more than a quarter of the total number of patients had their lesion on the lesser curvature.

Table 50 shows the site distribution of benign and malignant ulcers when all the cases of gastric ulceration in the combined series are considered collectively. It is notable that the prepylorus is involved in benign ulceration only infrequently, whereas the same site is common for malignant involvement. The lesser curvature does not enjoy the freedom from malignant ulceration that some authors would suggest. Confirmation of the frequency of involvement at various sites is apparent in Table 51.

THE DURATION OF SYMPTOMS OF BENIGN AND MALIGNANT GASTRIC ULCERS

It has been stated by Alvarez (1931), that the duration of symptoms is an important factor in the diagnostic decision regarding the benign or malignant character of a gastric ulcer. He found in his series that 79 per cent of patientswith gastric cancer had had symptoms for less than two years and on this evidence based his opinion that a history of relevant symptoms over a lesser period of time than this in a patient who had no previous dyspeptic complaints favoured a diagnosis of cancer, whereas a long history, over many years, was more indicative of a benign ulcer. Allen (1945), reported that patients with

TYPE OF ULCER	DISTRIBUTION OF ULCERS ACCORDING TO SITE AND TYPE (PER CENT)								
	Prepylorus	Antrum	Lesser Curvature	Greater Curvature and Fundus	Anterior Wall	Posterior Wall	Body	Cardia	Large Ulcer Involving More Than One Site
Malignant	25.4	8.6	19.1	4.3	0.6	1.4	4.3	5.5	0.6
Benign	1.6	1.7	24.3	1.0	-	0.6	-	1.0	-

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE DISTRIBUTION OF BENIGN AND MALIGNANT GASTRIC ULCERATION IN A TOTAL NUMBER OF 697 PATIENTS ACCORDING TO SITE

SASKATCHEWAN 1932-53

TABLE 50

SITE OF ULCER	TYPE OF ULCER			
	MALIGNANT		BENIGN	
	NUMBER	PER CENT	NUMBER	PER CENT
Prepylorus	177	94.2	11	5.8
Antrum	60	83.3	12	16.7
Lesser Curvature	133	43.9	170	56.1
Greater Curvature and Fundus	30	81.1	7	18.9
Anterior Wall	4	100.0	-	00.0
Posterior Wall	10	71.4	4	28.6
Body	30	100.0	-	00.0
Cardia	38	84.4	7	15.6
Large Ulcer Involving More Than One Site	4	100.0	-	00.0

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE PROPORTION OF BENIGN AND MALIGNANT ULCERS WHEN THE TOTAL NUMBER OF ULCERS IN EACH SITE ARE CONSIDERED

TABLE 51

ulcerative lesions of the stomach after middle life, having had symptoms for less than one year, were five times more likely to have cancer than benign ulcer. He also pointed out that in patients of this age group who had had symptoms for five or more years, the reverse was true.

Comfort (1950), reviewed 648 cases of both benign and malignant ulcers less than four centimetres in diameter and found that of those ulcers of less than one year's duration as indicated by the patients' histories, 38 per cent were malignant. Of those patients with symptoms of between one and two years' duration, 30 per cent of the gastric ulcers were malignant. The ulcers in patients with symptom duration of between two and four years, five and nine years, and over ten years were malignant in 15 per cent, 15 per cent and 8 per cent respectively. These data indicated that the longer the duration, as determined on the basis of the history of symptoms, the smaller was the chance that the ulcer was malignant.

In the Saskatchewan series 91.2 per cent of the patients with malignant ulceration had symptoms for two years or less, while only 46.4 per cent of the patients with benign lesions had similar duration (Table 52). In more than half of the cases of benign ulceration, the duration of symptoms was greater than two years.

Type of Ulcer	Total Cases	DURATION, IN MONTHS, OF SYMPTOMS FROM ONSET TO TREATMENT						
		Less Than one Month	1 - 3	4 - 6	7 - 12	13-24	25 and Over	Un-stated
Cancer	489	9	138	99	106	63	40	34
Benign	284	8	32	29	28	26	142	19
		DURATION OF SYMPTOMS: PER CENT OF STATED CASES IN EACH GROUP						
Cancer	455	1.9	30.4	21.8	23.3	13.8	8.8	-
Benign	265	3.0	12.1	10.9	10.6	9.8	53.6	-

BENIGN AND MALIGNANT GASTRIC ULCERATION

DURATION OF SYMPTOMS FROM ONSET TO FIRST TREATMENT IN EACH TYPE

SASKATCHEWAN 1932-53

TABLE 52

THE SYMPTOM PATTERN IN BENIGN AND MALIGNANT GASTRIC ULCERATION

There are certain symptoms which are common to both benign and malignant gastric ulcer. A survey was done on the cases which presented at the clinics of the Saskatchewan Cancer Commission in order to determine whether there was any difference in the frequency of occurrence of certain of those symptoms between the two groups (Table 53).

The incidence of pain, nausea, vomiting, dysphagia and regurgitation was comparable in the series of benign and malignant ulcers. Weakness and constipation were common to both, but with slight preponderance in the patients with cancer. Anorexia, postprandial fullness, loss of weight and palpable mass were much more evident in malignant ulceration than in benign, while diarrhoea, haematemesis and melaena were commoner in the benign group.

Enquiry into some of the characteristics of pain was unsatisfactory due to the paucity of information recorded on the patients' charts. In only 152 of the patients with malignant ulceration were data available on the relationship between the onset of pain and taking food. In one hundred of the benign lesions this information was also lacking. Information on the relief of pain by food and alkali was also deficient in 284 of the malignant lesions and on 86 of the benign ulcers. The available data are presented in Table 54. In both types of lesion the pain occurred

Type of Ulcer	INCIDENCE OF SYMPTOMS. NUMBERS OF PATIENTS IN WHOM STATED SYMPTOMS APPEARED															
	Unstated	None	Pain	Anorexia	Post Prandial Fullness	Weakness	Vomiting	Constipation	Loss of Weight	Diarrhea	Dysphagia	Haematemesis	Nausea	Palpable Mass	Regurgitation	Melaena
Cancer	14	7	355	237	182	182	206	194	367	22	20	35	107	123	34	40
Benign	-	-	216	81	54	93	123	92	125	21	13	36	68	9	24	51
	INCIDENCE OF SYMPTOMS (PER CENT OF ULCER CASES IN EACH GROUP)															
Cancer	-	1.5	74.9	49.9	38.3	38.3	43.4	40.9	77.3	4.6	4.2	7.2	22.5	25.9	7.1	8.4
Benign	-	-	79.6	28.5	19.0	32.7	43.3	32.4	44.0	7.4	4.6	12.7	23.9	3.2	8.3	17.9

BENIGN AND MALIGNANT GASTRIC ULCERATION

COMPARATIVE SYMPTOMATOLOGY

SASKATCHEWAN 1932-53

TABLE 53

Type of Ulcer	Total Number of Patients	Number of Patients with Pain	Relation to Food			Pain Relieved By		
			Before	After	Not Stated	Food	Alkali	Not Stated
Cancer	489	355	34	118	203	34	37	284
Benign	284	221	43	78	100	57	78	86
	Per Cent of Stated Cases							
Cancer	489	355	22.4	76.6	-	47.9	52.1	-
Benign	284	221	35.5	64.5	-	42.2	57.8	-

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE RELATIONSHIP BETWEEN PAIN AND THE TAKING OF FOOD

THE RELIEF OF PAIN

TABLE 54

after food with greater frequency than before, but this was apparent more frequently in malignant ulcers. The pain was relieved by food and alkali in both groups with comparable frequency.

Weight loss was a common feature of both conditions. The loss was calculated for each patient and expressed as a percentage of his normal weight (Table 55). The distribution of weight loss is similar in both conditions where the loss is less than twenty per cent of normal body weight. The only material difference occurs when the weight loss exceeds that level. Five per cent of patients with benign ulcer give a history of weight loss greater than one fifth of their normal body weight, whereas more than sixteen per cent of those with malignant ulceration exhibit this degree of weight loss.

The incidence of haematemesis in the two types of ulceration differs, with preponderance of incidence in the benign cases (Table 56). This parallels the comparative incidence of gross melaena.

FREE HYDROCHLORIC ACID VALUES IN BENIGN AND MALIGNANT GASTRIC ULCERATION

The values of free hydrochloric acid, after histamine, frequently influence judgement in concluding, preoperatively, whether a gastric ulcer is malignant or benign. La Due (1950), reported that gastric ulceration in a patient who also exhibited histamine fast achlorhydria was at least seven times more likely to be malignant than benign. Kiernan (1950), stated that the

Type of Ulcer	Total Number of Patients with Stated Data	WEIGHT LOSS CALCULATED AS PERCENTAGE OF NORMAL BODY WEIGHT																	
		None or Voluntary Loss		0-5		5-10		10-15		15-20		20-25		25-30		30-35		Greater Than 35	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Cancer	359	27	7.5	52	14.5	82	22.8	84	23.4	56	15.6	33	9.2	13	3.6	6	1.7	6	1.7
Benign	224	45	20.0	32	14.2	50	22.2	59	26.2	28	12.4	9	3.9	2	0.8	1	0.3	-	-

BENIGN AND MALIGNANT GASTRIC ULCERATION

WEIGHT LOSS COMPARISON BETWEEN THE TWO GROUPS

SASKATCHEWAN 1932-53

TABLE 55

Type of Ulcer	Number of Patients With Haematemesis	Per Cent of Total Number of Patients in Each Group
Cancer	35	7.2
Benign	36	12.7

BENIGN AND MALIGNANT GASTRIC ULCERATION

INCIDENCE OF HAEMATEMESIS
IN EACH TYPE OF ULCER

SASKATCHEWAN 1932-53

TABLE 56

presence of achlorhydria in a patient with gastric ulceration usually indicated that the lesion was malignant, irrespective of a benign appearance on radiological examination. Corroborative evidence has been presented by Smith and Jordan (1948), Welch and Allen (1949), Comfort (1937), La Due (1950), and many others.

Achlorhydria in the presence of gastric ulceration is not a sine qua non for the diagnosis of cancer. Some benign ulcers do occur in the achlorhydric patient, and some carcinomatous ulcers are associated with normal and even hyperchlorhydria.

The data presented in this series (Tables 57 and 58), indicate that the determination of free hydrochloric acid levels may increase the index of suspicion regarding the malignancy of a gastric ulcer if the patient is found to be achlorhydric, but demonstrate also that as an aid to differential diagnosis in the individual case, it is unreliable.

ANAEMIA IN ASSOCIATION WITH BENIGN AND MALIGNANT GASTRIC ULCERATION

The incidence of anaemia is high in gastric ulceration. In patients with benign ulcer approximately 40 per cent (Table 59) exhibited some degree of anaemia, but it was exceptional for this anaemia to be severe. In malignant ulcers, however, two thirds of the patients were anaemic and in one fifth the haemoglobin levels were less than sixty per cent of normal.

Type of Ulcer	Total Number of Patients with Relevant Data	DEGREE OF FREE HYDROCHLORIC ACID							
		Achlorhydria With Histamine		Hypochlorhydria With Histamine		Normal Acidity With Histamine		Hyperchlorhydria With Histamine	
		No.	%	No.	%	No.	%	No.	%
Cancer	347	248	71.5	53	15.3	29	8.4	17	4.8
Benign	245	49	20.0	58	23.7	73	29.8	65	26.5

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE LEVELS OF FREE HYDROCHLORIC ACID IN EACH TYPE OF ULCER

SASKATCHEWAN 1932-53

TABLE 57

Free Hydrochloric Levels After Histamine	Total Number of Cases	TYPE OF ULCER			
		CANCER		BENIGN	
		Number	Per Cent	Number	Per Cent
Achlorhydria	297	248	83.5	49	16.5
Hypochlorhydria	111	53	47.7	58	52.3
Normal Acidity	102	29	28.4	73	71.6
Hyperchlorhydria	82	17	20.7	65	79.3

BENIGN AND MALIGNANT GASTRIC ULCERATION

DISTRIBUTION OF BENIGN AND MALIGNANT ULCERS ACCORDING TO FREE HYDROCHLORIC LEVELS

SASKATCHEWAN 1932-53

TABLE 58

Type of Ulcer	Total Cases With Relevant Data	HAEMOGLOBIN LEVELS (PER CENT OF THE NORMAL 15.6 g. PER 100 cc.)															
		Less Than 30		30 - 39		40 - 49		50 - 59		60 - 69		70 - 79		80 - 89		90 and Greater	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Cancer	417	6	1.4	16	3.8	22	5.3	44	10.6	55	13.2	77	18.5	59	14.1	138	33.1
Benign	284	1	0.4	5	1.8	9	3.2	7	2.5	14	4.9	23	8.1	55	19.3	170	59.8

BENIGN AND MALIGNANT GASTRIC ULCERATION

COMPARISON OF HAEMOGLOBIN VALUES IN THE TWO GROUPS, WHEN FIRST REPORTING FOR TREATMENT

SASKATCHEWAN 1932-53

TABLE 59

THE SIGNIFICANCE OF THE SIZE OF THE ULCER IN THE DIFFERENTIATION BETWEEN A BENIGN AND MALIGNANT LESION

There is difference of opinion on whether the size of a gastric ulcer is a valid distinguishing feature between the malignant and benign types. The Mayo Clinic staff are convinced that almost every ulcerative lesion of the stomach which is more than four centimetres in diameter is cancerous. They routinely advise surgery for any ulcer which has a greater diameter than 2.5 cms. because they consider that the chance of such an ulcer being malignant is much greater than the risk associated with surgical therapy (Comfort 1950). Stout (1950) from the pathologist's standpoint, agrees that very few ulcers larger than four centimetres in diameter are benign. Others in agreement with this opinion are Lampert (1950), Alvarez (1928) and Benedict (1950).

The contrary opinion that the size of an ulcer is not a reliable basis for distinction between benign and malignant ulcer is held by Golden (1950), Jordan (1950), Boudreau (1951) and Helmer (1946).

Only 8.2 per cent of the malignant ulcers in the Saskatchewan series were less than two centimetres in diameter, while more than three quarters were larger than four centimetres. Of the benign ulcers, 86.3 per cent were less than two centimetres in diameter and only 3.2 per cent were greater than four centimetres (Table 60). Considering all of the ulcers in the two groups

Type of Ulcer	Total Cases With Relevant Data	SIZE OF LESION (DIAMETER IN CENTIMETRES)					
		0 - 2.0		2.1 - 4.0		4.1 & greater	
		No.	%	No.	%	No.	%
Cancer	282	23	8.2	44	15.6	215	76.2
Benign	95	82	86.3	10	10.5	3	3.2

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE SIGNIFICANCE OF THE SIZE OF THE ULCER

SASKATCHEWAN 1932-53

TABLE 60

collectively, 78 per cent with diameters of two centimetres or less, were benign and 98.6 per cent of the ulcers with a diameter greater than four centimetres were malignant (Table 61).

THE RADIOLOGICAL DIFFERENTIATION BETWEEN BENIGN AND MALIGNANT GASTRIC ULCERATION

It is not within the competence of the writer to discuss authoritatively, the radiological differentiation between benign and malignant gastric ulceration. Even to the highly trained and experienced roentgenologist there may be considerable difficulty in distinguishing between the two conditions. In a critical evaluation of a particular lesion the factors of location of the ulcer, its depth of penetration into the stomach wall, its contour and size and evidence of discontinuity of peristalsis and disturbance of the normal mucosal pattern in the immediate vicinity, together with the presence or absence of associated spastic phenomena are taken into consideration. In certain cases the radiologist is extremely cautious in expressing an opinion concerning the type of ulcer following a single radiological examination.

Ulcers in the prepyloric region and on the greater curvature are considered more likely to be malignant, while those on the lesser curvatures are more likely to be benign. Malignant ulcers frequently show irregularity of contour while the outline of benign lesions is characteristically smooth, unless distortion occurs as a result of the presence in the crater of

Size of Ulcer (Diameter in cms.)	Total Number of Cases	TYPE OF ULCER			
		CANCER		BENIGN	
		No.	%	No.	%
0 - 2.0	105	23	21.9	82	78.1
2.1 - 4.0	54	44	81.5	10	18.5
4.1 and Greater	218	215	98.6	3	1.4

BENIGN AND MALIGNANT GASTRIC ULCERATION

THE SIGNIFICANCE OF THE SIZE OF THE ULCER

SASKATCHEWAN 1932-53

TABLE 61

necrotic slough, granulation tissue, a mucous plug or food. Although the size of the crater does not constitute adequate grounds for a definite diagnosis, very large craters, especially if they are shallow, are frequently malignant.

The mucosal folds adjacent to a benign ulcer run into the crater. In a carcinomatous ulcer the mucosal folds often point to the crater, but they do not usually reach it. This sign is obviously absent in those cases in which the mucous membrane of the stomach is flattened and smooth as a result of atrophic gastritis.

The depth of penetration of the ulcer crater into the gastric wall may be of considerable diagnostic significance. The greater the penetration, the greater is the chance of the ulcer being benign (Bockus 1944). The degree of rigidity of the gastric wall adjacent to the ulcer may provide a diagnostic clue in the differentiation between the two types of ulcer. While shallow carcinomatous ulcers "may more or less rise with peristalsis as a plank rides a wave in the ocean" (Sussman 1950), it is usually possible to detect rigidity of the wall surrounding the ulcer. The more rigid the adjacent wall, the more likely it is that cancer is present. Interruption of peristalsis in the region of an ulcer may not always be indicative of the presence of cancer. Similar appearances may be seen around a chronic callous benign ulcer with marked fibrous reaction in the adjacent gastric wall. The association of a deep smooth spastic incisura on the greater

curvature opposite an ulcer niche is usually indicative that the ulcer is benign (Bockus 1944). The exception to this is in the prepyloric region, where the persistence of such an incisura on the greater curvature often indicates a small malignancy on the lesser curvature side.

The meniscus sign described by Carmen (1920), may be, for practical purposes, pathognomonic of ulcerating carcinoma, but is too inconsistent in carcinomatous ulcer to be of value in the differentiation from a benign lesion. The sign may be present in benign ulcer if the edges of the lesion are oedematous and raised.

In doubtful cases and in the absence of achlorhydria, if the ulcer niche is of the deeply penetrating type extending beyond the lesser curvature, a three or four weeks' trial of well controlled medical therapy followed by repeated radiological examination is justified. Lack of healing at the end of that time may be evidence that the ulcer is of the malignant type. A decrease in the size of the ulcer is not conclusive evidence of benignity. The appearance of a decrease in the size may be indicative of healing of the ulcer, subsidence of oedema of the edges of the ulcer with the depth of the ulcer becoming apparently less, filling of the crater by radiolucent material such as mucous, food, blood clot or tumour tissue, or scirrhous contracture of the ulcer. Reduction in size of the niche in benign ulcer occurs usually in depth and width simultaneously, while malignant ulceration may diminish in depth only, or in greater degree than in transverse diameter.

GASTROSCOPY IN THE DIFFERENTIATION BETWEEN BENIGN AND MALIGNANT GASTRIC ULCERATION

Gastric endoscopy is the natural complement to radiological examination of the stomach. Each method has its limitations, but the limitations of one rarely overlap those of the other. Perhaps the most notable service provided by gastroscopy is in the differentiation between benign and carcinomatous ulceration. The classical malignant ulcer with its thick raised edge is easily recognized provided the lesion is within the visual field of the instrument. There is, similarly, little difficulty in recognizing the typical benign ulcer. In such clear cut cases, however, the radiologist can give a fairly confident diagnosis. The difficulty arises in those patients in whom the radiological findings are not conclusive, or in whom the radiologist reports the findings of his examination as indicative of benign ulceration, when the clinical history and the other ancillary methods of examination suggest a diagnosis of malignant ulceration. The gastroscopic signs which suggest that a gastric ulcer is malignant are:

(a) a limiting wall on one side of the ulcer and blending infiltration on another.

(b) nodularity in the immediate environment of the ulcer.

(c) irregular nodularity and the presence of ridges of tissue on the ulcer floor.

(d) bleeding at the edge of the ulcer.

(e) diffuse infiltration of the stomach wall, even without actual visualization of the ulcer itself.

Schindler (1938), points out that the size of the ulcer, the presence of oedema in and irregularity of the edge of the ulcer are not proof of malignancy.

In contrast, the gastroscopic signs in favour of a diagnosis of benign ulcer are:

(a) a sharp edge without a surrounding wall.

(b) no infiltration of surrounding gastric mucosa.

(c) haemorrhage and pigment present in an otherwise normal mucosa.

(d) Henning's sign (arch-shaped distortion of the angularis).

(e) Mucosal folds converging on the ulcer.

Over the past few years gastroscopic examination has been extended to include gastric mucosal biopsy, but to date this has not been found practical in the differentiation between benign and malignant ulcer.

EXFOLIATIVE CYTOLOGY IN THE DIFFERENTIATION BETWEEN BENIGN AND MALIGNANT GASTRIC ULCERATION

The value of the exfoliative cytological method in the diagnosis of gastric cancer has been demonstrated by Cooper (1953), Graham (1954), Panico et al (1950) and Ayre (1953). It is not a new

diagnostic method. Marini in 1909 published the results of his classical study of gastric cytodiagnosis and predicted that "the day will come when physicians are as certain of the advantages of cytodiagnosis by lavage as they are now of urinary sediment examination." Even at this early date, the procedure was not a new one, but merely the revival of a clinico-pathological examination as old as the cellular concept of pathology itself. The method has a sound biological and experimental basis in that it depends upon the metastatic potentialities of malignant tumours, as modified by the growth rate of the individual lesion (Lemon 1952), and the reduced mutual adhesiveness of adenocarcinomatous cells as compared with normal epithelium [Coman (1944) and McCutcheon (1948)] . As a result of this reduced adhesiveness and greater growth rates of many gastric carcinomata, tumour cells may be exfoliated into the gastric lumen in quantity out of all proportion to the ratio of tumour surface area and the area of normal gastric epithelium. Several major difficulties in obtaining reasonable diagnostic accuracy with this method have been experienced. Perhaps the greatest of these has been the failure of approximately 40 per cent of gastric carcinomata to shed recognizable clumps of tumour cells into the gastric lumen. [Seybolt et al (1951), Iverson (1951), Richardson (1949), and Wollum (1951)].

Several ingenious methods of obtaining an abundance of well preserved exfoliated tumour cells have been developed.

Moderate success was claimed following gastric lavage with a special perforated tube [Graham et al (1948), Ulfelder et al (1948) and Imbriglia et al (1951)] . Hollander et al (1947) obtained a better yield of desquamated tumour cells by introducing a two per cent eugenol water suspension into the stomach before aspiration and Rosenthal and Traut (1951), published an account of their experience with mucolytic papain used in the same manner.

In carcinomatous ulcer, where exfoliation occurs with lesser frequency and in lesser degree than in polypoid cancers, the abrasive balloon, designed by Panico et al (1950), and modified by Rubin (1953), provides a means whereby cells adherent to the edges of the ulcer may be removed and made available for histological examination. The introduction into the stomach of the mucolytic agent chymotrypsin before passing the abrasive balloon improves the yield of cells and ensures against cytolysis. The Ayre brush, the purpose of which is similar to that of the abrasive balloon, has been used with success to provide sheets of cells suitable for histological assessment and has the added advantage of permitting comparative study of cells obtained from the different levels of the gastric mucosa.

From the theoretical point of view this type of investigation is of value provided it is found possible to obtain cells from the ulcer but it has been found in practice that the yield of cells from an ulcer is small.

UROPEPSIN EXCRETION AS A MEANS OF DISTINGUISHING BENIGN FROM MALIGNANT GASTRIC ULCERATION

Efforts have been made to determine the value of the quantitative uropepsin excretion in differentiating benign from malignant gastric ulcers. Certain studies in this field have been equivocal, [Janowitz et al (1950), Eastcott et al (1953), Bolt et al (1954), Balfour (1954) and Sircus (1954)] and the number of investigations on patients with gastric carcinoma too small for adequate evaluation. Bieling (1911), Takeda (1910), and Rothschild (1930), found low uropepsin values in a small number of patients with widespread infiltrative gastric cancer.

Gray (1955) concluded that, in the presence of an ulcerating gastric lesion, a uropepsin excretion of 1000 units or less indicated a high probability that the ulcer was malignant and that a uropepsin excretion of over 3000 units favoured a benign lesion. The test is by no means infallible and is probably the least efficient of the differential diagnostic aids.

CHAPTER 6

GENERAL FEATURES

It would be unwise to comment upon the results of therapy in gastric cancer without some knowledge of the general characteristics of the disease, especially in terms of its localisation and spread. Only upon these factors can the prognosis be determined.

SITE OF TUMOUR

Until some twenty years ago, the location of the disease within the stomach was of importance in respect of resectability. The surgical risk associated with operative procedures upon the proximal one third of the stomach was so great that few surgeons were willing to assume the responsibility for the attendant high operative mortality and consequently most patients with cancer in this site were considered incurable. Only over the last eight years of the period studied, were the operations of total gastrectomy and oesophago-gastrectomy considered as acceptable procedures for patients in Saskatchewan.

To a lesser extent, the type of lesion also influenced the surgeon's opinion regarding resectability. Until recent years the widely infiltrative lesions, as in linitis plastica, were considered beyond the limits of surgical extirpation.

For each sex there is no significant relationship between the age and site or age and type of the lesion within the stomach. (Tables 62, 62(i), 62(ii), 62(iii), 63 and 63(i)).

There is great prognostic significance in the presence or absence of metastatic tumour in the regional lymphatic glands. Balfour (1937) stated that when the growth and regional glands could be thoroughly extirpated thirty per cent of the patients lived five years. When there was no regional lymphatic glandular involvement, forty-eight per cent lived five years, whereas, when there was tumour present in the regional nodes, the five year survival rate was only eighteen per cent. Barclay (1951) reported similar findings in two separate groups of patients. In these, the five year survival rates, when the regional nodes were involved, were 8.6 per cent and 11.3 per cent, but were thirty-six per cent and fifty-one per cent when the lymphatic glands were not involved. These findings have been confirmed by others - (ReMine et al 1953). Table 64 presents the data concerning the influence of lymphatic glandular involvement in the Saskatchewan series. Of the cases in which information regarding the nodal status was available, 20.4 per cent had no glandular metastases. Of the 869 patients (79.6 per cent) in whom glandular metastases were

Site of Tumour	Total Cases	Males - Age					
		All Ages	0 - 44	45-54	55-64	65-69	70 & over
Total	2000	1493	88	196	445	281	483
Unstated	88	70	4	3	20	13	30
Prepylorus	662	480	30	63	140	85	162
Lesser Curvature	404	305	18	45	95	66	81
Antrum	210	163	7	26	40	31	59
Cardia	184	138	6	14	50	24	44
Greater Curvature & fundus	159	116	5	17	34	24	36
Body	139	107	6	10	36	17	38
Whole Stomach	107	75	9	12	19	15	20
Posterior Wall	30	24	1	4	5	5	9
Anterior Wall	13	11	2	2	3	1	3
Multiple Sites	4	4	-	-	3	-	1

GASTRIC CANCER: THE RELATIONSHIP BETWEEN SITE OF TUMOUR AND AGE AND SEX (MALE)

SASKATCHEWAN 1932-55

TABLE 62

Site of Tumour	Total Cases	Male					
		All Ages	Age 0 - 44	45-54	55-64	65-69	70 & over
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Unstated	4.4	4.7	4.5	1.5	4.5	4.7	6.2
Prepylorus	33.1	32.1	34.1	32.2	31.5	30.3	33.5
Lesser Curvature	20.2	20.3	20.5	22.9	21.3	23.5	16.8
Antrum	10.5	10.8	8.0	13.2	9.0	11.1	12.2
Cardia	9.2	9.5	6.8	7.2	11.2	8.6	9.2
Greater Curvature & fundus	7.9	7.8	5.6	8.7	7.6	8.6	7.4
Body	6.9	7.2	6.8	5.2	8.1	6.1	8.0
Whole Stomach	5.4	5.0	10.2	6.1	4.3	5.4	4.1
Posterior Wall	1.5	1.6	1.2	2.0	1.1	1.4	1.8
Anterior Wall	0.7	0.8	2.3	1.0	0.7	0.3	0.6
Multiple Sites	0.2	0.2	-	-	0.7	-	0.2

GASTRIC CANCER: THE RELATIONSHIP BETWEEN SITE OF THE TUMOUR WITHIN THE STOMACH AND AGE AND SEX (MALE)

SASKATCHEWAN 1932-55

TABLE 62 (i)

Site of Tumour	Total Cases	Female					
		All Ages	Age 0-44	45-54	55-64	56-69	70 and over
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Unstated	4.4	3.6	2.1	4.6	0	4.0	6.9
Prepylorus	33.1	35.8	31.9	41.9	34.1	34.7	37.5
Lesser Curvature	20.2	19.5	17.0	17.7	23.3	20.0	16.9
Antrum	10.5	9.3	12.7	7.3	10.6	6.7	8.7
Cardia	9.2	9.1	6.5	10.5	8.8	8.0	10.0
Greater Curvature & fundus	7.9	8.5	8.6	6.0	7.5	12.0	8.8
Body	6.9	6.3	2.1	7.3	7.0	8.0	5.6
Whole Stomach	5.4	6.3	14.9	3.1	8.1	5.3	3.8
Posterior Wall	1.5	1.2	2.1	1.6	0.6	1.3	1.2
Anterior Wall	.7	0.4	2.1	-	-	-	0.6
Multiple Sites	0.2	-	-	-	-	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN SITE OF THE TUMOUR WITHIN THE STOMACH AND AGE AND SEX (FEMALE)

SASKATCHEWAN 1932-55

TABLE 62 (iii)

Type of Tumour	Total Cases	Age - Male					
		All Ages	0 - 44	45-54	55-64	65-69	70 and over
Total	2000	1493	88	196	445	281	483
Unstated	1010	747	35	86	211	151	264
Ulcerating	566	428	36	70	143	65	114
Infiltrating	233	180	9	22	52	42	55
Polypoid	140	105	2	9	32	18	44
Linitis	37	23	6	5	4	4	4
Cancer and Ulcer	14	10	-	4	3	1	2
Type of Tumour	Total Cases	Age - Female					
		All Ages	0 - 44	45-54	55-64	65-69	70 and over
Total	2000	507	47	67	158	75	160
Unstated	1010	263	23	36	79	41	84
Ulcerating	566	138	14	19	52	16	37
Infiltrating	233	53	6	8	12	9	18
Polypoid	140	35	-	3	10	6	16
Linitis	37	14	4	-	5	1	4
Cancer and Ulcer	14	4	-	1	-	2	1

GASTRIC CANCER: THE RELATIONSHIP BETWEEN TYPE OF TUMOUR AND AGE AND SEX

SASKATCHEWAN 1932-55

TABLE 63

Type of Tumour	Total Cases	Age - Male					
		All Ages	0 - 44	45-54	55-64	65-69	70 & over
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Unstated	50.5	50.0	39.8	43.9	47.4	53.7	54.6
Ulcerating	28.2	28.7	40.9	35.7	32.1	23.1	23.6
Infiltrating	11.7	12.1	10.2	11.3	11.7	15.0	11.4
Polypoid	7.0	7.0	2.3	4.6	7.2	6.4	9.2
Linitis	1.9	1.5	6.8	2.5	0.9	1.4	0.8
Cancer and Ulcer	0.7	0.7	-	2.0	0.7	0.4	0.4
Type of Tumour	Total Cases	Age - Female					
		All Ages	0 - 44	45-54	55-64	65-69	70 & over
Total	100.0	100.0	100.0	100.0	100.0	100.0	100.0
Unstated	50.5	51.8	48.9	53.7	50.0	54.7	52.5
Ulcerating	28.2	27.2	29.8	28.4	32.9	21.4	23.1
Infiltrating	11.7	10.5	12.8	11.9	7.6	12.0	11.3
Polypoid	7.0	6.9	-	4.5	6.3	8.0	10.0
Linitis	1.9	2.8	8.5	-	3.2	1.3	2.5
Cancer and Ulcer	0.7	0.8	-	1.5	-	2.6	.6

GASTRIC CANCER: THE RELATIONSHIP BETWEEN TYPE OF TUMOUR AND AGE AND SEX IN PER CENT

SASKATCHEWAN 1932-55

TABLE 63 (i)

Number With Involved Lymph Nodes	Distribution of Lymphatic Glands Involved (Per Cent)					
	Regional Nodes Only	Supra-clavicular	Regional and Supra-clavicular	Regional and Distant	Regional Distant and Supra-clavicular	Distant Only
869	91.0	3.0	2.8	2.1	1.0	0.1

GASTRIC CANCER: THE DISTRIBUTION OF INVOLVED LYMPHATIC GLANDS

SASKATCHEWAN 1932-55

TABLE 64

found, the regional nodes only were involved in ninety-one per cent.

ReMine et al (1953) have commented on the prognostic significance of involvement of the sub-pyloric lymph nodes. The poor five year survival rates evident when these nodes are involved is said to be due to early associated spread to secondary nodal echelons behind the head of the pancreas and in the adjacent retroperitoneal tissues. These authors also reported a relationship between the prognosis and the distance of the involved lymph nodes from the nearest part of the gastric primary neoplasm. In the group of long term survivors, none of the involved nodes was at a greater distance from the primary tumour than 3 cms. whereas in the short-term survivors at least some of the involved nodes were at a greater distance. Comparative data are not available in this series.

No obvious relationship existed between the site of the gastric tumour and the incidence of lymphatic glandular involvement. (Tables 65 and 65(i)). Polypoid lesions would appear to be less prone to spread to lymphatic glands than other types, and when spread from this type of tumour does occur, it is more likely to remain regional than in the other types of lesion. (Tables 66 and 66(i)).

Many efforts have been made to correlate histological grading of gastric tumours with prognosis. To the writer, the subdivision of tumours into grades appears to be artificial in many cases and to be at best only relative. It involves to a large extent the personal factor and is based frequently on less than

Lymph Node Pathology	All Cases	Site of Tumour									
		Un-stated	Pre-pylorus	Lesser Curv.	Antrum	Cardia	Greater Curv. &fundus	Body	Whole Stomach	Post. Wall	Ant. Wall
Total	2000	89	661	405	210	186	158	137	111*	30	13
Not Stated	908	73	266	177	91	102	68	79	42	6	4
Total Stated	1092	16	395	228	119	84	90	58	69	24	9
All Nodes Negative	223	6	88	42	31	12	21	8	5	7	3
Regional Nodes Positive	791	8	286	171	74	60	66	48	55	17	6
Supraclavicular Nodes	26	1	5	8	4	5	1	1	1	-	-
Regional plus supraclavicular nodes	24	-	7	3	3	5	2	1	3	-	-
Regional plus Distant Nodes	18	-	8	2	4	1	-	-	3	-	-
Regional plus distant plus supraclavicular nodes	9	-	1	2	3	1	-	-	2	-	-
Distant Nodes	1	1	-	-	-	-	-	-	-	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE PRIMARY GASTRIC TUMOUR AND THE INCIDENCE AND DISTRIBUTION OF THE TUMOUR INVOLVED LYMPHATIC GLANDS SASKATCHEWAN 1932-55

*Includes 4 cases in which cancer was evident at multiple sites.

TABLE 65

Lymph Node Pathology	All Cases	Unstated	Pre-pylorus	Lesser Curv.	Antrum	Cardia	Greater Curv. and Fundus	Body	Whole Stomach	Post. Wall	Ant. Wall
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
All nodes negative	20.4	37.5	22.3	18.5	26.5	14.3	23.3	13.8	7.2*	29.2	33.3
Regional nodes positive	72.4	50.0	72.4	74.7	62.1	71.3	73.4	82.8	79.7	70.8	66.7
Supraclavicular positive	2.4	6.3	1.3	3.6	3.3	6.0	1.1	1.7	1.3	-	-
Regional plus supra-clavicular positive	2.2	-	1.8	1.4	2.4	6.0	2.2	1.7	4.4	-	-
Regional plus Distant	1.7	-	2.0	0.9	3.3	1.2	-	-	4.4	-	-
Regional plus Distant plus supraclavicular	0.8	-	0.2	0.9	2.4	1.2	-	-	3.0	-	-
Distant	0.1	6.2	-	-	-	-	-	-	-	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE PRIMARY GASTRIC TUMOUR AND THE INCIDENCE AND DISTRIBUTION OF THE TUMOUR INVOLVED LYMPHATIC GLANDS (PERCENT)
SASKATCHEWAN 1932-55

*Includes 4 cases in which cancer was evident at multiple sites.

TABLE 65 (i)

Lymph Node Pathology	Type of Tumour						
	Number of Cases						
	All Cases	Unstated	Ulcerating	Infiltrating	Polypoid	Linitis	Cancer on benign ulcer
Total	2000	1012	567	230	140	37	14
Unstated	908	658	141	56	34	14	5
Total stated	1092	354	426	174	106	23	9
All Nodes Negative	223	32	114	33	38	1	5
Regional Nodes Positive	791	279	297	129	66	16	4
Supraclavicular Positive	26	22	3	1	-	-	-
Regional plus supraclav. positive	24	15	6	1	1	1	-
Regional plus distant positive	18	5	5	5	1	2	-
Regional plus distant plus supraclavicular	9	1	1	5	-	2	-
Distant Nodes	1	-	-	-	-	1	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE TYPE OF TUMOUR AND THE INCIDENCE AND DISTRIBUTION OF LYMPHATIC GLANDS INVOLVED
SASKATCHEWAN 1932-55

TABLE 66

Lymph Node Pathology	Type of Tumour						
	Per Cent of Total Cases						
	All Cases	Unstated	Ulcerating	Infiltrating	Polypoid	Linitis	Cancer on benign ulcer
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0	100.0
All Nodes Negative	20.4	9.1	26.8	18.9	35.9	4.3	55.6
Regional Nodes Positive	72.4	78.9	69.7	74.1	62.3	69.5	44.4
Supraclavicular Positive	2.4	6.3	0.7	0.6	-	-	-
Regional Plus Supraclavicular	2.2	4.3	1.4	0.6	0.9	4.3	-
Regional Plus Distant	1.7	1.1	1.2	2.9	0.9	8.8	-
Regional Plus Distant Plus Supraclavicular	0.8	0.3	0.2	2.9	-	8.8	-
Distant Nodes Positive	0.1	-	-	-	-	4.3	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE TYPE OF TUMOUR AND THE INCIDENCE AND DISTRIBUTION OF LYMPHATIC GLANDS INVOLVED (PER CENT)

SASKATCHEWAN 1932-55

TABLE 66 (i)

adequate examination of the tissue. Multiple sections of the same tumour may reveal so many different histological appearances that frequently an individual tumour could be classed in any or all of the four grades described, depending upon which particular pathological section was taken to be representative of the whole tumour.

Histological grading was not assessed routinely in the cases described in this series, but for completeness of the record, those which were so graded are correlated with lymphatic glandular involvement. (Tables 67 and 67(i)). The numbers of cases in each group are too small for comparison and so no useful conclusions can be drawn from the data presented.

The relationship between the presence of a palpable mass in the abdomen and lymphatic glandular involvement is demonstrated in tables 68 and 68(i). The presence of a palpable mass would appear to coincide with a higher incidence of lymphatic glandular metastases, both regional and remote.

The direct extension of gastric neoplasms to neighbouring organs and structures, frequently reduces the chance of complete excision of the tumour, and consequently influences the prognosis. Tables 69, 69(i), 70 and 70(i) demonstrate the incidence of direct tumour extension in the various sites and types of primary gastric lesions. Tumours in the prepyloric region of the stomach would appear to extend directly to other organs and structures less frequently than tumours in other sites. The high incidence of

Lymph node Pathology	Broder Classification					
	Number of Cases					
	All Cases	Not Stated	Grade I	Grade 2	Grade 3	Grade 4
Total	2000	1580	11	179	195	35
Unstated	908	860	3	23	18	4
Total Stated	1092	720	8	156	177	31
All nodes negative	223	129	7	41	39	7
Regional nodes positive	791	545	1	101	121	23
Supraclavicular positive	26	25	-	1	-	-
Regional and supraclavicular pos.	24	14	-	5	4	1
Regional & distant positive	18	6	-	5	7	-
Regional & distant & supraclavicular pos.	9	-	-	3	6	-
Distant	1	1	-	-	-	-

GASTRIC CANCER: THE INCIDENCE OF NODAL INVOLVEMENT IN THE VARIOUS GRADES OF TUMOUR (BRODER)

SASKATCHEWAN 1932-55

TABLE 67

	Broder Classification					
	Per cent of Total Cases					
	All Cases	Not Stated	Grade 1	Grade 2	Grade 3	Grade 4
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0
All Nodes Negative	20.4	17.9	87.5	26.4	22.0	22.6
Regional Positive	72.4	75.8	12.5	64.6	68.4	74.2
Supraclavicular Positive	2.4	3.5	-	0.6	-	-
Regional and Supraclavicular Positive	2.2	1.9	-	3.2	2.3	3.2
Regional and Distant Positive	1.7	0.8	-	3.2	3.9	-
Regional and Distantand Supraclavicular Positive	0.8	-	-	2.0	3.4	-
Distant Positive	0.1	0.1	-	-	-	-

GASTRIC CANCER: THE INCIDENCE OF NODAL INVOLVEMENT IN THE VARIOUS GRADES OF TUMOUR (BRODER) (PER CENT)

SASKATCHEWAN 1932-55

TABLE 67 (i)

Lymph Node Pathology	Nodes and Palpable Mass			
	Number of Cases			
	All Cases	Unstated re Palpable Mass	With palpable mass	Without palpable mass
Total	2000	77	544	1379
Not Stated	908	45	283	580
Total Stated	1092	32	261	799
All nodes negative	223	9	29	185
Regional nodes positive	791	23	204	564
Supraclavicular positive	26	-	10	16
Regional plus supraclavicular positive	24	-	13	11
Regional plus distant positive	18	-	3	15
Regional plus distant plus supraclavicular pos.	9	-	2	7
Distant positive	1	-	-	1

GASTRIC CANCER: THE INCIDENCE OF LYMPHATIC GLANDULAR METASTASES RELATED TO THE PRESENCE OF PALPABLE ABDOMINAL MASS

SASKATCHEWAN 1932-55

TABLE 68

Lympth Node Pathology	Nodes and Palpable Mass			
	Per cent of Total Cases			
	All Cases	Unstated re Palpable Mass	With Palpable Mass	Without Palpable Mass
Total stated	100.0	100.0	100.0	100.0
All negative	20.4	28.1	11.1	23.2
Regional nodes positive	72.4	71.9	78.2	70.6
Supraclavicular nodes positive	2.4	-	3.8	2.0
Regional plus supraclavicular positive	2.2	-	5.0	1.4
Regional plus distant positive	1.7	-	1.1	1.8
Regional plus distant plus supraclavicular pos.	0.8	-	0.8	0.9
Distant positive	0.1	-	-	0.1

GASTRIC CANCER: THE INCIDENCE OF LYMPHATIC GLANDULAR METASTASES RELATED TO THE PRESENCE OF PALPABLE ABDOMINAL MASS (PER CENT)

SASKATCHEWAN 1932 - 55

TABLE 68 (i)

Direct Extension	Site of Tumour										
	All cases	Un-stated	Pre-pylorus	Lesser Curv.	Antrum	Cardia	Greater curv. & fundus	Body	Whole* stomach	Post. Wall	Anterior Wall
Total	2000	89	661	405	210	186	158	137	111*	30	13
Not Stated	1232	74	400	257	134	124	89	89	52	7	6
Total Stated	768	15	261	148	76	62	69	48	59	23	7
None	242	4	106	48	23	11	18	12	9	8	3
Other	173	2	45	37	13	16	22	14	16	6	2
To other organs	220	9	69	34	15	33	19	14	24	3	-
Transverse mesocolon	69	-	19	11	15	1	8	6	6	3	-
Gastrohepatic ligament	54	-	20	15	8	1	1	2	2	3	2
Gastrohepatic ligament & transverse mesocolon	10	-	2	3	2	-	1	-	2	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE GASTRIC TUMOUR AND DIRECT EXTENSION TO OTHER ORGANS AND NEIGHBOURING STRUCTURES

SASKATCHEWAN 1932-55

TABLE 69

*Includes 4 cases in which cancer was evident at multiple sites.

Direct Extension	Site of Tumour										
	All Cases	Un-stated	Pre-pylorus	Lesser Curv.	Antrum	Cardia	Greater Curv. &fundus	Body	Whole Stomach	Post. Wall	Ant. Wall
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0*	100.0	100.0
None	31.5	26.7	40.6	32.5	30.3	17.7	26.2	25.0	15.3	34.9	42.8
Other	22.5	13.3	17.2	25.0	17.2	25.8	31.9	29.1	27.1	25.8	28.6
To other organs	28.7	60.0	26.4	23.0	19.7	53.3	27.5	29.1	40.6	13.1	-
Transverse mesocolon	9.0	-	7.3	7.4	19.7	1.6	11.6	12.6	10.2	13.1	-
Gastrohepatic ligament	7.0	-	7.7	10.1	10.5	1.6	1.4	4.2	3.4	13.1	28.6
Gastrohepatic ligament & transverse mesocolon	1.3	-	0.8	2.0	2.6	-	1.4	-	3.4	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE GASTRIC TUMOUR AND DIRECT EXTENSION TO OTHER ORGANS AND NEIGHBOURING STRUCTURES (PER CENT)

SASKATCHEWAN 1932-55

*Includes 4 cases in which cancer was evident at multiple sites.

TABLE 69 (i)

Direct Extension	Direct Extension and Type						
	All Cases	Unstated	Ulcerating	Infiltrating	Polypoid	Linitis	Cancer on Ulcer
Total	2000	1012	567	230	140	37	14
Not stated	1232	788	289	75	62	14	4
Total Stated	768	224	278	155	78	23	10
None	242	32	119	47	34	3	7
Other	173	73	53	24	18	4	1
To other organs	220	67	63	63	15	11	1
Transverse mesocolon	69	28	23	9	5	4	-
Gastrohepatic ligament	54	17	20	9	6	1	1
Gastrohepatic ligament and transverse mesocolon	10	7	-	3	-	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE INCIDENCE OF DIRECT EXTENSION TO NEIGHBOURING ORGANS AND STRUCTURES AND THE VARIOUS TYPES OF PRIMARY GASTRIC NEOPLASM

SASKATCHEWAN 1932-55

TABLE 70

Direct Extension	Per cent of Cases Stated Re Direct Extra-Gastric Extension of Tumour						
	All Cases	Unstated	Ulcerating	Infiltrating	Polypoid	Linitis	Cancer on Ulcer
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0	100.0
None	31.5	14.3	42.8	30.3	43.6	13.0	70.0
Other	22.5	32.6	19.1	15.5	23.1	17.4	10.0
To other organs	28.7	29.9	22.7	40.5	19.2	47.8	10.0
Transverse mesocolon	9.0	12.5	8.2	5.9	6.4	17.4	-
Gastrohepatic ligament	7.0	7.6	7.2	5.9	7.7	4.4	10.0
Gastrohepatic ligament and transverse mesocolon	1.3	3.1	-	1.9	-	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE INCIDENCE OF DIRECT EXTENSION TO NEIGHBOURING ORGANS AND STRUCTURES AND THE VARIOUS TYPES OF PRIMARY GASTRIC NEOPLASM (PER CENT)

SASKATCHEWAN 1932-55

TABLE 70 (i)

direct extension to other organs by tumours in the cardia may be explained by the frequency with which these tumours extend intramurally into the oesophagus. Infiltrating tumours extend directly more frequently than ulcerative or polypoid types of lesions. The latter two types are comparable in this respect. The relationship between Broder's classification of the primary tumour and the incidence of direct extension indicates that the lower the pathological grade, the lower the incidence of direct extension, but a definite conclusion cannot be made from the inadequate data presented (Tables 71 and 71 (i)). The incidence of direct spread of tumour in patients who do not have a palpable mass is slightly smaller than in those in whom a palpable mass is found (Table 72 and 72 (i)).

DUODENAL AND OESOPHAGEAL INVOLVEMENT

Duodenal and oesophageal involvement by direct extension of tumour from the adjacent parts of the stomach, have been reported to occur with significant frequency. Coller et al (1941), Borrmann (1926), Konjetzny (1950), Zinninger et al (1949), Harvey et al (1951), reported direct spread into the duodenum in twenty-five per cent, twenty-nine per cent, sixty-seven per cent, thirty-three per cent and twenty-five per cent respectively. Oesophageal involvement was reported to occur in forty-eight per cent, sixty-four per cent and thirty-two per cent in the series of cases described by Ranson (1947), Canonico (1950) and Eker (1951).

Direct Extension	All cases	GRADE OF TUMOUR (BRODER)				
		Not Stated	Grade I	Grade II	Grade III	Grade IV
Total	2000	1566	11	182	206	35
Unstated	1232	1078	4	67	70	13
Total Stated	768	488	7	115	136	22
None	242	132	7	51	48	4
Other	173	129	-	20	17	7
To other organs	220	134	-	33	47	6
Transverse Mesocolon	69	52	-	-	14	2
Gastrohepatic ligament	54	32	-	11	9	2
Gastrohepatic ligament and transverse mesocolon	10	8	-	-	1	1

GASTRIC CANCER: THE RELATIONSHIP BETWEEN BRODER CLASSIFICATION OF TUMOUR AND DIRECT EXTENSION

SASKATCHEWAN 1932-55

TABLE 71

Direct Extension	All Cases	GRADE OF TUMOUR (BRODER)				
		Not Stated	Grade I	Grade II	Grade III	Grade IV
Total Stated	100.0	100.0	100.0	100.0	100.0	100.0
None	31.5	27.1	100.0	44.3	35.3	18.2
Other	22.5	26.4	-	17.4	12.5	31.8
To other organs	28.7	27.5	-	28.7	34.6	27.3
Transverse mesocolon	9.0	10.7	-	-	10.3	9.1
Gastrohepatic ligament	7.0	6.6	-	9.6	6.6	9.1
Gastrohepatic ligament and transverse mesocolon	1.3	1.7	-	-	0.7	4.5

GASTRIC CANCER: THE RELATIONSHIP BETWEEN BRODER CLASSIFICATION OF TUMOUR AND DIRECT EXTENSION (PER CENT)

SASKATCHEWAN 1932-55

TABLE 71 (i)

Direct Extension	Palpable Mass and Extension of Tumour			
	Number of Cases			
	All Cases	Unstated as to Palpable Mass	With Palpable Mass	Without Palpable Mass
Total	2000	78	544	1378
Not stated	1232	58	353	821
Total Stated	768	20	191	557
None	242	6	51	185
Other	173	6	46	121
To other organs	220	6	54	160
Transverse mesocolon	69	2	18	49
Gastrohepatic omentum	54	-	17	37
Gastrohepatic omentum and transverse mesocolon	10	-	5	5

GASTRIC CANCER: THE INCIDENCE OF DIRECT EXTENSION OF TUMOUR IN THOSE PATIENTS WHO EXHIBITED A PALPABLE MASS

SASKATCHEWAN 1932-55

TABLE 72

Direct Extension	Palpable Mass and Extension of Tumour			
	Per Cent			
	All Cases	Unstated as to Palpable Mass	With Palpable Mass	Without Palpable
Total Stated	100.0	100.0	100.0	100.0
None	31.5	30.0	26.4	33.2
Other	22.5	30.0	24.2	21.7
To other organs	28.7	30.0	28.4	28.8
Transverse Mesocolon	9.0	10.0	9.5	8.8
Gastrohepatic omentum	7.0	-	8.9	6.6
Gastrohepatic omentum and transverse mesocolon	1.3	-	2.6	.9

GASTRIC CANCER: THE INCIDENCE OF DIRECT EXTENSION OF TUMOUR IN THOSE PATIENTS WHO EXHIBITED A PALPABLE MASS (PER CENT)
SASKATCHEWAN 1932-55
TABLE 72 (i)

This type of spread is not always obvious on gross inspection or palpation and a true record of its actual incidence can be made only when the pathologist examines the lines of resection in all surgical specimens and the duodenum and oesophagus in all relevant autopsy material. There is no such complete record of the cases in the Saskatchewan series. In one clinic, the reported incidence is much lower than that of the other, but this is explained by the inadequate recording of data by the pathologists who were associated with that clinic (Table 73). Despite the absence of critical examination of the pathological material in many cases, the incidence of duodenal and oesophageal involvement is seen to be high. It will be observed from Table 74 that there are few sites in the stomach from which tumour may not spread directly into the duodenum. In this series the oesophagus is involved only in those cases in which the primary tumour is situated in the proximal part of the stomach.

Of the two thousand cases in the series, information on metastatic spread to tissues and structures other than lymph glands is available only in 993 cases (Table 75). Almost three-fifths of the patients had metastases. The distribution of these metastases was such that the patients in the group were beyond surgical salvage.

It is possible to consider the relationship between the site of the tumour and the distribution of metastases only in patients with prepyloric and lesser curvature lesions, because the numbers of patients with lesions in other sites on which data are

Clinic	Year	Total Cases	Number of Cases with Stated Data on Direct Duodenal and/or Oesophageal Extension of Tumour	Extension to Oesophagus		Extension to Duodenum		Extension to Oesoph. and Duodenum	
				Number	Per cent	Number	Per cent	Number	Per cent
I	1954	65	44	4	9.1	2	4.5	-	-
	1955	66	46	3	6.5	5	10.9	-	-
II	1954	70	50	9	18.0	9	18.0	-	-
	1955	83	54	9	16.7	10	18.5	1	1.8
III	1954	135	94	13	13.8	11	11.7	-	-
	1955	149	100	12	12.0	15	15.0	1	1.0

GASTRIC CANCER: THE INCIDENCE OF DIRECT TUMOUR SPREAD FROM THE STOMACH INTO THE DUODENUM AND OESOPHAGUS

SASKATCHEWAN 1954 and 1955

TABLE 73

Extension of Tumour	Incidence of Involvement (Per cent), by Site of Primary Tumour						
	Prepylorus	Lesser Curvature	Greater Curvature and fundus	Antrum	Body	Cardia	Whole Stomach
Duodenum	22.0	6.3	3.4	10.0	3.1	3.0	-
Oesophagus	-	8.3	6.9	-	6.2	36.4	27.7
Duodenum and Oesophagus	1.2	-	-	-	-	-	-

GASTRIC CANCER: THE DIRECT EXTENSION OF TUMOUR FROM THE VARIOUS SITES IN THE STOMACH INTO THE DUODENUM AND OESOPHAGUS

SASKATCHEWAN 1954 and 1955

TABLE 74

Total Stated Cases	Metastases (Per cent Distribution in Each Site)							
	None	Liver	Peritoneum	Rectal Shelf	Pancreas	Lungs	Multiple	Other
993	42.2	24.8	6.3	1.4	1.4	0.6	21.2	2.1

GASTRIC CANCER: THE INCIDENCE OF EXTRA-NODAL METASTASES IN 993 CASES OF CANCER OF THE STOMACH

SASKATCHEWAN 1932-55

TABLE 75

available are too small for comparison. In 331 prepyloric and 202 lesser curvature lesions, it is interesting to observe the similarity in the incidence of metastases in various other organs (Table 76). From both sites the incidence of metastases to liver, rectal shelf, pancreas, lungs and to multiple organs is similar. Prepyloric lesions appear to give rise to peritoneal seeding to a significantly greater degree than lesions of the lesser curvature. However, in prepyloric tumours, the overall tendency to metastasise is less than lesser curvature lesions. When the site of the primary tumour within the stomach is considered in terms of the metastases which are found in the liver, it is seen (Table 77) that lesser curvature tumours are more prone to give rise to hepatic metastases.

In the study of the site of extra lymphatic glandular metastases in relation to the type of tumour, again only two groups can be compared. Ulcerating tumours appear to metastasise to other organs less readily than infiltrating tumours (Table 78) but when the site of the metastases is considered in terms of the type of the tumour, it would appear that a greater proportion of secondary deposits in liver, peritoneum, pancreas, lungs and in multiple sites, arise from ulcerative lesions than from infiltrative lesions (Table 79).

Site of Tumour	No metastases	Liver	Peritoneum	Rectal Shelf	Pancreas	Lungs	Multiple	Other
Unstated	1.2	3.2	8.1	14.3	-	-	4.3	4.8
Prepylorus	42.9	29.9	27.5	14.3	28.6	16.7	24.8	4.8
Antrum	8.8	10.1	8.1	7.1	-	-	10.9	9.5
Lesser Curvature	18.1	25.9	14.4	14.3	28.6	16.7	20.5	14.3
Greater curvature and fundus	8.8	7.7	8.1	14.3	-	33.2	10.9	19.0
Anterior wall	1.2	0.4	1.6	-	-	-	0.9	-
Posterior wall	2.9	2.0	1.6	-	-	-	0.9	4.8
Body	5.8	7.7	8.1	14.3	14.3	16.7	5.7	19.0
Cardia	6.2	7.4	8.1	14.3	21.4	16.7	8.7	9.5
Whole stomach	3.3	5.7	14.4	7.1	7.1	-	12.4	14.3
Multiple sites	0.8	-	-	-	-	-	-	-

GASTRIC CANCER: THE INCIDENCE OF EXTRA LYMPHATIC GLANDULAR METASTASES BY SITE OF TUMOUR (PER CENT)

SASKATCHEWAN 1932-55

TABLE 76

Site of Metastases	Un-stated	Pre-pylorus	Antrum	Lesser Curv.	Greater curv.& fundus	Anterior Wall	Post. Wall	Body	Cardia	Whole stomach	Multiple Sites
None	16.7	54.4	39.8	37.6	40.2	55.6	57.1	34.9	34.7	20.6	100.0
Liver	26.6	22.4	26.8	31.6	20.6	11.1	23.8	27.6	24.0	20.6	-
Peritoneum	16.7	5.1	5.4	4.5	5.4	11.1	4.8	7.2	6.7	13.2	-
Rectal Shelf	6.7	0.6	1.1	1.0	2.2	-	-	2.9	2.7	1.5	-
Pancreas	-	1.2	-	2.0	-	-	-	2.9	4.0	1.5	-
Lungs	-	0.3	-	0.5	2.2	-	-	1.4	1.3	-	-
Multiple	30.0	15.7	24.7	21.3	25.0	22.2	9.5	17.4	24.0	38.2	-
Other	3.3	0.3	2.2	1.5	4.4	-	4.8	5.7	2.6	4.4	-

GASTRIC CANCER: THE DISTRIBUTION OF EXTRA LYMPHATIC GLANDULAR METASTASES
(PER CENT)
SASKATCHEWAN 1932-55

TABLE 77

Site of Tumour	No metastases	Liver	Peritoneum	Rectal Shelf	Pancreas	Lungs	Multiple	Other
Unstated	13.8	59.2	62.9	92.8	50.0	33.3	46.7	57.1
Polypoid	12.4	8.6	1.6	-	7.2	-	7.1	-
Ulcerating	46.6	23.5	22.6	-	28.4	50.0	24.3	14.3
Infiltrating	24.4	7.0	6.5	7.2	7.2	16.7	16.2	23.8
Linitis	0.7	1.7	4.8	-	7.2	-	5.7	4.8
Cancer or Ulcer	2.1	-	1.6	-	-	-	-	-

GASTRIC CANCER: THE INCIDENCE OF EXTRA LYMPHATIC GLANDULAR METASTASES BY TYPE (PER CENT)

SASKATCHEWAN 1932-55

TABLE 78

Site of Metastases	Unstated	Polypoid	Ulcerating	Infiltrating	Linitis	Cancer o. ulcer
None	15.4	57.8	59.5	61.9	12.5	90.0
Liver	39.0	23.3	17.7	10.3	16.8	10.0
Peritoneum	10.4	1.1	4.2	2.4	12.5	-
Rectal Shelf	3.5	-	-	0.6	-	-
Pancreas	1.9	1.1	1.2	0.6	4.1	-
Lung	0.5	-	0.9	0.6	-	-
Multiple	26.1	16.7	15.6	20.6	50.0	-
Other	3.2	-	0.9	3.0	4.1	-

GASTRIC CANCER: THE DISTRIBUTION OF EXTRA LYMPHATIC GLANDULAR METASTASES BY TYPE (PER CENT)

SASKATCHEWAN 1932-55

TABLE 79

CHAPTER 7

THE LYMPHATIC DRAINAGE OF THE STOMACH

Without an intimate knowledge of the lymphatic drainage of the stomach, studied and intelligent surgical management of gastric carcinoma is not possible. Fohmann (1833) was the first to investigate the lymphatic anatomy of the organ, and in later years his work was confirmed and new information added by Sappey (1874), Teichmann (1861), Loven (1873), Cuneo (1900), Disse (1911), and Jamieson and Dobson (1907). These workers traced the lymphatic drainage routes and networks in such a way that confirmatory investigations by Rouviere (1938) added little to the already existing knowledge of the subject. The following review is based on the data presented by Rouviere.

Within the stomach wall there is a continuous lymphatic pool composed of four intramural networks each of which

communicates with the others. The first of these, the mucous network, arises in the sheaths which surround the vascular capillaries and small veins (Disse, 1911). These tiny lymph vessels drain into the interglandular spaces, described by Loven (1873), form interglandular sinuses which reach into the deep layers of the mucosa and anastomose with each other to form a lymphatic network around the glands (Cuneo and Delamare 1900). They terminate in the mucous or subglandular lymphatic network which resides between the gastric glands and muscularis mucosae.

From this mucous network lymphatic channels arise which traverse the muscularis mucosae and end in the submucous network which is separated from the mucous network only by the muscularis mucosae. Its capillaries are larger than those of the mucous network and give origin to efferent lymph vessels which reach the muscular layers of the stomach and intermingle with the lymph capillaries of the muscular network. The latter is subdivided into several secondary lymphatic plexuses which are distributed between the several layers which compose the muscular coat (Cuneo and Delamare).

The muscular network drains into the subserous or subperitoneal plexus. This network is continuous over the entire stomach, but the inter-communicating lymphatic capillaries are not evenly distributed. The plexus is much denser over the mid-portion of the stomach than at the curvatures, the pylorus and cardia (Cuneo and Delamare 1900).

There is no longer doubt that the gastric lymphatic plexuses communicate with those of the oesophagus and duodenum. These communications have been demonstrated by Stephanis (1902), Jamieson and Dobson (1907), Comolli (1911) and Rouviere (1938). The evidence provided by these workers more than balances that of the opposing school of thought exemplified by Most (1927), Polya and Navratil (1903) and Poirier and Cuneo (1902). There is ample proof from clinical and surgical experience of the spread of gastric cancer to the duodenum and the oesophagus to demonstrate that a free communication does exist between the subserous lymphatic vessels of the stomach and both the duodenal subserous lymphatics and the paraoesophageal lymphatic vessels.

There are three glandular chains which compose the first echelon of perigastric glands and into which the collecting lymph trunks for the stomach wall drain. These glandular chains are the left gastric, the splenic and the hepatic chains and each appears to be the depot of drainage for the greater part of the lymph from certain regions of the stomach wall.

THE REGION OF THE LEFT GASTRIC CHAIN

This is the most extensive area of drainage and includes the medial two thirds of the vertical portion of the stomach. It is limited to the right by the lesser curvature. Its boundary to the left begins at or to the left of the fundus and extends parallel to the curvatures but nearer the greater than

the lesser. The left gastric region may overlap the middle portion of the horizontal segment of the stomach between the right gastric lymphatic region above and the right gastro-epiploic below.

The collecting lymphatic trunks from the subserous network of the left gastric region are directed to the lymph nodes which are usually grouped near the upper part of the lesser curvature. Those draining the horizontal segment of the stomach are directed obliquely upwards and to the left, those from the body of the stomach run vertically from the distal portion, obliquely upwards and to the right in the mid portion and almost horizontally from the proximal part. Those which drain the fundus are directed downwards and to the right. The collecting trunks from the anterior and superior wall of the fundus, and those from that part of the body of the stomach opposite the cardia, are usually interrupted by the left and anterior paracardial glands. There are some trunks, however, which by-pass these nodes and drain directly into the glands of the lesser curvature or into the lymph nodes situated in the left gastro-pancreatic fold. The posterior paracardial glands are frequently absent, and in this case the posterior collecting trunks from the proximal portion of the stomach drain directly into the paracardial glands of the lesser curvature or into the nodes of the left gastro-pancreatic fold. The parietal gastric lymph nodes of Letulle (1899), may be found along the course of the left gastric collecting vessels.

Three groups of glands compose the left gastric chain, the group of the left gastro-pancreatic fold, the nodes of the lesser curvature of the stomach and the parietal group to which are appended the nodes of the pars cardiaca (juxta-cardial nodes).

(a) The nodes of the left gastro-pancreatic fold. There may be one, two or three of these glands situated along the course of the left gastric artery between its origin and the lesser curvature, above and posterior to the vessel and lying in the substance of the left gastro-pancreatic fold. In the course of gastric resection for carcinoma the most constant of the glands, lying immediately behind the origin of the artery, should be searched for diligently. It may be hidden by the left gastro-hepatic fold. The highest and most lateral of these glands is frequently intimately related to the glands of the pars cardiaca and with those of the lesser curvature.

(b) The glands of the lesser curvature. These follow the paths of the left gastric artery, after it reaches the gastric wall, and of its terminal branches. They are situated in the upper part of the lesser curvature and rarely extend beyond the mid-point between the cardia and pylorus (Jamieson and Dobson 1907). They are closely related to the gastric wall, the left gastric blood vessels and the divisions of the left vagus nerve on the lesser curvature. There are usually from three to five nodes of which approximately half accompany the trunk of the left gastric artery and the remainder its terminal branches. One gland is usually to

be found lying in front of the anterior branch of the vessel, one behind its posterior branch and a third at the level of the division of the parent trunk.

(c) The glands of the pars cardiaca. This group of glands, first discussed by Letulle (1899), may be divided into three sub-groups, the anterior, left and posterior. The anterior paracardial glands are constant, the left group is often absent but may be composed of as many as seven glands [Jamieson and Dobson (1907) and Comolli (1911)] and the posterior group are inconstant in number and may be absent. When the anterior, left and posterior paracardial nodes are present they form, with the glands of the left gastro-pancreatic fold and those in the proximal part of the lesser curvature, a pericardial lymphoid ring.

The efferent lymph trunks from the nodes on the lesser curvature and the paracardial group drain into the left gastro-pancreatic fold. All of these glands are connected by numerous lymph vessels which run in the left gastro-pancreatic fold, some in front of and others behind and below the left gastric artery. The efferent trunks of the most distal node of the left gastric chain drain into the nodes of the hepatic and splenic chains and those around the coeliac axis. This lowermost node also gives rise to lymph trunks which descend to the left in front of the aorta and then cross the left aspect of the superior mesenteric artery at its origin from above downwards and terminate in the intestinal lymph trunk and in the nodes of the paraaortic group nearest the

left renal pedicle. The efferent trunks from the latter nodes terminate in the lumbar lymph trunk.

THE REGION OF THE SPLENIC CHAIN

This region lies in that part of the stomach lateral to the left gastric region and extends from the summit of the fundus to the mid point of the greater curvature. Most of the collecting trunks which drain this region are directed towards the splenic hilum by following the left gastro-epiploic artery and the vasa brevia and end in the lymph glands of the splenic chain situated at the splenic hilum, on the tail of the pancreas and in the phrenolienal ligament. A few collecting lymph vessels which drain the left extremity of the fundus and adjacent posterior wall of the stomach drain into nodes situated adjacent to the lateral part of the body of the pancreas. Those lymphatics which accompany the left gastro-epiploic artery, reach the gastric wall at the greater curvature, then follow the vessel upwards and to the left. On the path of these lymph collecting vessels and along the greater curvature as well as in the gastro-splenic interval the left gastro-epiploic lymph nodes are found.

There are usually three or four glands in the splenic chain and these lie on the course of the splenic artery along the upper border of the pancreas. The chain is continued in the phrenolienal ligament to the hilum of the spleen. From this point it may be continued in front of the hilum and along the left gastro-epiploic artery. There is some variation in the disposition of the glands in the latter part of the chain. Sometimes there is only one node present near the origin of the left gastro-epiploic artery where

it is in close approximation to the nodes at the hilum of the spleen. Alternatively there may be two or three nodes placed either on the left gastro-epiploic artery in the gastro-splenic ligament or at the origin of one of its gastric branches. Another variation is encountered in that there may be a series of as many as eight small glands present along the left gastro-epiploic artery from the spleen to the greater curvature of the stomach and the adjacent portion of the greater omentum

Several small infrapancreatic glands may be present distal to the tail of the pancreas and the lateral part of the body of the pancreas. All of the infrapancreatic glands do not belong to the splenic chain. The most medial glands to be found near the duodeojejunal angle belong to the group of superior mesenteric nodes and are situated along the course of the inferior pancreaticoduodenal artery and on the terminal portion of the inferior mesenteric vein.

The splenic chain terminates in a large node situated immediately to the left of the coeliac artery, behind and above the first part of the splenic artery. This node gives rise to large efferent lymphatic trunks which descend and terminate in the para-aortic nodes near the left renal pedicle and in the intestinal lymph trunk. The former nodes terminate in the lumbar lymph trunk.

THE REGION OF THE HEPATIC CHAIN

This region of the stomach is situated to the right of the left gastric and splenic regions. It is subdivided into a superior, or pyloric, and an inferior zone.

The superior zone gives origin to two or three collecting vessels. At least one of these is directed upwards and to the right, crosses the superior border of the stomach and accompanies the right gastric artery to a point some distance to the right of the pylorus. It then turns backwards and downwards and ends in a large lymph gland situated to the right of the horizontal segment of the hepatic artery. The supra-pyloric lymph node may be found on the path of this collecting vessel. An efferent vessel from this node was found by Polya and Navratil (1903) to drain directly into the porta hepatis. Another collecting trunk has been found which leaves the suprapyloric gland, crosses the superior surface of the pyloric groove and continues to the right passing in front of the hepatic pedicle to a point at the superior genu of the duodenum. This vessel is then directed downwards to end in the superior retro-pancreatico-duodenal gland.

The inferior hepatic zone is drained by the right gastro-epiploic and by the infrapyloric glands. The collecting vessels are directed downwards and reach the right gastro-epiploic arterial arch which they accompany to the pylorus. Some trunks empty into one of the right gastro-epiploic nodes immediately after reaching the artery. Others terminate in a lymph node near the pylorus and sometimes in an infrapyloric node. Some of the efferent trunks from the gastro-epiploic nodes leave the course of the artery and accompany the vein and terminate in the superior mesenteric lymph node.

There are five main groups of lymph glands composing the hepatic chain, namely, the hepatic, the gastroduodenal, the right gastroepiploic, the right gastric and the pancreatico-duodenal groups.

(a) The hepatic group. This consists of four to eight glands situated along the course of the hepatic artery. Some accompany the horizontal part of the vessel while others are found along its ascending portion in the gastrohepatic omentum. One very large gland is frequently found above and to the left of the hepatic artery in the angle formed by the hepatic and splenic vessels as they leave the coeliac axis. In this position it lies posterior to the left gastric artery. There are usually two or three glands in relation to the ascending limb of the hepatic artery. They may lie to the right or left or posterior to the vessel. There is usually at least one gland to be found in the interval between the hepatic artery and the common bile duct. Others may be related to the neck of the gall bladder and in relation to the cystic artery.

(b) The gastroduodenal group. This group of glands follows the course of the gastro-duodenal artery and lies between the duodenopyloric wall and the anterior surface of the pancreas.

(c) The right gastro-epiploic group. Jamieson and Dobson (1907) subdivided this group into the infraduodenopyloric and right gastro-epiploic groups. The infraduodenopyloric group lies adjacent to the first part of the duodenum and pylorus at the

level of the bifurcation of the gastroduodenal artery. Two or three glands are usually present and they lie in close approximation to the pyloroduodenal wall. The right gastro-epiploic group is a continuation of the infraduodenopyloric group and differs from it only by its location distal to the greater curvature of the stomach. The nodes are situated along the course of the right gastro-epiploic artery and therefore lie at some distance from the greater curvature.

(d) The right gastric group. This group of glands is inconstant. Jamieson and Dobson (1907) identified one fairly constant node which was situated on the superior surface of the pylorus but which frequently lay at some distance from the pylorus in the gastrohepatic omentum.

(e) The pancreaticoduodenal group. There are two subgroups composing the pancreaticoduodenal glands. These are anterior or prepancreaticoduodenal and posterior or retropancreaticoduodenal in site. The prepancreatic duodenal nodes are usually located near the pancreatic border of the duodenal loop, particularly near the first and the upper half of the second parts of the duodenum, but may be found at any point on the anterior surface of the head of the pancreas. In their usual position these glands are in contact with the superior pancreaticoduodenal artery. One of the glands has an almost constant situation near the proximal duodenal flexure and the others lie above, to the right and distal to the mesenteric attachments. Bartels (1907) has pointed out that while, in health, only three to six glands may be seen lying in front of

the pancreas on gross examination, many more may be evident on microscopic examination or when the glands are involved in a pathological process.

The posterior pancreaticoduodenal nodes are situated behind the head of the pancreas, along the common bile duct or on the arterial arcades formed by the pancreaticoduodenal artery. There are usually two to four glands in this group. One of them, the retropancreaticoduodenal node, is constant and is situated behind the angle formed by the first and second parts of the duodenum and to the right of the portal vein.

The anterior pancreaticoduodenal nodes are united with each other by anastomotic lymph vessels and drain into the infra-pyloric nodes. The efferent lymphatic vessels of the posterior gastroduodenal glands drain into those nodes which are situated to the right and distal to the origin of the superior mesenteric artery. They may also drain into the glands of the horizontal portion of the hepatic artery. The larger gland of the horizontal hepatic chain lying adjacent to the coeliac artery is joined by efferent trunks from the left gastric and splenic nodes. Two or three efferent trunks descend from it behind the pancreas to terminate, like those of the splenic and left gastric chains in the para-aortic nodes near the left renal pedicle and in the intestinal lymph trunk. The efferent trunks from the former nodes terminate in the lumbar lymph trunk.

While the lymphatic pathways from the stomach are complex, there are two outstanding features evident which are of the greatest importance in relation to the potential intragastric lymphatic spread from gastric cancer. The first of these is the continuity of the intramural lymphatic pool. The mucous, the submucous, the muscular and the subserous plexuses are in continuity within themselves and with each other. The subserous network drains all the lymph from the gastric wall and is continuous over the entire stomach. The intramural lymphatic vessels are avalvular and consequently, although there appears to be a predisposition for the lymph flow from certain areas of the stomach to be directed along definite lymph channels towards certain groups of glands in health, the same uniformity of lymph flow may not be evident in diseased processes in the stomach. When the stomach is involved in a carcinomatous process the search for involvement of glands outside the group in which secondary involvement is expected is therefore mandatory and the importance of removing the whole of the potential lymphatic spread of the disease is emphasised.

The second feature of importance is the convergence of the lymph flow from the glands draining all parts of the stomach to a small group of nodes concentrated in the region of the coeliac axis and especially to one large gland, situated above and to the left of the hepatic artery in the angle formed by the latter vessel and the splenic artery as they leave the coeliac axis. The potential

spread of the tumour to certain glands of the second echelon such as the superior retropancreaticoduodenal, the superior mesenteric nodes and to the nodes in the porta-hepatis creates a problem when adequate surgical therapy for a patient with gastric cancer is under consideration.

CHAPTER 8

THE TREATMENT OF GASTRIC CANCER

"The surgeon has no right to take the patient's life but he is otherwise under no obligation to employ fantastic measures to prolong it in the face of intolerable and clearly terminal disease."

GENERAL CONSIDERATIONS

A physician or surgeon who devotes his entire professional life to cancer therapy requires to be an optimist by nature, else he would forsake his calling and enter a more rewarding field. His efforts to effect cures in some of the most lethal cancers such as may arise in the pancreas, lung and stomach, have been so ineffectual in the past that his continued striving for greater success is a mark of the victory of his optimism over his experience.

Surgery is the only therapeutic measure which, with our present knowledge, can offer the patient with cancer of the stomach a chance of cure, but experience has proved

that even with the adoption of the most radical surgery, the ultimate salvage of life is small. The increasing awareness of the inadequacy of surgery in all but a few cases has led to the repeated examination of the problem of treatment and the attempt to design an operative procedure which would satisfy the demands of the generally accepted principles of cancer surgery.

The object of an adequate cancer operation has been described as a generous en bloc excision of all tumour tissue and the potential lymphatic spread from the involved organ or organs without causing a significant permanent disturbance of the patient's physiology or feeling of well-being and with little or no risk of life.

To comply with the anatomical principles of this definition the mandatory operation in gastric cancer would be one of total gastrectomy with division of the oesophagus high in the chest, pancreaticoduodenectomy with splenectomy, the removal of the gastrohepatic and greater omenta in their entirety, the removal of a generous margin of the soft tissue around these organs to provide a means of encompassing the lymphatic channels as well as the lymphatic glands in the immediate perigastric and paraoesophageal regions and those adjacent to the pancreas and duodenum. It would be essential to dissect meticulously the lymphatic tissue around the coeliac axis, hepatic artery, common bile duct and the vascular pedicle of the liver. Similar dissection would be required around the cystic artery and duct, along the

inferior vena cava especially that part lying posterior to the third portion of the duodenum, around the origin of the superior mesenteric artery and in the para-aortic region. Any other organ involved by direct spread would require to be removed in addition, together with the potential lymphatic drainage of that organ.

From the purely theoretical point of view, the general adoption of this operative procedure as routine treatment should improve the salvage rate of carcinoma of the stomach. From the practical standpoint, however, it is probable that any advantage associated with this relegation of therapy to a mere anatomical exercise would be outweighed by the disadvantages of a high rate of operative mortality and postoperative morbidity. It is obvious that a patient, deprived of his entire stomach, duodenum and pancreas would suffer permanent disturbance of his digestive physiology with resultant secondary nutritional and metabolic impairment.

It is apparent, then, that in the treatment of gastric cancer, it is not possible to satisfy all of the demands of the theoretically ideal cancer operation. Consequently, it is important to recognise that any deviation from the principles of cancer surgery which may be found essential to maintain a reasonable quality of postoperative life for the patient, necessarily alters the basic intention of treatment from that of cure to one of making a compromise with the disease.

Compromise and conservatism are not necessarily synonymous. Compromise admits that there is a limit of perigastric tumour spread beyond which surgery cannot be used without causing permanent and gross physiological disturbance, but recognises that, within certain limits of spread, surgery can be successfully employed to extirpate the tumour tissue completely. Conservatism, frequently the refuge of the discouraged and the inept, implies a lack of confidence by the surgeon in the efficacy of his art to cure the disease. Palliation is its objective and an improvement, however temporary, in the quality rather than the quantity of the patient's life its primary aim. This philosophy of pessimism is often the result of previous and repeated failure to extirpate the disease and to provide longevity with comfort for the patient. The inexorable course of the disease in certain patients, despite radical therapy, and the very moderate increase in salvage following a marked rise in the operability and resectability rates have convinced many that gastric cancer is at present a surgically insoluble problem. Warren (1949) and Meissner (1949) have demonstrated that, even in those patients in whom the tumour appears to be localized to the stomach without involvement of regional lymphatic glands, contiguous organs or liver, there may be invasion of the blood vessels by the neoplasm and consequent appearance, later, of metastases carried by the vascular route. This evidence has been accepted by some as justification for the adoption of their conservative philosophy. To others, the

phenomenon of vascular invasion in localized lesions is not considered sufficiently universal to vindicate surgical surrender.

There are many surgeons who would hesitate to admit that their efforts are directed at palliation rather than cure but who, by the very type of surgery they perform, pay unwitting allegiance to the conservative school. These are the surgeons who, for distal lesions of the stomach, perform the routine partial gastrectomy originally designed for the treatment of duodenal ulcer and who leave intact the major portion of the greater and lesser omenta. They are content to extirpate only those lymphatic nodes which occur along the course of the major gastric vessels and ignore the other equally important potentially involved members of the first echelon of perigastric lymph glands. For proximal lesions of the stomach they exhibit a curious reluctance to open the thoracic cage to perform either a total gastrectomy or an oesophago-gastrectomy. These are the surgeons who, by failure to understand the behaviour of the disease, by lack of knowledge of the principles of cancer surgery or lack of confidence in their own ability to perform the major surgical procedures necessary, share, with those of the conservative school, much of the responsibility for the low salvage rate following therapy.

It is probable that the surgeon who, unwillingly, has to accept compromise will make every effort to ensure that the

compromise with the disease is the best possible one even if, as a result of his efforts, the patient will endure some modification of his normal feeling of well-being. By extending the compromise to the quality of the patient's postoperative life, the surgeon hopes to exchange a moderately impaired quality of life for an enhanced chance of survival. This is a compromise which would be acceptable to most patients and which should be the objective of those surgeons who assume the responsibility for the care and treatment of patients with gastric carcinoma.

THE CHOICE OF THERAPY

The recent advances in the understanding of electrolyte and water balance, the physiology of respiration, the control of infection and the greater knowledge regarding the choice and action of anaesthetic drugs and techniques have permitted the surgical attack on gastric cancer to be more radical with little or no increase in the operative risk to the patient.

The choice of which radical operative procedure should be advocated as routine therapy has given rise to considerable controversy. There are only two alternatives, namely total gastrectomy and subtotal gastrectomy. There is little disagreement regarding the wisdom of adopting total gastrectomy when conditions of size, extent and site of the gastric tumour demand such a procedure, but in those cases in which subtotal gastrectomy would appear to be adequate to encompass the primary lesion

there is difference of opinion on whether total extirpation of the stomach should also be the procedure of choice. Irrespective of which procedure is adopted, much depends upon the skill and experience of the surgeon and upon his determination to take serious risks if thereby he can give the patient a chance of cure (Taylor, 1947).

SUBTOTAL GASTRECTOMY

For carcinoma of the distal half of the stomach, the most widely accepted method of treatment is subtotal gastrectomy and for proximal lesions oesophago-gastrectomy. It is unfortunate that neither of these procedures is well defined in terms of the amount of stomach removed and it is probable that many operations which are described as being subtotal in extent have little claim to belong to that category of radical procedure.

A properly conducted subtotal gastrectomy is a radical procedure and should include removal of all of the first part of the duodenum, the pylorus, antrum, all of the lesser curvature of the stomach and of the greater curvature to within four or five centimetres of the cardia. The gastro-colic, gastro-hepatic and greater omenta should be removed in their entirety. Careful dissection and removal of the lymphatic tissue along the lesser curvature, in the region of the coeliac axis including that along the superior border of the pancreas, in the subpyloric and

subhepatic and paracardial regions should be done. The gastrolienal nodes should be removed by combining splenectomy with the procedure. This latter component of the operation of subtotal gastrectomy was advised by Lahey and Marshall (1941) and Coller (1941). Eker (1951) demonstrated metastases in the splenic nodes in 21.7 per cent of forty-six resected gastric specimens. Fly (1956) found splenic nodal involvement in 30 per cent of patients with cancer situated in the distal part of stomach and who had had splenectomy done as part of the operative procedure, and in forty per cent of those patients on whom there was pathological evidence of spread to regional lymphatic glands. In some of the cases only the splenic nodes were involved. The splenic hilum nodes were involved in 43.5 per cent of eighty-five specimens with nodal metastases and in 36.3 per cent of the total of 102 cases. It has not been a common practice to include splenectomy with subtotal gastrectomy in treatment of cancer of the distal segment of stomach, although Lewis and Wangensteen (1950) have advised it as an essential part of the routine surgical procedure. The results of therapy are so poor that any suggestion which might increase the salvage is valuable. Splenectomy with gastrectomy is one such example. When splenectomy is performed along with subtotal gastrectomy a better opportunity is afforded for the removal of the lymph nodes along the superior border of the pancreas. Recently Wangensteen (1954), has advocated extending

the procedure to include dissection of lymphatic tissue along the hepatic artery, common bile duct, the lymph nodes adjacent to the cystic duct and artery, as well as those lying along that part of the inferior vena cava related to the duodenum. The importance of extending the operation in this way is evident when it is realized that at least three quarters of the gastric lymphatic drainage ultimately reaches the hepatic chain of glands.

The advocates of subtotal gastrectomy believe that as satisfactory a lymph gland dissection can be done by subtotal as by total gastrectomy, that past experience has proved that a better five year survival rate can be obtained by subtotal than by total gastrectomy, that the increase in the operative mortality rate following complete extirpation of the stomach over that of the subtotal procedure does not justify the adoption, as routine treatment, of the more radical operation, and that total gastrectomy is always followed by gross nutritional disturbance which can be avoided by preservation of even a tiny cuff of stomach.

TOTAL GASTRECTOMY

From the theoretical point of view there are few surgeons who would not agree with the declaration made by Kroenlein in 1898, that total gastrectomy is the most definitive operation for gastric cancer. The advantages associated with the procedure include the wide operative exposure which permits a lymphatic gland dissection probably superior to that of subtotal gastrectomy,

the removal, with the stomach and the adjacent portions of duodenum and oesophagus, of the whole intramural gastric lymph pool which might otherwise be a potential source of recurrent tumour, and the opportunity to remove widely the paracardial and paraoesophageal lymphatic tissue which can be done only inadequately in subtotal gastrectomy. The spleen, with the gastrolienal and the lieno-renal ligaments can be removed widely without anxiety regarding the conservation of the blood supply to the small gastric remnant left in the subtotal procedure. The practical objections to the routine use of total gastrectomy include the high immediate mortality, the short survival periods of those patients who recover from the operation, because of recurrence of cancer in the gastric bed or the later appearance of distant metastases, and the postoperative unpleasant digestive discomfort and nutritional disturbance which occur during the months of life salvaged by the procedure. Pack in 1943 rationalized the apparent inconsistency of theoretical approbation of and the practical objection to the procedure by stating "If we could banish the thought of all operative dangers and postoperative complications, in theory we could assert that total gastrectomy is a far more logical procedure than partial or subtotal gastrectomy."

When a malignant tumour of the stomach is so extensive that it cannot be safely or completely removed by subtotal gastrectomy, or when there is a more localized tumour but

with metastases extending high and adherent to the lesser curvature near the cardia, or when there are multiple primary gastric cancers present and in cases of the superficial spreading type of gastric carcinoma, total gastrectomy is the procedure of choice. These types of cases would fall naturally into the classification "Gastrectomie Totale de Necessite," described by Gutgemann (1952).

THE HAZARDS ASSOCIATED WITH TOTAL GASTRECTOMY

The objections to total gastrectomy are directed at the adoption of the procedure in those cases in which subtotal gastrectomy would appear to be adequate to encompass the local tumour, that is, to the "Gastrectomie Totale de Principe" of Gutgemann. These objections are substantial and are worthy of investigation.

OPERATIVE MORTALITY FOLLOWING TOTAL GASTRECTOMY

Total gastrectomy is not a procedure which should be undertaken lightly, and should be performed only by surgeons whose training, experience and surgical discrimination are such that the operative risk to the patient is minimized. Until recent years, even in the most competent surgical hands, the operative mortality was formidable, and in some cases prohibitive. With the advances in technique and technical skill which reduced the risk of leakage from the anastomotic site, and with the judicious

use of antibiotic therapy, the incidence of peritonitis, subphrenic abscess and mediastinitis which were among the commonest causes of postoperative death following total gastrectomy, became greatly reduced. Careful anaesthesia and attention to the maintenance of normal respiratory function before, during and after the operation reduced the incidence of atelectasis, pneumonia and empyema. The adoption of preventative measures against the onset of operative shock and the treatment of that condition if it supervened, in addition to careful attention to water and electrolyte balance contributed greatly to the reduction in operative mortality.

The reduction in the rate of operative death following this procedure has been dramatic over the past five or six years (Table 80) and is now approaching the mortality following the less radical procedure of subtotal gastrectomy.

POSTOPERATIVE SURVIVAL FOLLOWING TOTAL GASTRECTOMY FOR GASTRIC CANCER

The mere fact that more patients now survive the procedure is not sufficient to justify its routine adoption unless, as a result, the prolongation of life of the operative survivors is greater than that of those undergoing subtotal gastrectomy. On this rock of survival comparison the argument for routine use of total extirpation of the stomach would appear to founder.

Tables 81 and 82 present a selection of the comparative five year

Authors	Year Reported	Number of Patients	% Operative Mortality
Walters et al	1942	27	66.7
Finsterer	1953	150	57.3
Finney et al	1929	67	53.8
Troell	1953	54	51.0
Finsterer	1950	103	49.5
Pack et al	1943	298	37.6
Lahey et al	1944	73	33.3
Pack et al	1947	41	31.7
Winkelbauer	1952	32	25.0
de Amesti	1950	30	13.3
ReMine et al	1952	62	12.9
Cooper	1952	38	11.0
Longmire	1947	20	10.0
Scott et al	1949	63	9.5
Lahey et al	1950	64	9.4
Walters et al	1953	22	9.1
Marshall et al	1954	127	8.7
Rhoads	1951	23	8.6
McNeer et al	1952	33	6.1
Walters et al	1952	19	5.3
Wangensteen	1949	28	3.5

OPERATIVE MORTALITY FOLLOWING TOTAL GASTRECTOMY

TABLE 80

Author	Five-year Survival Rate		
	Year Reported	% of Cases Resected	% of cases who survived Resection
Jemerin et al	1952	21.7	
Gray	1942	28.9	
State et al	1947	29.4	
Harnett	1947	29.0	
Cooper	1952	34.8	
Walters	1953	34.8	
Lahey et al	1950		22.3
Barclay et al	1951		26.5
Pack et al	1948		34.7

REPORTED FIVE YEAR SURVIVAL FOLLOWING SUBTOTAL GASTRECTOMY

TABLE 81

Author	Year Reported	Five year Survival Rate	
		% of Cases Resected	% of Cases who survived Resection
Sweet	1953	7.1	-
Lahey et al	1950	9.4	21.1
de Amesti	1950	11.0	-
Finsterer	1953	-	17.3
ReMine et al	1952	18.0	-
Marshall et al	1954	-	14.1
Walters	1951	28.9	-
Pack et al	1948	50.0	-

REPORTED FIVE YEAR SURVIVAL RATES FOLLOWING TOTAL GASTRECTOMY

TABLE 82

survival rates of those patients who were treated by total and subtotal gastrectomy. It is evident from this comparison that greater success has followed the routine use of subtotal gastrectomy. This evidence, however, is not without bias. Most authors who have reported their results following total gastrectomy for gastric carcinoma have emphasised that their results were obtained in cases in which the tumour in the stomach was too widespread to allow resection by subtotal gastrectomy and which would otherwise have required to be regarded as hopeless. There have been no reports presented in the literature of the five year survival rate when total gastrectomy has been performed in patients whose local gastric lesions could have been extirpated by subtotal gastrectomy. Until such reports are made available total gastrectomy should not be condemned as an acceptable form of treatment on the basis of the apparently better survival rates following subtotal gastrectomy. On the other hand, claims should not be advanced in support of total gastrectomy merely because, theoretically, it would appear to be the better procedure. With the appreciation of the potential lymphatic spread of the disease and the limitations of surgery to encompass all of the potentially involved tissues, it is possible that subtotal gastrectomy may be able to offer a compromise with the disease comparable with total gastrectomy without giving rise to the postoperative complications usually associated with the latter procedure. This has not yet been satisfactorily proven, however,

and in the light of the lack of marked surgical success in the treatment of the disease by subtotal gastrectomy, it might be justifiable for a group of experienced and capable surgeons to determine whether, in localized lesions in the stomach with lymphatic metastases, greater salvage could be effected by the more radical operation.

NUTRITIONAL AND METABOLIC DISTURBANCE FOLLOWING TOTAL GASTRECTOMY

Following total gastrectomy patients may present a symptom complex completely unassociated with the disease for which the operation was performed, but which arises as a direct consequence of the operative procedure itself. Not the least important of this group of symptoms is the nutritional derangement which becomes evident almost immediately after the operation and which is considered by many to be an inevitable sequel to the procedure [Meyer (1951), Mikkelson (1949), Scott (1949), Smith (1947) and West (1949)].

In order to ascertain the cause of this nutritional impairment, investigations have been conducted to assess the ability of totally gastrectomized patients to digest and absorb simple dietary constituents from their alimentary tracts.

ASSIMILATION OF FAT

In 1928, Flint described the light coloured liquid faeces which were evacuated following total gastrectomy and which

indicated a deficiency in fat absorption. The phenomenon had previously been observed by MacDonald in 1898 but its significance was not appreciated at that time.

Sufficient evidence has been collected over the past fifteen years to demonstrate that steatorrhoea commonly occurs following total gastrectomy and that it may be of severity great enough to explain at least part of the nutritional disturbance which follows the operation. [Reckers (1943), Farris (1943), Everson (1952), MacDonald (1947), Brain (1953) and (1951)].

To reduce the fat malabsorption, Brain and Stammers (1951) have recommended that each patient should be given a diet, containing 100-120 grams of fat daily with a correspondingly high caloric intake. The diet should be divided into small amounts so that fat is taken eight to ten times in the twenty-four hours. They have reported that on this regime some of their patients had a greater food intake postoperatively than was considered normal for them preoperatively and that a satisfactory gain in weight was common. By administering this high fat diet and dividing the twenty-four hour dietary sample into small regular feeds they were able to raise the amount of fat absorbed in some cases to a degree approaching normality.

PROTEIN ASSIMILATION

A normal person, on an average, excretes between one and two grams of nitrogen per day (Peters 1946). Survey of the

literature reveals that of the fourteen patients on whom protein utilization studies were performed, in only six was there evidence of impaired assimilation after total gastrectomy. The largest nitrogen loss was 4.8 grams reported in a patient operated upon three months previously (Bull 1934).

Blake (1953) investigated the serum protein levels in nine patients who had had total gastrectomy performed less than three years previously and in none was there any evidence of abnormality. Eight of the patients had a normal albumin-globulin ratio and in one the ratio was reversed.

CARBOHYDRATE ASSIMILATION

Alimentary intake-excretion studies do not provide a satisfactory method of determining carbohydrate assimilation. The carbohydrate which is not digested and absorbed in the small intestine may be utilized by the carbohydrate fermenting organisms of the intestinal tract and consequently the quantity of carbohydrate which is not assimilated cannot be measured accurately by fecal determinations.

Following total gastrectomy, glucose tolerance curves have been studied and have been found to differ from normal curves in that the initial hyperglycaemic phase is more pronounced and is followed by a rapid fall to hypoglycaemic levels [Farris (1943), Ingelfinger (1944)].

Evenson (1941) and MacDonald et al (1947) were unable

to demonstrate the hypoglycaemic phase in their series. The variation from normal may be the result of an increase in the rate of absorption of glucose since intravenous glucose tolerance tests have been shown to give normal curves.

VITAMIN ASSIMILATION

Few studies have been made on vitamin assimilation following total gastrectomy. MacDonald (1947) had vitamin A tolerance tests done on three patients who had had total gastrectomy performed ten, five and three years previously. After giving 300,000 units of vitamin A by mouth, they estimated the blood levels of the vitamin and found that in two cases there was a normal increase and in the third case (upon whom total gastrectomy had been performed five years previously) the tolerance curve was quite flat. A remarkable feature of this study was the delayed absorption of the vitamin evident in all three patients. Ingelfinger (1943) found that when vitamin A was introduced directly into the jejunum of a normal person the blood level was sharply elevated within two hours. In the series reported by MacDonald (1947) two and three hour blood levels of Vitamin A were lower than corresponding levels obtained in normal persons taking the test dose by mouth. There was no clinical evidence of vitamin A deficiency in these patients despite the findings of the investigation.

Brain (1951) has reported clinical evidence of vitamin B

complex deficiency in nine of forty patients who had had total or almost total gastrectomy. The onset of this deficiency occurred within a few days after operation. It is likely that factors other than the removal of the stomach were responsible for the changes observed.

Apart from those patients who exhibited megaloblastic anaemia, there was no evidence of a vitamin B deficiency in the studies undertaken by Blake and Rechnitzer (1953). None of their patients demonstrated any other evidence of vitamin deficiency.

MacDonald et al (1947) report normal levels of serum calcium and phosphorus after total gastrectomy and this is taken to indicate that at least an adequate amount of vitamin D is absorbed postoperatively. Similarly they interpreted postoperative normal prothrombin times as an indication of absorption of vitamin K. Pack (1943) also made a study of the prothrombin levels in the plasma in six patients who had been subjected to total gastrectomy. In all of them the level was reduced by twenty to forty-five per cent of the preoperative concentrations. They found that the hypoprothrombinaemia in these individuals did not respond to the parenteral administration of vitamin K and on this basis believed that it was due to hepatic dysfunction rather than to the removal of the stomach directly.

It would appear from the evidence presented that the assimilation of fat is the only common alimentary disturbance

following complete removal of the stomach. Further examination of the problem of post-gastrectomy inanition, with consideration of the other theories of causation which have been advanced, is of interest.

LOSS OF APPETITE

The presence of the stomach is not essential for the maintenance of appetite [Brigham (1898), Paterson (1906) and Schlatter (1898)] nor for the enjoyment of good food - (Harvey 1900). Longmire (1947) reported that of ten patients observed for four to nineteen months postoperatively, all had good appetites. In a group of thirteen patients who had survived total gastrectomy for five years, nine had unimpaired appetite (ReMine 1952).

LOSS OF RESERVOIR AND TRITURATING FUNCTIONS OF STOMACH

To the loss of the normal reservoir function of the stomach and its ability to transfer food into the duodenum intermittently and in small quantities has been attributed part of the postoperative nutritional upset in patients who have had complete removal of their stomach and an anastomosis performed between the oesophagus and jejunum.

The special physiological mechanism which prevents the sudden entrance of large quantities of fat into the small intestine is upset. Normally, the hormone enterogastrone, one of whose major actions is to inhibit gastric motility, is liberated

when fat is placed in the small intestine, and consequently overburdening of the fat digesting and fat absorbing capacities of the intestine are prevented. With the removal of the stomach and an anastomosis performed between oesophagus and jejunum, this controlled feeding of fat into the intestine is lost.

The normal triturating function of the stomach, which breaks down the ingested food into fine particles which are readily available to the digestive enzymes of the gastrointestinal tract, is also lost. The large pieces of unprepared food which are passed into the small intestine are not so readily digestible and this has been offered as one of the major reasons for the post-operative nutritional imbalance. Evidence to the contrary has been presented by Everson (1952) who reported that even with the food finely ground and the non-appearance of grossly unchanged food in the stool after total gastrectomy, there was still evidence of impairment of assimilation of fat and protein in experimental animals.

LOSS OF DIGESTIVE FUNCTION OF THE STOMACH

The satisfactory nutritional status of some patients with achlorhydria and with pernicious anaemia, in which pepsin is either lacking or inactivated by the absence of hydrochloric acid, is strong, but perhaps presumptive evidence against the loss of these factors in the gastric juice being responsible for the production of the nutritional imbalance which occurs after

total gastrectomy. Pack and McNeer (1943) report the failure of the addition of large doses of ventriculum to the diet of three gastrectomized patients over ten days to reduce abnormal fat excretion in the faeces. Similarly the administration of hydrochloric acid had no beneficial effect.

REDUCTION IN NORMAL PANCREATIC SECRETION AND INADEQUATE MIXING OF FOOD WITH PANCREATIC AND BILIARY SECRETIONS

It has been postulated that the secretion of pancreatic enzymes is greatly reduced after removal of the stomach - (Ingelfinger 1944). Deficient pancreatic secretion may result either because the vagi have been sectioned at operation (Shingelton 1951) or because secretin is not elaborated in normal amounts if acid chyme fails to enter the duodenum. Likewise, vagal control of biliary secretion has been demonstrated by Tanuri (1938). It has been observed that pancreatic and biliary dysfunction has not been evident in patients upon whom vagotomy has been performed in the treatment for duodenal ulceration. Following removal of the stomach and the union of the oesophagus to the jejunum, the stimulation of the pancreas to produce its digestive enzymes is not entirely abolished, but is reduced because of the lesser ability of the jejunum to produce secretion as compared with that of the duodenum - [Baylers (1903), Burns (1948), Drewyer (1929), Friedman (1950) and Mellanby (1926)].

It has been postulated (Everson 1952) that, following total gastectomy and oesophago-jejunostomy, the ingested food may have passed far into the distal intestinal tract before the pancreatic and biliary secretions are delivered to the upper intestine. To avoid this, Brain (1953) advocates the administration of a high fat content diet in small numerous meals given throughout the day. He believes that this simple procedure will correct the altered gastro-duodenal dynamics caused by oesophago-jejunostomy and improve the co-ordination between the entrance of food into the intestinal tract and the secretion of bile and pancreatic enzymes. Melbourne (1954) has shown, however, that the fat loss in the faeces differs little whether the oesophagus is anastomosed to the duodenum or jejunum. This casts doubt upon the theory that diminished fat utilization occurs because the pancreatic juice and bile chase food instead of mixing with it.

INCREASED INTESTINAL MOTILITY

It has often been stated that the passage of food down the intestinal tract is more rapid following total gastrectomy with oesophago-jejunostomy and that, as a consequence, less time is available for the intestinal digestion and absorption of the normal dietary constituents. Farris et al (1943) found that there was a transitory increased motility of the intestinal tract after operation, but when these patients were observed over a period of months, this subsided and eventually a moderate decrease in motility was

observed. Pack and McNeer (1943) by using charcoal as a marker, and MacDonald et al (1947), by radiological methods and kymographic tracings, confirmed the observations made by Farris. The diarrhoea which is often evident immediately after operation has usually subsided within three months after operation (Brain and Stammers, 1951).

DIMINISHED CAPACITY FOR FOOD

The total daily intake of food is invariably less after complete removal of the stomach than before operation. It tends to increase with time, but with standard feeding routine, rarely reaches normal. Without adequate food intake the patient's ability to work diminishes and difficulty is experienced in maintaining weight. ReMine and Priestley (1952) reported that of thirteen patients who survived for five years or longer after total gastrectomy, sixty-nine per cent regained their preoperative weight but not their normal weight. The remainder were unable to gain or maintain a satisfactory weight level. Longmire (1947) reported that of ten patients followed postoperatively, all gained weight, but the increase was not great and none attained their normal level. Marshall (1954) found the general trend of weight to be downward, and of his 149 patients on whom total gastrectomy had been performed only ten maintained their postoperative weight, eleven gained from two to twenty pounds, and the remainder lost weight. Of these 149 patients only thirty-nine lived three years

or more, and since it is assumed that most of these patients died of recurrences of their neoplasm or with an extension of the original pathological process still in existence, weight increase in them was not to be expected. Everson (1952) reported only three cases out of forty in whom the weight returned to normal.

Few efforts have been made to correlate the postoperative weight loss with the measurement of the quantity and quality of the patient's food intake. Attention has been concentrated mainly on the failure of the intestinal tract to utilize the food ingested. While it would be rash to state that no alteration in the ability of the intestine to assimilate the necessary constituents of diet after total gastrectomy occurs, it is possible that the disorganization of the normal digestive and absorptive processes is of lesser degree than is generally assumed, and that by the simple expedient of administering to the patients small frequent meals of high caloric value and containing a large amount of fat and protein, many of the problems of weight maintenance and post total gastrectomy inanition would be overcome. Brain and Stammers (1951) reported that five patients, in whom this dietary regime was adopted, "ate more food than they had ever eaten in their lives" and all of them returned to approximately their normal weights.

ANAEMIA FOLLOWING TOTAL GASTRECTOMY

The normal stomach has an intimate relationship with the haemopoietic system in that it secretes hydrochloric acid

which facilitates iron absorption [Bruusgaard (1946), Barer (1937) and Moore (1939)] and liberates intrinsic factor, the presence of which is necessary for the absorption of Vitamin B_{12} and normal erythropoiesis. (Castle 1939, 1929, 1930). In the absence of hydrochloric acid a microcytic type of anaemia develops, while, with failure of absorption of Vitamin B_{12} a megaloblastic anaemia ensues, with typical changes in the peripheral blood and in the bone marrow.

Following subtotal gastrectomy and as an immediate result of total gastrectomy, iron deficiency anaemia is common. The onset of true megaloblastic anaemia following total gastectomy is not an immediate phenomenon, however, and usually takes two or three years to become evident. Until recently it was difficult to correlate this delayed appearance of the Addisonian type of anaemia with the belief that the intrinsic factor of Castle was exclusively of gastric origin. Because of the absence of this type of anaemia in many patients studied at short intervals after total gastrectomy [Beebe (1949), Pack (1943) and Scott and Longmire (1949)], the possibility that the intrinsic factor could also be elaborated in the duodenum and small intestine were entertained by some.

Recent studies on the intestinal absorption of Co^{60} - labelled Vitamin B_{12} have resulted in a better understanding of the problem, and have provided an explanation of the late development of megaloblastic anaemia following total gastrectomy.

Heinle et al (1952), demonstrated that patients with pernicious anaemia could not absorb Co^{60} - vitamin B_{12} in normal amounts after its oral administration. Similar findings were obtained in comparable studies done on patients following total gastric resection, [Swendseid (1953), Halsted et al (1954), Ley and Sharpe (1954), Callender (1954), and Krevans et al (1954)] . These workers demonstrated that the impaired or entirely abolished absorption of vitamin B_{12} could be overcome by the oral addition of normal human gastric juice or potent intrinsic factor preparations from hog stomach. This evidence corroborated Castle's original theory that the intrinsic factor is formed exclusively in the stomach and that the total removal of this organ results in the complete absence of intrinsic factor from the body, thus creating a circumstance similar to that present in pernicious anaemia. This work was confirmed by Schilling (1953).

Pitney and Beard (1955), showed that as a result of the sudden withdrawal of intrinsic factor as, for example, after total gastrectomy, there was not an immediate gross reduction in the serum levels of vitamin B_{12}. The level fell slowly after operation, but did not fall below the normal minimum of 86 μg. per ml. until eight months had elapsed. After nineteen months the serum level of vitamin B_{12} was below the upper limit evident in patients with pernicious anaemia (50 μg. per ml.) but despite this there was no evidence that the patient had developed megaloblastic anaemia.

Jerzy Glass (1955), has shown by the measurement of radioactivity over the liver following the administration of Co^{60} - vitamin B_{12} in a patient whose entire stomach had been removed, that there is prolonged hepatic storage of vitamin B_{12} in the liver. It is possible that the vitamin B_{12} which has been stored in the liver before operation is sufficient to maintain the patient in reasonable haematological balance for long periods after the operation and that this hepatic vitamin B_{12} depot may explain the long interval between the completion of the total gastrectomy and the appearance of the vitamin B_{12} depletion with clinical manifestations of megaloblastic anaemia.

It is possible to maintain the normal vitamin B_{12} level by the parenteral administration of the vitamin, or by giving it by the oral route combined with potent intrinsic factor.

SUBSTITUTE STOMACH

Following on the assumption that the nutritional imbalance which follows total gastrectomy is probably due to a combination of several factors such as the lack of a gastric reservoir for an adequate food intake, the deviation of food from the normal alimentary pathway through the duodenum and perhaps the dumping syndrome, efforts have been made to restore continuity between the oesophagus and duodenum. Oesophagoduodenostomy was first advocated by Brigham in 1898 and more recently by Priestley (1948), Palumbo (1950) and Judd (1952). The technical difficulties

of this procedure are considerable and the risk of undue strain on the anastomosis is great, especially when there is involvement of more than the distal one inch of the oesophagus.

To avoid these and other less desirable features of direct communication between the oesophagus and duodenum, such as proximal reflux of upper intestinal content, attention was directed to means whereby a substitute stomach could be formed by employing a segment of the intestinal tract to form a bridge between the lower end of the oesophagus and the proximal open end of the duodenum. The procedure was first suggested by Nicoladoni in 1887. Segments of jejunum [Tomoda (1951), Teishin (1941) and Longmire (1952)] , the right colon with a portion of ileum - [Lee (1951), McAleese (1952), Carleson (1952), McGlone (1953), Szilagyi (1952), Hunnicut (1952)] and transverse colon, [State (1951), D'Errico (1950) and Laborith (1951)], have been utilised for this purpose. None of these procedures has obtained ready acceptance and it is difficult, in view of the paucity of information available on large numbers of patients who have been treated in this way, to evaluate which of the methods gives the best results, or indeed to determine whether the principle on which the procedures are based is sound. The writer has experience only in the use of the transverse colon, which because of its anatomical position as well as its ability to distend, appears to be a suitable substitute for the excised stomach. In common with the use of the terminal

ileum, caecum and ascending colon segment advocated by Lee (1951), the transverse colon has one other important virtue, namely that long segments can be mobilized and used when the carcinoma of stomach extends into and involves the oesophagus for a long distance proximally. In the standard oesophago-jejunostomy the level to which the oesophagus can be removed and to which the small bowel can be brought up for anastomosis is limited even when the initial branches of the superior mesenteric artery at the base of the small intestinal mesentery have been cut and a Roux Y type of procedure used as has been suggested by Reynolds and Young (1948). At autopsy the writer, with his co-workers (Barclay, State and Kelly 1951), was able to mobilize the transverse colon and descending colon sufficiently to allow the almost complete replacement of the entire thoracic oesophagus.

In the procedure described by the writer, the colonic segment was purposely placed in an anti-peristaltic fashion because it was felt that the reverse peristalsis would result in a longer retention of food within the colonic pouch and enhance the value of this segment of bowel as a food reservoir. Fluoroscopic examination following ingestion of barium demonstrated that peristaltic activity was prominent in the distal part of the colonic segment. However, in addition, waves were seen to proceed slowly from the oesophageal side to the duodenum, carrying barium with each peristaltic wave. Subsequent

examination of the patient confirmed the impression that the pouch had taken on some of the characteristics of a reservoir in that approximately fifty per cent of the barium was retained for an appreciable time and, in addition, the peristaltic activity presented to the duodenum material from the colonic pouch in a manner similar to that accomplished by the stomach.

The fact that ingested food passes into the duodenum in approximately a normal fashion following this procedure and that, in consequence, the pancreas, duodenum and biliary tree receive a stimulus to secrete, would appear to support the plea that such a procedure has merit. Proper and timely admixture of ingested food with the pancreatic, biliary and upper intestinal secretions, the ability of the patient to ingest a greater quantity of food at each meal and the advantage of being able to introduce hydrochloric acid into the duodenum and stimulate the production of secretin would commend a more extensive trial of the operation to prove whether the theoretical advantages can be realised in practice.

CHAPTER 9

CONTROLLABLE FACTORS WHICH INFLUENCE THE SALVAGE RATE IN GASTRIC CANCER

There are several factors to which can be attributed the responsibility for the poor salvage rate in patients with gastric cancer. A major contributing factor is the very nature of the disease itself (MacDonald 1954) with its tendency to present symptoms only when the pathological process is advanced and has already extended beyond the limits of the stomach to the regional lymphatic glands and to adjacent structures. When the disease has extended beyond the limits of surgical extirpation the prognosis is hopeless and no efforts by the surgeon or any other therapist will alter materially the inexorable course of the disease.

In many cases the patient is culpable by harbouring the disease for long periods after it has become symptomatic without seeking medical care. It is reasonable to assume, since we have no evidence to suggest that gastric cancer is other than a progressive

disease however slow the progression may be in some cases, that with delay the chance of extra-gastric spread increases. This reasoning is not necessarily at variance with the demonstration by certain workers that the survival period following subtotal gastrectomy varies directly with, or has no relationship to, the stated duration of symptoms [Barclay (1952), Balfour (1937), Walters et al (1940), Swynnerton (1952)].

It is notable that there is a loss of salvage which cannot entirely be divorced from the surgeon's responsibility. His judgement in the determination of operability and resectability, and the operative mortality attendant upon the surgical procedures performed by him are factors which are of the greatest significance. Furthermore, his surgical courage in endeavouring to excise all tumour tissue, and his operative experience in performing this successfully, are prognostic factors of importance.

OPERABILITY

The determination of operability is the first step in the treatment of carcinoma of the stomach. The responsibility for the decision on whether the patient is operable rests with the surgeon alone. Each patient requires to be assessed on an individual basis, and factors relating to his general physical status, as well as the condition of his gastric lesion must be taken into consideration. Only by surgical exploration of the abdomen can the resectability of the lesion be determined, and denial of this investigation to a

patient automatically removes him from the group of potential long term survivors.

Operability rates, as reported in the medical literature, vary from centre to centre. The variations probably reflect not only the extent to which the disease has progressed in the average patient when he is admitted for treatment but also the philosophy of the surgeons into whose hands the patients are placed. Large medical centres and university hospitals frequently report high operability rates, while those recorded from smaller clinics and general non-teaching hospitals are usually more modest. The higher operability rates from the larger centres probably indicate the selectivity of the patients referred to them since, in general, these institutions rarely admit obviously inoperable patients or the patients are screened by medical colleagues prior to their reference to the surgeons. In the Saskatchewan series presented in this monograph, selection of patients is minimal since most of the cases of gastric cancer in the province are referred to the cancer clinics irrespective of the extent of the disease or the general condition of the patients.

The generally accepted criteria for inoperability are the presence of umbilical metastasis, rectal shelf, Virchow's nodes and general intraperitoneal carcinomatosis with ascites. Concurrent disease of the cardiovascular, respiratory and renal systems

may be of such severity that surgical exploration may be contraindicated. Advanced age in itself should not be a contraindication to surgery although, in combination with severe and irremediable degenerative systemic disease, it may increase the hazards of surgery beyond reason.

In the Saskatchewan series during the twenty-four year period between 1932 and 1955, there was a steady increase in operability, attaining an acceptable average rate of 72.6 per cent over the last four years of the study (Table 83). When the 614 cases in which the reason for inoperability was stated are studied certain features are notable (Table 84). Forty patients, representing 6.5 per cent of the total cases in which the reasons for inoperability were given, elected to have treatment outside the province of Saskatchewan. Of these, information on operability is available on thirty-eight. Twenty had gastric resections, four had palliative procedures and fourteen had abdominal exploration only. All of the eight patients (1.3 per cent) who were referred outside the province for treatment were operated upon, two having gastric resections, one a palliative procedure and five had surgical exploration only. Those patients who had adequate resections performed elsewhere before reporting to the cancer clinics in Saskatchewan, and those who reported on the first occasion with inoperable recurrence of their disease, were responsible for 6.3 per cent of the group under review.

Years	Total Number of Patients	Number Operated Upon	Number Not Operated Upon	Operability (per cent)
All years	2000	1189	811	59.5
1932-36	138	54	84	39.1
1937-41	204	94	110	46.3
1942-46	395	198	197	50.4
1947-51	686	424	262	61.8
1952-55	577	419	158	72.6

GASTRIC CANCER: THE OPERABILITY RATES DURING FIVE SUCCESSIVE PERIODS

SASKATCHEWAN 1932-55

TABLE 83

Stated Reason For Inoperability	Per Cent					
	All Years	1932-36	1937-41	1942-46	1947-51	1952-55
Treatment refused	11.6	12.5	6.9	6.9	15.7	13.1
Concurrent disease	1.8	-	1.7	0.6	0.5	5.0
Disease too advanced	68.0	70.8	75.8	78.3	61.4	61.9
Patient too old	2.8	8.3	1.7	-	3.0	5.0
Patient went elsewhere for treatment	6.5	4.2	6.9	5.7	10.7	2.5
Patient referred elsewhere for treatment	1.3	-	-	2.8	1.6	-
Treatment elsewhere with inoperable recurrence	3.9	4.2	5.3	2.8	3.5	5.0
Treatment elsewhere with no evidence of recurrence	2.4	-	1.7	2.3	2.6	3.1
Mistaken diagnosis thought to be benign ulcer	1.0	-	-	-	-	3.8
Unsuspected cancer	0.7	-	-	0.6	1.0	0.6

GASTRIC CANCER: STATED REASONS FOR INOPERABILITY IN 614 PATIENTS

SASKATCHEWAN 1932-55

TABLE 84

It is remarkable that only eleven patients (1.8 per cent) were considered inoperable because of concurrent disease. Seventeen patients (2.8 per cent) were not operated upon solely because of advanced age.

Gastric disease which was considered to have extended beyond the scope of surgery was given as the reason for inoperability in 418 patients (68 per cent). Six of these patients lived for more than five years. Retrospectively it is impossible to determine in how many of these patients the surgical opinion was faulty but it would appear that a proportion of patients might have been salvaged had the opinion on incurability been based on observations made at the time of surgical exploration rather than on clinical impressions only.

The sex of the patient did not appear to be a factor in determining operability (Table 85), and it is doubtful if the influence of age is a factor (Table 86). The apparent overall poorer operability rate in the age group over sixty-five is due mainly to the low operability rates in the earlier years. In the last four years of the study, during which there was a better understanding of preoperative supportive therapy, age did not appear to be a significant contraindication to operation.

Year	Both Sexes		Male		Female	
	Number of Operable Patients	Operability (Per cent)	Number of Operable Patients	Operability (Per cent)	Number of Operable Patients	Operability (Per cent)
All years	1189	59.5	889	59.6	300	59.1
1932-36	54	39.1	40	40.0	14	50.0
1937-41	94	46.3	72	45.3	22	52.2
1942-46	198	50.4	150	49.8	48	57.3
1947-51	424	61.8	318	63.5	106	74.4
1952-55	419	72.6	309	72.0	110	74.3

GASTRIC CANCER: OPERABILITY BY SEX

SASKATCHEWAN 1932-55

TABLE 85

Year	All Ages		Age in Years							
			Less than 45		45-54		55-64		65 and over	
	Number of patients operated upon	Operability (per cent)	Number of patients operated upon	Operability (per cent)	Number of patients operated upon	Operability (per cent)	Number of patients operated upon	Operability (per cent)	Number of patients operated upon	Operability (per cent)
All Years	1189	59.5	100	74.6	183	69.6	416	68.9	490	49.0
1932-36	54	39.1	7	53.9	12	54.6	22	37.7	13	23.1
1937-41	94	46.3	8	57.1	26	54.2	40	54.8	20	29.4
1942-46	198	50.4	24	70.6	39	72.2	78	56.9	57	33.9
1947-51	424	61.8	39	92.9	60	87.0	151	77.4	174	45.8
1952-55	419	72.6	22	71.7	46	78.7	125	86.3	226	65.6

GASTRIC CANCER: OPERABILITY BY AGE GROUP

SASKATCHEWAN 1932-55

TABLE 86

The data presented in Table 87 would indicate that the duration of symptoms before presenting for treatment has little influence upon the operability of the patient. Of those with a history of symptoms of less than one month, approximately the same proportion of patients were operable and inoperable. This similarity is evident in each of the other duration groups.

The site of the tumour within the stomach proved, in this series, to be a major factor in assessing operability (Table 88). In the earlier years of the review carcinoma of the proximal portion of the stomach was considered to be non-resectable and only in those cases in which obstruction was present was operation contemplated, and then only a jejunostomy or gastrostomy was performed. In the later years, lesions in this site were no longer considered to be non-resectable and consequently surgical exploration was performed in an increasing number of cases with gastric resection in view (Table 89). Despite the increase in operability rate of lesions of the cardia, however, there is still a significant statistical difference between that and the operability rate of lesions of other sites in the stomach.

The relationship between operability and the type of tumor is presented in Table 90. There is no statistical significance between the operability rates in any of the individual types of lesions. Polypoid lesions are usually considered to be eminently suitable for operation, but this was not apparent in

Operability	All Cases (per cent)	Intervals, in months, between onset of symptoms to treatment				
		Less Than One Month (per cent)	1 - 3 (per cent)	4 - 6 (per cent)	7 - 12 (per cent)	13 and over (per cent)
All Cases	100.0	2.8	31.4	22.8	24.0	19.0
Operable	100.0	2.6	31.8	22.1	22.4	21.0
Inoperable	100.0	3.1	30.8	23.8	25.9	16.4

GASTRIC CANCER: OPERABILITY RELATED TO THE INTERVAL BETWEEN THE ONSET OF SYMPTOMS AND TREATMENT

SASKATCHEWAN 1932-55

TABLE 87

	All Cases		Unstated		Pre-pylorus		Lesser curvature		Antrum		Cardia		Greater curvature & fundus		Body		Whole Stomach		Post. Wall		Ant. Wall	
	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability	Number of Cases	% Operability
Total	2000	-	89	-	661	-	405	-	210	-	186	-	158	-	137	-	111	-	30	-	13	-
Not Stated	5	-	-	-	-	-	2	-	2	-	-	-	-	-	-	-	-	-	-	-	1	-
Total Stated	1995	-	89	-	661	-	403	-	208	-	186	-	158	-	137	-	111	-	30	-	12	-
Operable	1189	59.5	31	34.8	451	68.2	245	60.8	116	55.5	85	45.7	93	58.9	66	48.2	64*	57.7	27	90.0	10	83.3

*Includes 4 cases in which cancer was evident at multiple sites in stomach

GASTRIC CANCER: OPERABILITY RELATED TO SITE OF TUMOUR

SASKATCHEWAN 1932-55

TABLE 88

Years	Operability Rates (per cent)	
	Cardia	Body
1932 - 36	35.3	9.1
1937 - 41	27.3	0.0
1942 - 46	31.0	30.8
1947 - 51	50.9	55.8
1952 - 55	57.9	61.4

GASTRIC CANCER: OPERABILITY OF LESIONS OF THE CARDIA AND BODY OF THE STOMACH

SASKATCHEWAN 1932-55

TABLE 89

	All Cases		Type of Tumour											
			Unstated		Ulcerating		Infiltrating		Polypoid		Linitis Plastica		"Ulcer Cancer"	
	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)
Total	2000	-	1014	-	566	-	229	-	140	-	37	-	14	-
Not stated	5	-	-	-	2	-	3	-	-	-	-	-	-	-
Total Stated	1995	-	1014	-	564	-	226	-	140	-	37	-	14	-
Operable	1189	59.5	464	45.7	414	73.4	179	79.2	98	70.0	20	54.0	13	92.9

GASTRIC CANCER: OPERABILITY RELATED TO TYPE OF TUMOUR

SASKATCHEWAN 1932-55

TABLE 90

the present series. It appeared pertinent to enquire why there should be this deviation from the general pattern. Had the site of the polypi been predominantly in the body and cardia of the stomach in which the operability rate has already been shown to be low, the explanation would have been obvious. Table 91 demonstrates, however, that in only 14.3 per cent of the patients, the polypi were situated in these regions. In 66.7 per cent of those cases in which the reason for inoperability was stated, the lesions were thought to be too advanced for surgery (Table 92). There may be a relationship between this clinical impression and the fact that fifty per cent of the patients on whom no operative procedure was performed and in whom the gastric lesion was polypoid in type, there was an associated palpable epigastric mass. The presence of a palpable mass is seen from Table 93 to lead to a lower operability rate irrespective of the type of gastric lesion present. This difference in operability rate between those patients with and without a palpable mass is statistically significant.

Table 94 demonstrates that the operability rate varies inversely with the size of the gastric lesion. In the larger lesions the most commonly occurring stated reason for inoperability was that the disease had extended beyond the bounds of surgical excision (Table 95).

	Site of Polypoid Lesions									
	Pre-pylorus	Antrum	Lesser curvature	Greater curvature and fundus	Anterior Wall	Posterior Wall	Body	Cardia	Whole Stomach	Un-stated
Number	39	9	34	25	2	4	9	11	4	3
Per cent	27.9	6.4	24.3	17.9	1.4	2.9	6.4	7.9	2.8	2.1

GASTRIC CANCER: SITE OF POLYPOID LESIONS

SASKATCHEWAN 1932-55

TABLE 91

Reason for Inoperability	Per Cent
Unstated	25.7
Lesion too advanced	66.7
Unsuspected cancer	3.8
Patient refused treatment	3.8

GASTRIC CANCER: REASONS GIVEN FOR INOPERABILITY IN POLYPOID LESIONS

SASKATCHEWAN 1932-55

TABLE 92

	All Cases		Data on palpable mass not documented		With palpable mass		Without palpable mass	
	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)	Number of Cases	Operability (per cent)
Total	2000		77		544		1379	
Not Stated	5		-		-		5	
Total Stated	1995		77		544		1374	
Operable	1189	59.5	46	59.7	255	46.8	887	64.6
Inoperable	806	40.5	31	40.3	289	53.2	487	35.4

GASTRIC CANCER: OPERABILITY IN PATIENTS WITH PALPABLE MASS

SASKATCHEWAN 1932-55

TABLE 93

Size of Lesion (Diameter Centimetres)	Number of Cases in which the size of the lesion was documented	Operability (Per cent)
0 - 2.0	45	86.7
2.1 - 4.0	77	85.7
4.1 and Greater	474	70.7

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SIZE OF THE GASTRIC LESION AND OPERABILITY

SASKATCHEWAN 1932-55

TABLE 94

Reasons Stated for Inoperability	Size of lesion (cms. - diameter)					
	0 - 2.0		2.1-4.0		4.1 and greater	
	Number of Patients	Per cent	Number of Patients	Per cent	Number of Patients	Per cent
Total not operated upon	6	13.3	12	14.3	129	29.3
Patients refused treatment	-	-	3	25.0	7	5.4
Previous treatment elsewhere satisfactory	-	-	1	8.3	1	0.8
Patient went elsewhere for treatment	1	16.7	-	-	8	6.2
Treatment elsewhere now with inoperable recurrence	-	-	1	8.3	2	1.6
Referred elsewhere	-	-	-	-	3	2.3
Concurrent disease	-	-	1	8.3	2	1.6
Too advanced disease	3	50.0	5	41.8	80	62.0
Mistaken diagnosis thought to be benign ulcer	-	-	-	-	5	3.8
Unsuspected cancer	1	16.7	-	-	-	-
Unstated	1	16.7	1	8.3	19	14.8

GASTRIC CANCER: REASONS GIVEN FOR INOPERABILITY IN 147 PATIENTS, RELATED TO THE SIZE OF THE GASTRIC LESION

SASKATCHEWAN 1932-55

TABLE 95

RESECTABILITY

Gastric cancer is a curable disease provided the lesion is localized to the stomach wall and adequate gastric resection is performed. Salvage is also possible in those cases in which the first echelon of perigastric lymphatic glands is involved in tumour spread if complete extirpation of the tumour tissue has been accomplished by resection of the stomach combined with a block dissection of the regional lymphatic tissue. Long periods of survival result from extensive surgical excision of the stomach and adjacent organs along with block dissection of the first and second echelons of the lymphatic drainage in patients in whom the tumour has spread more widely.

The resectability of a gastric lesion can be determined only if a laparotomy is performed and the extent of the local disease defined. The lesion may be considered non-resectable because of its local extent and fixation. In other cases resection may be possible, but not indicated because of widespread peritoneal or liver metastases. It may be considered justifiable to perform palliative gastric resections in some of these patients in order to provide symptomatic improvement by relieving obstruction at the pylorus or the cardia and to reduce the degree of anaemia. In these cases, despite the symptomatic relief obtained by the removal of the primary tumour, the course of the disease will progress as

surely as though no surgical procedure had been performed. In other cases there may be no gross evidence of metastases and the local lesion may be suitable for resection, but the general physical condition of the patient may be considered to be too poor to permit the performance of a major surgical procedure. A quarter of a century ago this was considered to be a valid reason for performing palliative gastro-enterostomy in preference to resection, but with the advance in the understanding of preoperative and postoperative supportive therapy, the prevention and control of respiratory infection and the improved methods of anaesthetic technique, fewer patients are now denied definitive treatment on this account. Age is no longer an important factor in determining whether a patient should have gastric resection. Many patients over the age of eighty years are in satisfactory general condition and may be better surgical risks than others twenty years their junior. No longer must the decision against gastric resection be made merely because a patient has reached an age where his expectation of life, as calculated by actuarial methods, is considered to be short.

It is commonly assumed that concomitant with increased resectability rates there must be an improvement in the five year survival rates. Pack (1946) stated that if the general average rate of resectability was materially increased a large increase in resultant long term cures would be certain and that, if those who

did not resect the stomach for cancer could be stimulated to do so "the total number of definitive cures would at once triple or quadruple the number now obtained." From the theoretical point of view increased resectability should result in a greater number of cured patients, but there are certain factors which might prevent the theoretical expectation being proved in practice.

Gastric resection alone will cure only those individuals whose lesions are localized to the stomach wall. Where extra-gastric tumour spread has occurred a procedure greater in extent than removing part, or even all, of the stomach is necessary before salvage can be anticipated. Therefore, the plea for an increased resectability rate should be accompanied by an equally strong plea for adequate en bloc excision of the perigastric lymphatic channels and glands.

A call for more, and wider, resections cannot be made without making recommendation regarding the basic qualifications of the surgeons who should perform these procedures. Welch in 1885 stated that gastric resection for cancer was not an operation for the casual surgeon, but that its successful accomplishment was dependent upon a high degree of specialisation, and required the cooperation of a specially trained surgical team. This is still true. The poorly trained or inexperienced surgeon may not have the technical ability to perform the type of radical surgery required in gastric cancer, and his effort to fulfil the requirements of the

definition of an adequate procedure may result in an unacceptably high operative mortality.

In the Saskatchewan series the overall resectability rate for the period 1932-1955 inclusive was 28.9 per cent. There was a steady increase in the rate from 15.2 per cent in the period 1932-1936 to 39.7 per cent between 1952 and 1955. In the latter year the resectability rate was 45.6 per cent (Table 96). It is not within the design of this monograph to prophesy what the outcome of the increased resectability in the last few years will be, but if pathological reports concerning the number of lymph nodes removed with the resected stomachs are any indication of the completeness of the operation done substantial improvement in the five year survival rate is not anticipated. In many specimens there are as few as three lymph nodes present, and the average is in the region of eight. This would indicate that most of the operative effort has been expended in removing the stomach, and too little in extirpating, at least, the first echelon of lymph glands. The medical literature does not lack reports on the increasing resectability rates from centres over the world. [Wilson (1950), Cooper (1952), Jemerin and Colp (1952), State et al (1947)] . Table 97 demonstrates that the sex of the patient has no significant relationship to resectability.

From the data presented in Table 98 it is evident that there is a diminishing reluctance on the part of the surgeons of

Years	Total Number of Patients	Number With Gastric Resection	Number Without Gastric Resection	% Resect-ability (total cases)	% Resect-ability (Operable cases)
All years	2000	578	1422	28.9	48.6
1932-36	138	21	117	15.2	38.9
1937-41	204	42	162	20.7	44.7
1942-46	395	87	308	22.0	43.9
1947-51	686	199	487	29.0	46.9
1952-55	577	229	348	39.7	54.7

GASTRIC CANCER: RESECTABILITY RATES

SASKATCHEWAN 1932 - 55

TABLE 96

Year	Both Sexes		Male		Female	
	Number of Resectable Cases	% Resectability	Number of Resectable Cases	% Resectability	Number of Resectable Cases	% Resectability
All years	578	28.9	443	29.5	135	26.6
1932 - 36	21	15.2	13	13.0	8	21.1
1937 - 41	42	20.7	35	22.0	7	15.9
1942 - 46	87	22.1	67	22.3	20	21.7
1947 - 51	199	29.0	152	30.3	47	25.4
1952 - 55	229	39.7	176	40.4	53	36.1

GASTRIC CANCER: THE RELATIONSHIP BETWEEN RESECTABILITY AND THE SEX OF THE PATIENT IS NOT STATISTICALLY SIGNIFICANT

SASKATCHEWAN 1932-55

TABLE 97

Years	All Ages		Age in Years							
			Less than 45		45-54		55-64		65 and over	
	Number of Resections	Resectability (Per Cent)	Number of Resections	Resectability (Per Cent)	Number of Resections	Resectability (Per Cent)	Number of Resections	Resectability (Per Cent)	Number of Resections	Resectability (Per Cent)
All years	578	28.9	48	35.8	97	36.8	196	32.2	237	23.7
1932 - 36	21	15.2	3	23.1	6	18.2	7	13.2	5	12.8
1937 - 41	42	20.7	5	35.7	16	33.3	14	19.2	7	10.3
1942 - 46	87	22.1	10	29.4	16	29.6	35	25.6	26	15.5
1947 - 51	199	29.0	22	52.4	28	40.6	66	33.9	83	21.8
1952 - 55	229	39.7	8	29.3	31	55.3	74	50.3	116	33.7

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE AGE OF THE PATIENT AND RESECTABILITY

SASKATCHEWAN 1932-55

TABLE 98

the province to perform gastric resection on patients in the older age groups.

There is no relationship evident between the duration of symptoms and resectability. In each of the duration groups a similar proportion of non-resectable and resectable neoplasms was found (Table 99). Any differences which might appear to exist do not satisfy the tests for statistical significance.

There is a very definite relationship between resectability and the site of the tumour within the stomach (Table 100). With the exception of the last few years of the period studied, carcinoma of the cardia was considered non resectable. Until 1953 the resectability rate for lesions in this site was 8.5 per cent. During the years 1954 and 1955 there was a marked increase to 27.3 per cent resectability in the thirty-three cases which occurred during that time, giving an overall average rate of 11.8 per cent. In these later years the advantage of the combined abdomino-thoracic approach to the proximal part of the stomach was realised and an increasing proportion of tumours in that region were successfully extirpated either by oesophago-gastrectomy or total gastrectomy. Large tumours which involved most or all of the stomach were no longer regarded as hopeless by a few surgeons and total gastrectomy was employed by them, in the later years on more frequent occasions than formerly. The resectability rate of lesions of the cardia is statistically

Resectability	All Cases	Interval in Months Onset of Symptoms to Treatment				
		Less than 1 month	1 - 3	4 - 6	7 - 12	13 +
All cases	100.0	2.9	31.4	23.0	24.0	18.7
Resectability	100.0	2.0	28.6	26.1	21.5	21.8
Non Resectability	100.0	3.2	32.5	21.8	24.9	17.6

GASTRIC CANCER: THE RELATIONSHIP BETWEEN DURATION OF SYMPTOMS BEFORE TREATMENT AND RESECTABILITY

SASKATCHEWAN 1932 - 55

TABLE 99

	All Cases		Site of Tumour																			
			Site Unstated		Pre-pylorus		Lesser Curv.		Antrum		Cardia		Gt. curv. & fundus		Body		Whole Stomach		Post. Wall		Ant. Wall	
	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%	Number of cases	%
Total	2000		89		661		405		210		186		158		137		111		30		13	
Not stated	3		-		-		2		1		-		-		-		-		-		-	
Total stated	1997		89		661		403		209		186		158		137		111		30		13	
Resectable	578	28.9	13	14.6	256	38.7	117	29.0	60	28.7	22	11.8	43	27.2	27	19.7	17	15.4	16	53.3	7	53.9

GASTRIC CANCER: RESECTABILITY RELATED TO SITE OF TUMOUR

SASKATCHEWAN 1932-55

TABLE 100

significant in comparison with lesions in other sites.

There was a lower resectability rate in polypoid lesions than might have been expected (Table 101). There may have been an association between the bulky nature of some of these tumours and the decision that the lesions could not be removed. There was a statistically significant difference between the resectability rates in patients with and without palpable mass (Table 102). The rate varied inversely with the size of the lesion (Table 103).

It is of interest to compare the records of the various surgeons who assume the responsibility for the operative care of patients with cancer of the stomach in respect of resectability. For obvious reasons these surgeons have not been named, but each has been given a code number in order that their identity may be preserved for this record. These surgeons have been divided into three groups according to the number of operative procedures done by them for gastric cancer. These surgeons performing less than ten operative procedures are not individualised, but are combined with the resectability record of those surgeons outside the province who undertook the treatment of some of the patients reviewed (Table 105). Table 104 explains the manner in which the remaining surgeons have been categorised. It will be noted that the range of resectability rate for those surgeons who performed twenty or less operative procedures is wide, from

	All Cases		Type of Tumour											
			Type Unstated		Ulcerating		Infiltrating		Polypoid		Linitis		"ulcer cancer"	
	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)
Total	2000		1012		567		230		140		37		14	
Not Stated	3		-		1		2		-		-		-	
Total Stated	1997		1012		566		228		140		37		14	
Resectable	578	28.9	88	8.7	284	50.2	121	53.1	69	49.3	5	13.5	11	78.6

GASTRIC CANCER: RESECTABILITY RELATED TO THE TYPE OF THE TUMOUR

SASKATCHEWAN 1932-55

TABLE 101

Resectability	All Cases		Data on Palpable Mass not Documented		With Palpable Mass		Without Palpable Mass	
	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)	Number of Patients	Resectability (Per cent)
Total	2000		78		544		1378	
Not Stated	3		-		-		3	
Total Stated	1997		78		544		1375	
Resectable	578	28.9	27	34.6	87	16.0	464	33.7
Non resectable	1419	71.1	51	65.4	457	84.0	911	66.3

GASTRIC CANCER: THE RELATIONSHIP BETWEEN RESECTABILITY AND THE PRESENCE OF A PALPABLE MASS

SASKATCHEWAN 1932 - 55

TABLE 102

Size of Lesion (Diameter in Centimetres)	Number of Cases	Resectability (Per cent)
0 - 2.0	43	83.7
2.1 - 4.0	73	68.5
4.1 and greater	505	50.7

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SIZE OF THE GASTRIC LESION AND RESECTABILITY IN THE 621 PATIENTS IN WHOM THE PERTINENT DATA WERE AVAILABLE

TABLE 103

Surgeon (Code)	Resectability (Per cent)
Those Performing Between 10 and 20 Gastric Procedures	
13	55.0
15	52.2
24	26.9
25	17.6
27	22.2
29	52.4
32	60.0
47	38.9
53	58.9
55	41.2
ThosePerforming Between 21 and 50 Gastric Procedures	
12	50.0
17	43.4
26	51.2
43	68.8
45	52.8
54	57.6
59	55.8
Those Performing More Than 50 Gastric Procedures	
03	40.9
07	45.8
35	47.4

GASTRIC CANCER: THE RESECTABILITY RATE OF INDIVIDUAL SURGEONS WHO PERFORMED MORE THAN TEN OPERATIVE PROCEDURES FOR GASTRIC CANCER

SASKATCHEWAN 1932-55

TABLE 104

Number of Operative Procedures	Number of Resections of Stomach	Resectability (Per cent)
362	174	48.1

GASTRIC CANCER: RESECTABILITY RATE IN THOSE PATIENTS TREATED BY SURGEONS OUTSIDE THE PROVINCE, AND BY THOSE IN THE PROVINCE WHO HAD, DURING THE INTERVAL OF TIME STUDIED, PERFORMED FEWER THAN TEN GASTRIC PROCEDURES

TABLE 105

17.6 per cent to 60.0 per cent. The average resectability rate for the group is 42.5 per cent. For those performing more than twenty, but less than fifty gastric procedures the range of resectability rate is smaller, with only one less than fifty per cent. The average for this group is 54.2 per cent. The three surgeons in the province who performed more than fifty gastric procedures during the interval of time studied have an average resectability rate of 44.7 per cent. It might have been expected that these three would have had a better resectability rate than the less experienced surgeons in the other groups, but it has been apparent from the data on the patients charts that their record in respect of resectability suffers because of the advanced state of the disease in some patients who were referred to them in the hope that, by virtue of their greater experience, greater chance for cure could be anticipated. The number of patients treated by these surgeons also reflects the length of time they have been in practice in the province and also the size of their practice (Table 104).

OPERATIVE MORTALITY

Operative mortality is of primary importance in that group of patients in whom gastric resection could appear to hold a reasonable chance of cure. To a certain degree it is under the control of the surgeon. By the judicious selection of patients, by adequate preoperative and postoperative supportive therapy, by intelligent anticipation of complications and their prevention, by

the choice of an expert anaesthetist and by the exhibition of reasonable operative skill, the risk consequent upon subtotal gastrectomy for gastric cancer should be within the limits of five percent. This is an arbitrary level of risk which may be modified by the standard of resectability set by the individual surgeon. Low rates of operative mortality following resection may not necessarily mean that the standard of surgical skill has been high, but may be an indication that there has been a very strict selection of the patients upon whom the operative procedure has been performed. If resection is reserved for those patients in whom the lesion is small, confined and localized to an easily accessible part of the stomach, the operative mortality rate in practiced hands should be negligible. If, however, the dominant thought of the surgeon is to extend the benefits of excisional surgery to the maximum number of patients, and especially to those of borderline resectability, the operative risk to the patient, even when the greatest surgical skill has been employed, will be comparatively high.

There are many surgeons who believe that any post-resection mortality risk is justifiable because of the inevitable mortality associated with the disease when it is left untreated and who are satisfied that a definitive cure more than balances even a high postoperative loss of life. This philosophy is permissible only when it is held in regard to those cases in

which the disease is not readily resectable and should not be offered as an excuse by the less skilled surgeon for a high operative death rate in the more readily resectable cases. In the latter group every operative death must be carried forward as a debit against the number of years of life which the patient might have enjoyed had the resection been performed by a more experienced surgeon. There is a very obvious difference between offering a patient a twenty per cent chance of five year survival at a five per cent post resection risk, and a five per cent chance of survival for the same period at a twenty per cent operative risk. In those cases where lesions are of border line resectability, a high post resection mortality rate is justified because any definitive cure represents true salvage.

The operative mortality rate in the Saskatchewan series has been calculated on the basis of the number of patients who died within a period of thirty days after operation or where deaths occurred in hospital after thirty days as a direct or indirect result of their treatment. During the twenty-four years under review one fifth of all patients who were operated upon died during this defined postoperative period (Table 106). A steady decline in the operative mortality rate is evident, but remains high at the level of 13.8 per cent during the last four years of the study.

Year	Total cases Operated Upon	Post-Operative Deaths	
		Number	Per cent
All Years	1189	238	20.0
1932 - 36	54	16	29.6
1937 - 41	94	33	35.1
1942 - 46	198	42	21.2
1947 - 51	424	89	21.0
1952 - 55	419	58	13.8

GASTRIC CANCER: POSTOPERATIVE MORTALITY RATE

SASKATCHEWAN 1932-55

TABLE 106

The operative mortality following gastric resection is formidable even during the last few years of the review period. (Table 107). The overall rate was 17.1 per cent. While there was a considerable reduction in the mortality rate from the period 1937-41 in which nineteen patients of a total of 42 resected died (45.1 per cent) to 28 deaths in 229 patients who had gastric resection in the period 1952-55 (12.2 per cent) it is noted that in the year 1954 the rate was 16.4 per cent, a figure no better than the record of the period 1942-46 in which 14 deaths occurred after 87 resections (16.1 per cent). Of the 99 post resection deaths, twenty-two were found to have no extra gastric spread of the disease. These represent a very definite loss of salvage.

Table 108 demonstrates an interesting change in the distribution of deaths after operation for gastric cancer. In the succeeding groups of years since 1942 a marked reduction in the proportion of deaths within the first ten postoperative days occurred with a corresponding increase in the proportion of deaths after the tenth day. The lack of progress notes on the patients' hospital charts, in many cases, has made the study of the causes of death impossible. It is noted, however, that in the period 1942-46 there was a high incidence of death within the first twenty-four hours after operation, possibly indicative of the lack of preoperative and immediate postoperative supportive therapy.

Year	Total Number of Resections	Postoperative Deaths	
		Number	Per cent
All Years	578	99	17.1
1932 - 36	21	8	38.1
1937 - 41	42	19	45.1
1942 - 46	87	14	16.1
1947 - 51	199	30	15.1
1952 - 55	229	28	12.2

GASTRIC CANCER: POSTOPERATIVE DEATHS IN PATIENTS UPON WHOM GASTRIC RESECTION WAS PERFORMED

SASKATCHEWAN 1932-55

TABLE 107

Year	Total Number of Deaths (100.0 %)	Deaths (per cent) Occurring in Various Post-operative Periods Expressed in Days							
		1	2	3	4	5 - 9	10 - 19	20 - 29	30 and over
All years	238	6.7	7.6	4.6	8.0	21.8	19.8	17.2	14.3
1932-36	16	-	6.2	6.2	12.5	31.3	18.8	6.2	18.8
1937-41	33	6.0	12.1	3.0	21.2	27.3	15.2	9.1	6.1
1942-46	42	16.7	4.8	4.8	11.9	21.4	11.9	9.5	19.0
1947-51	89	5.6	6.7	6.7	2.2	21.4	23.6	21.4	12.4
1952-55	58	3.4	8.6	1.9	5.2	17.2	22.4	24.1	17.2

GASTRIC CANCER: DISTRIBUTION OF POST-OPERATIVE DEATHS ACCORDING TO THE NUMBER OF DAYS AFTER OPERATION

SASKATCHEWAN 1932-55

TABLE 108

It has been possible to determine with reasonable accuracy the surgeons' impressions on whether the gastric resection performed was of a palliative or "curative" nature only during the years 1954 and 1955. In previous years the descriptions by the surgeons of the operative findings were such that in most cases no assessment regarding the postoperative prognosis could be made. Table 109 reveals that during these two years the loss of salvage due to post resection mortality was respectively 16.3 per cent and 5.4 per cent in those patients in whom a reasonable chance for cure appeared to be possible.

The relationship between postoperative mortality and the individual surgeons who performed more than ten gastric procedures is seen in Table 110. The postoperative mortality data for those surgeons performing less than ten gastric procedures and by those surgeons outside the province is given in Table 111.

Comparison of the data in Table 104 and 110 reveals no evidence which would support the theory that with increased experience of performing gastrectomy the postoperative mortality decreases.

INVOLVEMENT OF THE LINES OF RESECTION

Reference has already been made (Chapter 6 - General Features) to the frequency with which cancer of the stomach extends into the oesophagus and duodenum and attention

Year	Number of Resections		Post-operative Deaths			
	Palliative	"Curative"	Palliative		"Curative"	
			Number	Per cent	Number	Per cent
1954	12	43	2	16.7	7	16.3
1955	12	56	4	33.3	3	5.4

GASTRIC CANCER: POST-OPERATIVE DEATHS ON WHOM PALLIATIVE AND "CURATIVE" GASTRIC RESECTIONS WERE PERFORMED DURING THE YEARS 1954 AND 1955

TABLE 109

Surgeon (Code)	Number of abdominal explora-tions performed	Post-operative mortality (per cent)	Number of gastric procedures performed	Post-operative mortality (per cent)	Number of gastric resections performed	Post-operative mortality (per cent)
13	20	25.0	13	-	10	-
15	23	21.7	17	17.6	12	16.7
24	26	11.5	18	16.7	7	42.9
25	17	52.9	12	75.0	9	11.1
27	18	22.2	10	30.0	4	-
29	21	23.8	16	25.0	11	18.2
32	10	-	10	-	6	-
47	18	11.1	13	14.6	7	-
53	17	-	12	-	10	-
55	17	23.5	13	23.1	7	14.3
12	48	27.1	39	28.2	24	16.7
17	53	15.1	41	12.2	23	8.7
26	43	34.9	33	42.4	22	45.5
43	48	12.5	38	13.2	33	9.1
45	36	25.0	26	30.8	19	31.6
54	33	18.2	29	17.2	19	15.8
59	43	23.3	33	24.2	24	20.8
03	93	21.5	76	22.4	38	18.4
07	142	19.7	95	17.9	65	15.4
35	116	13.8	78	12.8	55	10.9

GASTRIC CANCER: THE RELATIONSHIP BETWEEN POST-OPERATIVE MORTALITY AND THE INDIVIDUAL SURGEONS WHO PERFORMED MORE THAN TEN GASTRIC PROCEDURES

SASKATCHEWAN 1932-55

TABLE 110

Number of abdominal explorations performed	Post-operative mortality (per cent)	Number of gastric procedures performed	Post-operative mortality (per cent)	Number of gastric resections performed	Post-resection operative mortality (per cent)
362	20.6	253	20.2	172	19.2

GASTRIC CANCER: THE POST-OPERATIVE MORTALITY IN THOSE PATIENTS TREATED BY SURGEONS OUTSIDE THE PROVINCE AND BY THOSE IN THE PROVINCE WHO HAD, IN THIS SERIES, PERFORMED FEWER THAN TEN GASTRIC OPERATIVE PROCEDURES

TABLE 111

drawn to the difficulty, other than by histological examination of the tissue, of determining whether these adjacent parts of the gastro-intestinal tract are involved in the tumour process. The same difficulty may be experienced in determining to what extent the neoplasm extends within the stomach wall. To overcome this difficulty it has been accepted practice to resect the stomach at least two inches beyond visible and palpable evidence of tumour. Zinninger and Collins (1949) found that although great variation in the extent of microscopic invasion of the gastric wall beyond the grossly palpable tumour existed, there was rarely any spread beyond one inch from the margin of the primary tumour. Amesti (1943), found that in forty of one hundred cases examined by him, carcinoma infiltrated intramurally three to four centimetres beyond palpable evidence of the local tumour. Verbrugghen (1934) found that the Broder method of grading tumours gave a rough indication of the extent to which a given cancer would be likely to invade the surrounding gastric wall. Most grade one and grade two cancers demonstrated no lateral extension while grades three and four tumours extended widely, the latter to a greater degree than the former.

The present writer (unpublished data) planned a study to determine whether the surgeon is ever justified in his confidence that he can judge, by gross inspection and

palpation, when he has resected the stomach extensively enough to avoid leaving residual tumour within the gastric wall. His series comprised 467 gastric cancers which had been resected. The group was not made up of consecutive cases, but the only criteria of selection used were that an assessment of the extent of the gastric resection performed had been made at the time of the operation and that the exact site of the primary lesion had been documented. In all cases it was the surgeon's opinion that the stomach had been resected widely enough to ensure no tumour involvement at the lines of resection. Histological examination of these lines of resection, made subsequent to the completion of the operation, proved that the expected margin of safety had not been realised in many cases (Tables 112 and 113).

In 201 cases the lesion was situated in the distal one quarter of the stomach, and in ninety-three per cent of these a resection greater than seventy-five per cent of the stomach had been performed. Of this group, nine cases (4.5 per cent) presented microscopic evidence of tumour spread within the proximal one quarter of the stomach, with one, from the prepyloric area extending into the oesophagus. In this latter case total gastrectomy had been performed in the belief that possibly this extensive procedure was the treatment of choice for all gastric carcinoma. A similar

Number of Cases	Gastric Resection Estimated Amount of Stomach Removed %	Number of Cases with Resection Lines Involved in Tumour	Involvement of Lines of Resection						Incidence of Cases With Involved Lines of Resection %
			Number of Cases	Incidence %	Number of Cases	Incidence %	Number of Cases	Incidence %	
			Proximal		Distal		Both Lines		
33	25 - 74	1	1	3.0	-	-	-	-	3.0
230	75 - 84	21	14	6.1	6	2.6	1	0.4	9.1
148	85 - 99	12	6	4.1	3	2.0	3	2.0	8.1
56	100	8	5	8.9	2	3.6	1	1.8	14.3
Total 467		42	26	5.6	11'	2.4	5	1.1	8.9

GASTRIC CANCER: THE INCIDENCE OF INVOLVEMENT OF THE LINES OF RESECTION IN A SERIES OF PATIENTS WITH CANCER OF THE STOMACH, FOLLOWING GASTRECTOMY OF VARIED EXTENT

TABLE 112

Number of Cases	Site of Lesion	Involvement of Lines of Resection			
		Number of Cases	Incidence %	Number of Cases	Incidence %
		Proximal		Distal	
142	Prepylorus	6	4.2	5	3.5
59	Antrum	3	5.1	1	1.7
143	Lesser Curvature	8	5.6	6	4.2
35	Body	2	5.7	1	2.9
19	Posterior Wall	2	10.5	1	5.3
39	Greater Curvature	5	12.5	0	0
24	Cardia	5	20.8	2	8.3
6	Fundus	0	0	0	0

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE LESION AND INVOLVEMENT OF THE LINES OF RESECTION IN A SERIES OF PATIENTS WITH CANCER OF THE STOMACH FOLLOWING GASTRECTOMY

TABLE 113

experience was encountered with an apparently localised lesion in the antrum for which total gastrectomy was done.

In thirty cases in which the tumour appeared to be confined to the proximal one quarter of the stomach and for which at least seventy-five per cent of the stomach was removed by oesophago-gastrectomy in eighteen cases, and by total gastrectomy in ten cases, microscopic evidence of tumour spread within the distal one quarter was evident in two cases. In one patient in whom a total gastrectomy had been performed for an apparently localised lesion in the cardia, duodenal involvement was found at the distal line of resection.

It is of interest to speculate on the methods by which lesions, which appear to be localised, may demonstrate tumour tissue spread at a line of resection far removed from it, and especially to consider those cases in which apparently localised lesions in the distal and proximal portions of the stomach may exhibit involvement of the oesophagus and duodenum respectively. Canonico (1950) postulates that there are three methods of extension of cancer within the gastric wall or to the duodenum or oesophagus. The first of these is by continuous lymphatic permeation by extension of cellular chains of cancer cells through the lymphatics until a favourable location for further development is reached. He states that the regional process may remain in contact with the primary growth, or may break connection, with

formation of apparently new nodules of tumour. The second method of extension may be by lymphatic embolism, or discontinuous metastatic dissemination. The third method may be by contiguity. All of these methods are possible because of the continuous lymphatic pool present in the gastric wall, and which has been described in a previous chapter.

Stout (1942, 1948) described a type of carcinoma of the stomach which is characterised by the growth starting in the submucosa and spreading superficially in the mucosa and submucosa without penetrating deeply into the muscularis until it has covered a considerable surface area. Hebbel (1950) described eleven cases of superficial carcinoma of the stomach similar to those described by Stout and to "le cancer gastrique, mucoerosif, a marche lente" of Gutmann (1939), the cancer in situ of Mallory (1940) and the mucosal carcinoma described by Konjetzny (1938). Some of these tumours cover broad expanses of the mucosa and may show shallow erosions or closely resemble benign penetrating ulcer in one area with the remainder of the tumour being confined to the superficial layers. Ewing (1940) expressed the opinion that this type of tumour may be an example of progressive multicentric carcinomatous origin.

The concept of multicentric origin of cancer is in conflict with that of the unicentric principle propounded by Conheim (1889). This concept is based on the belief that cancer

is the result of the centrifugal reproduction of a single cell nidus. Willis (1948) and Collins et al (1952) believe that this is not always the case, and that an obviously malignant lesion may represent the confluence of neighbouring, but independent, lesions. Willis states that gastric cancer arises often, not from a single focus, but progressively over a field of prepared mucous membrane of small or great area, and quotes the occasional discovery of multiple separate areas of diffusely infiltrating carcinoma of the stomach. Collins (1952) found multiple malignant lesions in twenty-six of 117 resected stomachs.

The observations made on the series of cases described (Tables 112 and 113) are interesting and extremely disquieting. They demonstrate that however radical a gastric resection may be, even to total gastrectomy the surgeon cannot feel confident that he has resected wide of the tumour tissue if he relies on inspection and palpation only. The necessity for microscopic examination of all lines of resection is evident in order to determine the adequacy of the resection performed.

In the Saskatchewan series of cases, of 578 cases who had gastric resection for cancer, eighty-two (4.1 per cent) were found to have involvement of one or both lines of resection by tumour. With increased rate of resectability there proved to be an increasing rate of involvement of lines of resection (Table 114).

Years	Number of Resections	Lines of Resection Involved	
		Number	Per cent
1932 -55	578	82	4.1
1932 - 36	21	1	0.8
1937 - 41	42	1	0.5
1942 - 46	87	14	3.5
1947 - 51	199	28	4.1
1952 - 55	229	38	6.6

GASTRIC CANCER: THE INCIDENCE OF INVOLVEMENT OF LINES OF RESECTION

SASKATCHEWAN 1932-55

TABLE 114

For the years 1954 and 1955, when it was possible to differentiate between palliative and "curative" resection, as estimated by the surgeon, the incidence of the lines of resection were calculated (Table 115). Of the "curative" resections, twelve had the proximal lines of resection involved and seven the distal line. In three cases both lines of resection were involved.

The relation between the site of the tumour within the stomach and the incidence of lines of resection involvement in those patients with "curative" resection is seen in Table 116.

Of those receiving "curative" resection during the last two years of the survey and who had involved lines of resection, five patients had no tumour involvement of the regional lymphatic glands. These patients were a definite loss to salvage. The larger the gastric lesion the more likely the lines of resection are to be involved (Table 117). Infiltrating tumours had a greater incidence of involved lines of resection (Table 118).

When the tumour is found in the lines of resection the removal of a further portion of stomach, duodenum or oesophagus is indicated. This can be done most conveniently at the time of the initial operation and requires the cooperation of a pathologist who can give a competent report on frozen sections of tissue removed from the resected edges. If quick section facilities are not available and if tumour involvement is only evident on examination of paraffin sections, re-operation with wider resection is mandatory.

Gastric Resection					
Curative			Palliative		
Number of Cases	Number With Involved Lines of Resection	Incidence of Involved Lines of Resection %	Number of Cases	Number With Involved Lines of Resection	Incidence of Involved Lines of Resection %
99	22	21.2	24	7	25.0

GASTRIC CANCER: THE INCIDENCE OF INVOLVEMENT OF THE LINES OF RESECTION IN "CURATIVE" AND PALLIATIVE GASTRIC RESECTION DURING THE YEARS 1954 AND 1955

TABLE 115

Site of Tumour	Number of Patients with Involvement of lines of Resection in "curative" group		
	Proximal	Distal	Proximal and Distal
Prepylorus	2	4	2
Antrum	-	1	-
Lesser Curvature	1	2	-
Greater Curvature and Fundus	1	-	-
Anterior Wall	1	-	-
Posterior Wall	1	-	-
Body	1	-	-
Cardia	2	-	1
Whole stomach including Multiple Cancers	3	-	-

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SITE OF THE TUMOUR IN THE STOMACH AND INVOLVEMENT OF THE LINES OF RESECTION IN PATIENTS WHO HAD "CURATIVE" GASTRIC RESECTION DURING THE YEARS 1954 AND 1955

TABLE 116

Lines of Resection	Size of Tumour (cms. diameter)			
	Not Stated	0 - 2	2 - 4	4 or Greater
Number in curative Resection	4	1	-	17
Number in palliative Resection	3	-	1	3

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE SIZE OF THE GASTRIC LESION AND INVOLVEMENT OF THE LINES OF RESECTION IN THOSE PATIENTS WHO HAD "CURATIVE" AND PALLIATIVE GASTRIC RESECTIONS DURING THE YEARS 1954 AND 1955

TABLE 117

Lines of Resection	Type of Tumour		
	Polypoid	Ulcerating	Infiltrating
Number in Curative Resections	1	7	14
Number in Palliative Resections	2	2	3

GASTRIC CANCER: THE RELATIONSHIP BETWEEN THE TYPE OF TUMOUR AND INVOLVEMENT OF THE LINES OF RESECTION IN THOSE PATIENTS WHO HAD "CURATIVE" AND PALLIATIVE GASTRIC RESECTIONS DURING THE YEARS 1954 AND 1955

TABLE 118

"I thought it no disgrace to let the world see where I failed of success, that those that come after me may learn what to avoid"

Richard Wiseman
Sergeant-chirurgeon
1621-1676

CHAPTER 10

SURVIVAL IN GASTRIC CANCER

A survey of the survival times of patients with cancer of the stomach provides an opportunity to study the natural course of the disease from the onset of the first symptom until death in those patients who have not been treated and also permits an assessment to be made of the degree to which the natural behaviour of the disease may be modified by the incident of treatment.

Although Cole has recently suggested that there may be a few exceptions to the rule, it may be assumed that the opinion expressed by Welch in 1885 "that there is no sufficient evidence to believe that cancer of the stomach has ever been completely destroyed by any natural process" is still true and that all patients who have gastric carcinoma and who do not receive treatment will die as a direct or an indirect result of the presence of the disease. In these patients the period of survival after the onset of symptoms

varies and appears to be controlled by certain factors. One of these is the altered nutrition of the patient which may be a direct result of the site of the tumour. A patient with unrelieved obstruction due to cancer at the cardia or the pylorus may die more rapidly than a comparable patient with a lesion of similar type and activity involving the body of the stomach because of his inability to transport food to the digestive and absorptive part of the intestinal tract. The vomiting which may be the concomitant of such obstruction, enhances the nutritional debility of the patient by adding to it the effects of dehydration and electrolyte imbalance.

Another factor may be the metabolic derangement consequent upon hepatic failure, the result either of destruction or replacement of the liver parenchyma by massive tumour metastases or changes in the liver tissue secondary to nutritional abnormalities. The grossly debilitated patient may die prematurely because he is more prone to develop other lethal complications such as bronchopneumonia, the latter being the immediate cause of death and the gastric tumour and its sequelae only contributing factors.

Many patients who have demonstrated no clinical or laboratory evidence of liver dysfunction die and at autopsy are found to have moderately small gastric lesions which are not causing obstruction nor undue haemorrhage and which have

not metastasised beyond the perigastric lymph glands. In these it is difficult to attribute the death to the gastric neoplasm and yet, in many, no other gross abnormality is found. None of the theories of toxic or humoral disturbances arising as a result of the presence of cancer in the body and which have been presented as the possible explanations of the deaths of some of these patients has been proved.

The nature of the tumour itself may be a contributing factor. The growth potential of the neoplastic cells comprising the primary gastric tumour is immeasurable, but its influence upon the prognosis or survival time may be apparent. It was on this basis that MacDonald (1954) based his philosophic thesis of biological predeterminism as a limiting factor in the curability of the disease.

The survival times of the patients with gastric carcinoma who were admitted to the cancer clinics between the years 1932 and 1950 permits an assessment to be made of the five year survival rate of the whole group.

The survival time, assessed from the date of onset of the first symptom, was determined in 1179 patients (Table 119). More than three quarters of them survived six months, almost half were dead within one year and almost seventy per cent had succumbed within two years. Less than six per cent of the total number of patients were alive five years after the onset of the

Survival period from date of onset of symptoms (Months)	Patients surviving specified periods of time from onset of symptoms							
	Total (1179 patients)		Received no treatment (726 patients)		Received only palliative treatment (174 patients)		Gastric resection performed (277 patients)	
	Number	%	Number	%	Number	%	Number	%
Less than 6 months	911	77.3	531	73.1	131	75.3	247	89.2
6 - 11.9	642	54.5	341	46.9	87	50.0	213	76.9
12 - 23.9	336	28.5	141	19.4	39	22.4	156	56.3
24 - 35.9	191	16.2	59	8.1	15	8.6	117	42.2
36 - 47.9	131	11.1	38	5.2	5	2.9	88	31.8
48 - 59.9	97	8.2	19	2.6	4	2.3	74	26.7
60 +	69	5.9	6	0.8	1	0.6	62	22.4

GASTRIC CANCER: THE SURVIVAL PATTERN IN THE 1179 PATIENTS IN WHOM THE DATE OF ONSET OF SYMPTOMS WAS KNOWN. THE TYPE OF TREATMENT WAS INDEFINITE IN TWO CASES

SASKATCHEWAN 1932-50

TABLE 119

first symptom. When no treatment, or only palliative procedures were given, there was a rapid loss of life amounting to more than one quarter of the patients in each of the first three years after the onset of symptoms. Seven patients survived five years from the onset of symptoms without the benefit of excisional surgery. When an effort to cure the patient was made by performing gastric resection, the rate of loss of life was slower, and more than twenty per cent survived five years. In this group the loss of 10.8 per cent occurring within the first six months included the patients who had died as a result of the operative procedure and not of the disease directly. This technical loss is reflected in the proportion who died during each of the succeeding survival periods specified.

It is customary to select the date of treatment as the starting point for the calculation of survival times. For the purposes of this monograph the date of admission to the clinics of the 1274 patients who were seen between 1932 and 1950 was the base line selected because, for practical purposes, it approximated to the date of treatment in those patients upon whom an operative procedure was done and also because it provided a convenient time from which to calculate the survival in those who were not treated.

Less than two fifths of the patients survived the first six months after admission to the clinic (Table 120). Only one fifth of them lived for more than one year and just over five per cent survived for five years.

Survival period from date of admission to clinics (Months)	Patients surviving each time interval	
	Number	Per cent
Less than 6 months	484	38.0
6 - 11.9	268	21.0
12 - 23.9	155	12.2
24 - 35.9	110	8.6
36 - 47.9	86	6.8
48 - 59.9	68	5.3
60 or more	67	5.3

GASTRIC CANCER: THE PROPORTION OF PATIENTS SURVIVING AT CERTAIN SPECIFIED PERIODS OF TIME FROM THE DATE OF TREATMENT (OR AFTER THE DATE OF THE FIRST ADMISSION TO THE CLINICS IN THOSE PATIENTS WHO RECEIVED NO TREATMENT. THE 1274 PATIENTS SEEN AT THE CLINICS BETWEEN 1932 AND 1950 ARE STUDIED

SASKATCHEWAN 1932-50

TABLE 120

Treatment	Total number of patients	Number of patients who survived five years	Five year survival rate (per cent)
Resection	304	60	19.7
Palliative	185	1	0.5
No treatment	785	6	0.8
Total	1274	67	5.3

GASTRIC CANCER: THE FIVE YEAR SURVIVAL RATE, CALCULATED FROM DATE OF TREATMENT, OR FROM THE DATE OF FIRST ATTENDANCE AT CLINICS, RELATED TO THE TYPE OF TREATMENT GIVEN

TABLE 120 (i)

The Saskatchewan five year survival rate of 5.3 per cent (Tables 120 and 121 (i)) contrasts with the published reports from other centres over the past five years (Table 121). The reasons for this difference may rest in the lack of selection of the patients which comprise the clinical material in this series, the philosophic attitude to the disease and its treatment by the profession within the province, or in the difference in the quality of the surgery performed.

There is no material difference in the survival patterns between the males and the females of the group (Table 122). Approximately the same proportion of the patients in each sex is found to have survived the various time intervals. The five year survival rate is similar at 5.2 per cent for males and 5.5 per cent for females.

When age and sex are considered it is evident that the greatest loss of survivors occurs during the first year after which the survival curve falls less steeply (Table 123). There is no significant difference in the survival rates between the age groups except for those patients over seventy years of age, which are significantly lower.

The fallacy of assuming that the date of onset of symptoms is synchronous with the onset of the disease is obvious to everyone who has had clinical experience of gastric cancer. The time of onset and the degree of severity of the symptoms bear little relationship to the stage of development of the local gastric lesion, its spread to contiguous viscera or to regional

Author	Reported Five-year Survival (Per cent)
Minnes et al (1936)	3.5
Pach and Livingstone (1940)	4.0
Harnett (1947)	4.0
Walters (1943)	5.0
Custer (1945)	5.6
Gray (1942)	6.2
Jemerin (1952)	6.4
State et al (1947)	6.6
Weese (1940)	7.6
Cooper (1952)	8.5
Ochsner (1953)	9.9
Jemerin (1952)	11.6
Barclay (1951)	12.2
Pach (1951)	12.3
Walters (1953)	14.0

GASTRIC CANCER: REPORTED FIVE - YEAR SURVIVAL RATES, CALCULATED ON THE BASIS OF ALL PATIENTS SEEN

TABLE 121

Survival period from date of admission to clinic (Months)	Patients surviving each time interval			
	Males (964 patients)		Females (310 patients)	
	Number	Per cent	Number	Per cent
Less than 6 months	367	38.1	117	37.7
6 - 11.9	209	21.7	59	19.0
12 - 23.9	123	12.8	32	10.3
24 - 35.9	90	9.3	20	6.5
36 - 47.9	68	7.1	18	5.8
48 - 59.9	51	5.3	17	5.5
60 +	50	5.2	17	5.5

GASTRIC CANCER: THE PROPORTION OF MALES AND FEMALES WHO SURVIVED AT CERTAIN SPECIFIED INTERVALS OF TIME AFTER TREATMENT (OR DATE OF CLINIC ADMISSION IN THOSE PATIENTS WHO HAD NO TREATMENT). TOTAL NUMBER OF PATIENTS 1274

SASKATCHEWAN 1932-50

TABLE 122

Survival period from date of admission to clinic (Months)	Proportion of patients surviving (per cent) in each age group (years)						
	Both sexes						
	All ages 1274 patients	25 - 34 21 patients	35 - 44 72 patients	45 - 54 189 patients	55 - 64 423 patients	65 - 69 228 patients	70 and over 339 patients
Less than 6 months	38.0	38.1	54.2	41.3	39.0	36.8	32.4
6 - 11.9	21.0	33.3	26.4	23.3	21.5	22.4	16.5
12 - 23.9	12.2	23.8	15.3	13.2	13.0	11.4	9.7
24 - 35.9	8.6	19.0	12.5	11.1	8.5	9.2	5.6
36 - 47.9	6.8	14.3	9.7	9.0	6.9	7.0	4.1
48 - 59.9	5.3	9.5	5.6	7.9	5.0	5.7	3.8
60 and more	5.3	9.5	5.6	7.4	5.0	5.7	3.8

GASTRIC CANCER: THE CRUDE SURVIVAL RATES BY AGE AND SEX, THE SURVIVAL TIME BEING MEASURED FROM THE DATE OF TREATMENT (OR IN THOSE PATIENTS IN WHOM NO TREATMENT WAS GIVEN, FROM THE DATE OF ADMISSION TO THE CLINIC). THE AGE OF ONE PATIENT WAS UNSTATED. ONE PATIENT, YOUNGER THAN 24 YEARS OF AGE, DIED WITHIN THE FIRST SIX MONTHS.

SASKATCHEWAN 1932-50

TABLE 123

Survival period from date of admission to clinic (Months)	Proportion of patients surviving (per cent) in each age group (years)						
	Males						
	All ages 964 patients	25 - 34 9 patients	35 - 44 52 patients	45 - 54 141 patients	55 - 64 312 patients	65 - 69 187 patients	70 & over 262 patients
Less than 6 months	38.1	55.6	55.8	44.7	38.5	34.8	32.4
6 - 11.9	21.7	55.6	28.8	27.0	21.8	21.4	16.4
12 - 23.9	12.8	33.3	15.4	14.2	14.7	11.2	9.5
24 - 35.9	9.3	33.3	11.5	11.3	10.3	9.1	6.1
36 - 47.9	7.1	22.2	7.7	8.5	8.0	7.5	4.2
48 - 59.9	5.3	11.1	1.9	7.1	5.4	5.9	4.2
60 or more	5.2	11.1	1.9	6.4	5.4	5.9	4.2

GASTRIC CANCER: THE CRUDE SURVIVAL RATE BY AGE AND SEX, THE SURVIVAL TIME BEING MEASURED FROM THE DATE OF TREATMENT (OR IN THOSE PATIENTS IN WHOM NO TREATMENT WAS GIVEN, FROM THE DATE OF ADMISSION TO THE CLINIC). ONE PATIENT, OMITTED FROM THE TABLE, WAS IN THE 0-24 YEAR AGE GROUP AND DID NOT SURVIVE SIX MONTHS

SASKATCHEWAN 1932 - 50

TABLE 123 (i)

Survival period from date of admission to clinic (Months)	Proportion of patients surviving (per cent) in each age group (years)						
	Females						
	All ages 310 patients	25 - 34 12 patients	35 - 44 20 patients	45 - 54 48 patients	55 - 64 111patients	65 - 69 41 patients	70 and over 77 patients
Less than 6 months	37.7	25.0	50.0	31.2	40.5	46.3	32.5
6 - 11.9	19.0	16.7	20.0	12.5	20.7	26.8	16.9
12 - 23.9	10.3	16.7	15.0	10.4	8.1	12.2	10.4
24 - 35.9	6.5	8.3	15.0	10.4	3.6	9.8	3.9
36 - 47.9	5.8	8.3	15.0	10.4	3.6	4.9	3.9
48 - 59.9	5.5	8.3	15.0	10.4	3.6	4.9	2.6
60 and over	5.5	8.3	15.0	10.4	3.6	4.9	2.6

GASTRIC CANCER: THE CRUDE SURVIVAL RATES BY AGE AND SEX, THE SURVIVAL TIME BEING MEASURED FROM THE DATE OF TREATMENT (OR IN THOSE PATIENTS IN WHOM NO TREATMENT WAS GIVEN, FROM THE DATE OF ADMISSION TO THE CLINIC ONE PATIENT, AGE UNSTATED, DID NOT SURVIVE SIX MONTHS

SASKATCHEWAN 1932-50

TABLE 123 (ii)

lymph glands. There can be no guarantee that a patient presenting for treatment on the first day of his first symptom will have a better chance of cure than another who may delay several months before seeking treatment. The concept that early diagnosis, measured from the date of onset of symptoms, will result in a high survival rate is as false as it is popular.

Several reports have appeared in the medical literature over the past five years which correlate the duration of symptoms with the prognosis. Swynnerton (1952) in a review of 375 cases noted that "an outstanding finding ---- is the marked prognostic significance of the length of history among patients treated with surgical resection. The greater the length of history, the better is the prognosis." Without presenting any clinical or statistical data to support his statement, MacDonald (1954) elaborated upon the observations made by Swynnerton, "Patients with progressively longer periods of delay, from the onset of symptoms to the time of exploration, enjoy increasingly better chances of resection and long term survival." Barclay (1952) studied the relationship between the duration of symptoms and the survival of 526 patients who had gastric cancer and concluded that when all the cases were considered, and not only those in whom resection of the stomach had been performed as in the Swynnerton group, duration had little influence on the survival time. Evidence has already been presented in this monograph to show that there is no relationship

between the duration of symptoms and operability and resectability rates in the unselected series of patients drawn from the Saskatchewan Cancer Clinics.

It is interesting to note the similarity in the proportion of survivors in all the duration groups except in the survival periods under six months and between six and twelve months (Table 124). In these two early groups it would appear on casual examination that the longer the duration of symptoms the greater is the proportion of survivors. This difference is not statistically significant. When the duration is longer than twelve months even this apparent trend is not evident. When the patients are grouped according to whether they received no treatment, palliative treatment or gastric resection and their survival compared with the duration of their symptoms there is no significant difference in survival between those with the short, intermediate and long durations of symptoms (Tables 125, 126, 127).

There is no statistically significant difference between survival and the site of the primary tumour within the stomach except in the comparisons between lesions of the cardia and other sites. This significant difference is probably related to the lower operability and resectability rates for lesions of the cardia rather than to any material difference in the behaviour or natural character of the disease in this site (Tables 128, 129, 130, 131).

Survival period from date of admission to clinic (Months)	Total (1274 patients)	Proportion of patients surviving (per cent) in each duration group(months)						
		Less than one month 35 patients	1 - 3 362 patients	4 - 6 265 patients	7 - 9 134 patients	10 - 12 162 patients	13 - 24 138 patients	25 - 178patients
Less than 6 months	38.0	22.9	33.7	40.4	32.1	45.1	40.6	42.1
6 - 11.9	21.0	11.4	17.7	22.6	14.9	23.5	23.2	28.1
12 - 23.9	12.2	11.4	8.6	12.8	8.2	11.7	15.2	19.7
24 - 35.9	8.6	8.6	6.6	7.9	3.7	8.6	12.3	14.6
36 - 47.9	6.8	8.6	5.2	5.7	3.0	5.6	9.4	12.9
48 - 59.9	5.3	8.6	4.4	4.9	2.2	3.7	8.7	8.4
60 and over	5.3	8.6	4.1	4.9	2.2	3.7	8.7	8.4

GASTRIC CANCER: THE CRUDE SURVIVAL RATES BY THE DURATION FROM THE ONSET OF SYMPTOMS TO FIRST TREATMENT (OR FROM ONSET OF SYMPTOMS TO THE DATE OF FIRST ADMISSION TO THE CLINIC IN THOSE PATIENTS WHO WERE NOT GIVEN TREATMENT

SASKATCHEWAN 1932-50

TABLE 124

Survival period from date of first admission to clinic (Months)	Total (782 patients)	Proportion of patients surviving (per cent) in each duration group		
		Short duration (0-2.9 months) (250 patients)	Intermediate duration (3 - 11.9 months) (357 patients)	Long duration (more than 20 months) (175 patients)
Less than 6 months	25.9	22.4	29.4	24.0
6 - 11.9	9.3	8.4	10.0	9.1
12 - 23.9	3.9	3.2	4.2	2.3
24 - 35.9	1.9	2.0	1.7	1.7
36 - 47.9	1.2	2.0	0.6	0.9
48 - 59.9	0.8	1.2	0.3	0.9
60 and over	0.8	1.2	0.3	0.9

GASTRIC CANCER: THE CRUDE SURVIVAL RATE CALCULATED FROM THE DATE OF FIRST ADMISSION TO CLINIC, OF PATIENTS WHO HAD NO TREATMENT, BY THE DURATION OF SYMPTOMS. THERE WERE SIX PATIENTS WHO SURVIVED FIVE YEARS WITHOUT TREATMENT. THREE WERE IN THE SHORT DURATION GROUP, ONE IN INTERMEDIATE, AND TWO IN LONG DURATION GROUP. THREE PATIENTS HAD INSUFFICIENT DATA REGARDING DURATION

SASKATCHEWAN 1932-50
TABLE 125

Survival period from date of first admission to clinic (Months)	Total (185 patients)	Proportion of patients surviving (per cent) in each duration group		
		Short duration (0 - 2.9 months) (68 patients)	Intermediate duration (3 - 11.9 months) (72 patients)	Long duration (more than 12 months) (45 patients)
Less than 6 months	38.4	35.3	36.1	46.7
6 - 11.9	13.5	10.3	11.1	22.2
12 - 23.9	4.3	4.4	1.4	8.9
24 - 35.9	1.1	-	-	4.4
36 - 47.9	0.5	-	-	2.2
48 - 59.9	0.5	-	-	2.2
60 and over	0.5	-	-	2.2

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO RECEIVED PALLIATIVE TREATMENT, BY THE DURATION OF SYMPTOMS. THERE WAS ONLY ONE FIVE YEAR SURVIVOR.

SASKATCHEWAN 1932-50

TABLE 126

Survival period from date of first treatment (Months)	Total	Proportion of patients surviving (per cent) in each duration group		
		Short duration (0 - 2.9 months)	Intermediate duration (3 - 11.9 months)	Long duration (12 months and more)
	304 patients	79 patients	130 patients	95 patients
Less than 6 months	68.4	63.3	70.0	70.5
6 - 11.9	55.6	50.6	56.9	57.9
12 - 23.9	37.8	30.4	36.9	45.3
24 - 35.9	30.3	27.8	26.2	37.9
36 - 47.9	25.0	21.5	20.0	34.7
48 - 59.9	20.1	20.3	16.2	25.3
60 and over	19.7	19.0	16.2	25.3

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO RECEIVED GASTRIC RESECTION DIRECTED AT CURE, BY THE DURATION OF SYMPTOMS. THERE WERE SIXTY FIVE YEAR SURVIVORS IN THIS GROUP, WITH THIRTEEN IN THE SHORT DURATION, TWENTY-ONE IN THE INTERMEDIATE AND TWENTY-SIX IN THE LONG DURATION GROUP.

SASKATCHEWAN 1932-50

TABLE 127

Survival period from first treatment or admission to clinic (months)	Total 1274 patients	Proportion of patients surviving (per cent) in each site group						
		Prepylorus 454 patients	Antrum 151 patients	Lesser curvature 262 patients	Greater curvature and fundus 86 patients	Cardia 130 patients	Other 154 patients	Unstated 37 patients
Less than six months	38.0	43.8	31.8	40.1	40.7	24.6	34.4	32.4
6 - 11.9	21.0	26.4	19.9	21.8	22.1	7.7	15.6	21.6
12 - 23.9	12.2	16.3	8.6	13.0	16.3	2.3	6.5	18.9
24 - 35.9	8.6	11.9	6.0	8.8	11.6	0.8	4.5	16.2
36 - 47.9	6.8	9.5	4.0	7.3	9.3	0.8	3.2	10.8
48 - 59.9	5.3	8.1	2.6	5.0	7.0	0.8	3.2	5.4
60 and over	5.3	8.1	2.6	4.6	7.0	0.8	3.2	5.4

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM FIRST TREATMENT (IN THOSE WHO HAD TREATMENT) OR FIRST ADMISSION TO THE CLINIC (IN THOSE WHO HAD NO TREATMENT) RELATED TO SITE. THERE WERE THIRTY-SEVEN WITH PREPYLORIC LESIONS WHO SURVIVED FIVE YEARS, FOUR PATIENTS WITH ANTRAL LESIONS, TWELVE WITH LESSER CURVATURE, SIX WITH GREATER CURVATURE, ONE WITH CARDIAC AND FIVE IN VARIOUS SITES ALSO SURVIVED FIVE YEARS. TWO PATIENTS IN WHOM THE SITE WAS NOT SPECIFIED SURVIVED FIVE YEARS

SASKATCHEWAN 1932-50

TABLE 128

Survival period from first admission to clinic (months)	Total 782 patients	Proportion of patients surviving (per cent) in each site group						
		Prepylorus 215 patients	Antrum 90 patients	Lesser curvature 172 patients	Greater curvature and fundus 62 patients	Cardia 100 patients	Other 114 patients	Unstated 29 patients
Less than six months	25.9	28.4	14.4	27.9	27.4	22.0	31.6	20.7
6 - 11.9	9.3	11.1	5.6	8.7	6.5	9.0	12.3	6.9
12 - 23.9	3.9	6.0	1.1	3.5	6.5	3.0	1.8	6.9
24 - 35.9	1.9	3.3	-	2.3	1.6	1.0	0.9	3.5
36 - 47.9	1.2	1.9	-	1.7	1.6	1.0	-	-
48 - 59.9	0.8	1.9	-	0.6	-	1.0	-	-
60 or more	0.8	1.9	-	0.6	-	1.0	-	-

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF FIRST ADMISSION TO THE CLINIC, OF PATIENTS WHO HAD NO TREATMENT, BY THE SITE OF THE LESION. SIX PATIENTS SURVIVED FIVE YEARS AFTER ADMISSION TO THE CLINICS, FOUR WITH LESIONS IN THE PYLORUS, ONE ON LESSER CURVATURE AND ONE AT THE CARDIA

SASKATCHEWAN 1932-50

TABLE 129

Survival period from date of treatment (months)	Total	Proportion of patients surviving (per cent) in each site group						
		Prepylorus	Antrum	Lesser curvature	Greater curvature and fundus	Cardia	Other	Unstated
	185 patients	93 patients	17 patients	30 patients	6 patients	23 patients	14 patients	2 patients
Less than six months	38.4	39.8	35.3	43.3	50.0	30.4	28.6	50.0
6 - 11.9	13.5	15.1	11.8	20.0	16.7	-	7.1	50.0
12 - 23.9	4.3	3.2	5.9	10.0	16.7	-	-	-
24 - 35.9	1.1	-	-	3.3	16.7	-	-	-
36 - 47.9	0.5	-	-	3.3	-	-	-	-
48 - 59.9	0.5	-	-	3.3	-	-	-	-
60 or more	0.5	-	-	3.3	-	-	-	-

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD ONLY PALLIATIVE TREATMENT, BY THE SITE OF THE LESION. ONE PATIENT, WITH A LESION ON THE LESSER CURVATURE, SURVIVED FIVE YEARS.

SASKATCHEWAN 1932-50

TABLE 130

Survival period from date of treatment (months)	Total	Proportion of patients surviving (per cent) in each site group						
		Prepylorus	Antrum	Lesser curvature	Greater curvature & fundus	Cardia	Other	Unstated
	304 patients	146 patients	43 patients	58 patients	18 patients	7 patients	26 patients	6 patients
Less than six months	68.4	69.2	67.4	72.4	83.3	42.9	50.0	83.3
6 - 11.9	55.6	56.2	53.5	60.3	77.8	14.3	34.6	83.3
12 - 23.9	37.8	39.7	25.6	41.4	50.0	-	30.8	83.3
24 - 35.9	30.3	32.2	20.9	29.3	44.4	-	23.1	83.3
36 - 47.9	25.0	26.7	14.0	25.9	38.9	-	19.2	66.7
48 - 59.9	20.1	22.6	9.3	19.0	33.3	-	19.2	33.3
60 or more	19.7	22.6	9.3	17.3	33.3	-	19.2	33.3

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD GASTRIC RESECTION DIRECTED AT CURE, BY THE SITE OF THE LESION. OF SIXTY PATIENTS WHO SURVIVED FIVE YEARS, THIRTY-THREE HAD LESIONS IN PREPYLORUS, FOUR IN THE ANTRUM, TEN IN LESSER CURVATURE, SIX IN GREATER CURVATURE AND FUNDUS, FIVE IN OTHER SITES AND TWO IN SITES UNSTATED

SASKATCHEWAN 1932-50

TABLE 131

At each survival period no significant difference between the type of tumour and the survival rate was found. (Tables 132, 133, 134, 135).

There was statistical significance between the survival of those patients with involved and those with uninvolved lymphatic glands. There was also significance found in the comparison of the survival of those patients with positive glands and of those in whom the data on glandular metastases were not available (Tables 136, 137, 138, 139). With lack of significance between those with negative glands and those in the unstated group, the inference was drawn that there was a preponderance of patients with negative nodes in the unstated group. (Tables 136, 137, 138, 139). Of the 159 patients in whom gastric resection was performed and in whom the regional lymphatic glands were involved in tumour only nine survived five years (5.7 per cent). There were twenty-four patients survived five years in the group of eighty-seven in whom gastric resection was performed and in whom the regional glands were not involved. These results contrast with those published from other centres.

Critical examination of the records of those patients in whom gastric resection was performed reveals three major and possibly preventable sources of loss of salvage. (Table 140 Fig. 7). High rates of postoperative mortality reduces the

potential five year survival rates of those cases who had resection done. The incidence of involvement of the lines of resection in the stomach was high and there were very few of these patients whose chance of cure was improved by having further and more adequate resection done following the discovery that residual tumour was present in the gastric pouch, duodenum or oesophagus. The pathological reports which described only small fragments of greater and lesser omenta removed with the stomach were taken to represent less than adequate excisional surgery.

These three factors were evident in the several periods reviewed (Table140). In the overall study they were responsible for a further loss of 12.7 per cent in the potential five year survival rate, reducing it from 28.9 per cent to 16.2 per cent. In the consecutive divided periods the loss, due to these causes was comparable at 7.4, 13.7, 13.1, 12.6 and 15.3 per cent. When only those patients who had gastric resection are considered, the total loss of potential salvage is formidable (Table 141). Even in the most recent years this loss amounts to almost two fifths of the potential salvage.

Survival period from first treatment or admission to clinic (months)	Total 1274 patients	Proportion of patients surviving (per cent) in each type group				
		Polypoid 61 patients	Ulcerating 385 patients	Infiltrating 77 patients	Other 28 patients	Unstated 723 patients
Less than six months	38.0	42.6	45.2	39.0	35.7	33.7
6 - 11.9	21.0	23.0	30.9	32.5	14.3	14.7
12 - 23.9	12.2	13.1	20.0	13.0	7.1	8.0
24 - 35.9	8.6	13.1	15.8	9.0	7.1	4.4
36 - 47.9	6.8	13.1	13.0	5.2	7.1	3.2
48 - 59.9	5.3	13.1	10.1	3.9	7.1	2.2
60 and over	5.3	13.1	10.1	2.6	7.1	2.2

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE FIRST TREATMENT (IN THOSE WHO HAD TREATMENT) OR FIRST ADMISSION TO THE CLINIC (IN THOSE WHO HAD NO TREATMENT) RELATED TO THE TYPE OF LESION. SIXTY-SEVEN PATIENTS SURVIVED FIVE YEARS. OF THESE EIGHT HAD POLYPOID LESIONS, THIRTY-NINE HAD ULCERATIVE LESIONS, TWO HAD INFILTRATING LESIONS AND TWO HAD CANCER SUPERIMPOSED ON BENIGN GASTRIC ULCER. IN SIXTEEN CASES THE TYPE OF TUMOUR WAS NOT STATED.

SASKATCHEWAN 1932-50

TABLE 132

Survival period from clinic admission (months)	Total	Proportion of patients surviving (per cent) in each type group				
		Polypoid	Ulcerating	Infiltrating	Other	Unstated
	782 patients	30 patients	163 patients	28 patients	18 patients	543 patients
Less than six months	25.9	30.0	19.6	14.3	33.1	28.0
6 - 11.9	9.3	6.7	7.4	7.1	11.1	10.1
12 - 23.9	3.9	3.3	1.2	-	-	5.2
24 - 35.9	1.9	-	1.2	-	-	2.2
36 - 47.9	1.2	-	0.6	-	-	1.5
48 - 59.9	0.8	-	0.6	-	-	0.9
60 and over	0.8	-	0.6	-	-	0.9

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF FIRST ADMISSION TO THE CLINIC, OF PATIENTS WHO HAD NO TREATMENT, BY THE TYPE OF LESION. THERE WERE SIX PATIENTS SURVIVED FIVE YEARS, IN ONE THE LESION WAS ULCERATIVE, BUT IN THE OTHERS THE TYPE OF LESION WAS UNSTATED.

SASKATCHEWAN 1932-50

TABLE 133

Survival period from date of treatment (months)	Total	Proportion of patients surviving (per cent) in each type group				
		Polypoid	Ulcerating	Infiltrating	Other	Unstated
	185 patients	5 patients	52 patients	11 patients	4 patients	113 patients
Less than six months	38.4	20.0	40.4	27.3	-	40.7
6 - 11.9	13.5	20.0	11.5	18.2	-	14.2
12 - 23.9	4.3	-	5.8	-	-	4.4
24 - 35.9	1.1	-	1.9	-	-	0.9
36 - 47.9	0.5	-	-	-	-	0.9
48 - 59.9	0.5	-	-	-	-	0.9
60 and over	0.5	-	-	-	-	0.9

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD ONLY PALLIATIVE TREATMENT, BY THE TYPE OF LESION. ONE PATIENT SURVIVED FIVE YEARS, BUT THE TYPE OF LESION IN THIS CASE WAS UNSTATED IN THE PATIENT'S RECORD

SASKATCHEWAN 1932-50

TABLE 134

Survival period from date of treatment (months)	Total 304 patients	Proportion of patients surviving (per cent) in each type of lesion group				
		Polypoid 26 patients	Ulcerative 169 patients	Infiltrating 36 patients	Other 6 patients	Unstated 67 patients
Less than six months	68.4	61.5	71.0	61.1	66.7	68.7
6 - 11.9	55.6	42.3	59.8	55.6	33.3	52.2
12 - 23.9	37.8	30.8	42.6	25.0	33.3	37.3
24 - 35.9	30.3	30.8	34.3	16.7	33.3	28.4
36 - 47.9	25.0	30.8	29.0	11.1	33.3	20.9
48 - 59.9	20.1	30.8	22.5	8.3	33.3	14.9
60 or over	19.7	30.8	22.5	5.6	33.3	14.9

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD GASTRIC RESECTION DIRECTED AT CURE, BY TYPE OF LESION. OF SIXTY PATIENTS WHO SURVIVED FIVE YEARS THIRTY-EIGHT HAD ULCERATIVE LESIONS, EIGHT HAD POLYPOIDAL LESIONS AND TWO INFILTRATING LESIONS. TWO PATIENTS WITH CANCER SUPERIMPOSED ON BENIGN ULCER LIVED FIVE YEARS. IN TEN PATIENTS THE TYPE OF LESION WAS UNSTATED

SASKATCHEWAN 1932-50

TABLE 135

Survival period from first treatment or first admission to clinic (months)	Total 1274 patients	Proportion of patients surviving (per cent) in relation to regional lymph gland status		
		Negative 116 patients	Positive 528 patients	Unstated 630 patients
Less than six months	38.0	67.2	38.4	32.2
6 - 11.9	21.0	60.3	20.1	14.6
12 - 23.9	12.2	43.1	10.4	7.9
24 - 35.9	8.6	38.8	5.9	5.4
36 - 47.9	6.8	33.6	3.8	4.1
48 - 59.9	5.3	29.3	2.1	3.7
60 and over	5.3	29.3	1.9	3.7

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE FIRST TREATMENT (IN THOSE WHO HAD TREATMENT) OR FIRST ADMISSION TO THE CLINIC (IN THOSE WHO HAD NO TREATMENT) RELATED TO THE PATHOLOGICAL STATUS OF THE REGIONAL LYMPHATIC GLANDS. SIXTY-SEVEN PATIENTS SURVIVED FIVE YEARS. THIRTY-FOUR HAD NO REGIONAL GLAND INVOLVEMENT. IN TEN TUMOUR WAS PRESENT IN THE GLANDS, AND IN TWENTY-THREE THE DATA ON GLANDULAR METASTASES WERE NOT AVAILABLE

SASKATCHEWAN 1932-50

TABLE 136

Survival period from first admission to clinic (months)	Total 782 patients	Proportion of patients surviving (per cent) in relation to regional lymph gland status		
		Negative 22 patients	Positive 254 patients	Unstated 506 patients
Less than six months	25.9	27.3	22.4	27.7
6 - 11.9	9.3	9.1	7.9	10.1
12 - 23.9	3.9	-	1.6	5.3
24 - 35.9	1.9	-	0.4	2.7
36 - 47.9	1.2	-	-	1.8
48 - 59.9	0.8	-	-	1.2
60 and over	0.8	-	-	1.2

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF FIRST ADMISSION, OF PATIENTS WHO HAD NO TREATMENT, RELATED TO THE PATHOLOGICAL STATUS OF THE REGIONAL LYMPHATIC GLANDS. SIX PATIENTS SURVIVED FIVE YEARS. IN NONE WERE THE DATA CONCERNING REGIONAL GLANDULAR METASTASES AVAILABLE

SASKATCHEWAN 1932-50

TABLE 137

Survival period from the date of treatment (months)	Total 185 patients	Proportion of patients surviving (per cent) in relation to regional lymph gland status		
		Negative 7 patients	Positive 113 patients	Unstated 65 patients
Less than six Months	38.4	42.9	37.2	40.0
6 - 11.9	13.5	28.6	11.5	15.4
12 - 23.9	4.3	-	6.2	1.5
24 - 35.9	1.1	-	1.8	-
36 - 47.9	0.5	-	0.9	-
48 - 59.9	0.5	-	0.9	-
60 and over	0.5	-	0.9	-

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD ONLY PALLIATIVE TREATMENT, RELATED TO THE PATHOLOGICAL STATUS OF THE REGIONAL LYMPHATIC GLANDS. ONE PATIENT, WITH INVOLVED LYMPH GLANDS SURVIVED FIVE YEARS.

SASKATCHEWAN 1932-50

TABLE 138

Survival period from date of treatment (months)	Total 304 patients	Proportion of patients surviving (per cent) in relation to regional lymph gland status		
		Negative 87 patients	Positive 159 patients	Unstated 58 patients
Less than six months	68.4	79.3	64.8	62.1
6 - 11.9	55.6	75.9	45.3	53.4
12 - 23.9	37.8	57.5	27.0	37.9
24 - 35.9	30.3	51.7	17.0	34.5
36 - 47.9	25.0	44.8	12.6	29.3
48 - 59.9	20.1	39.1	6.3	29.3
60 and over	19.7	39.1	5.7	29.3

GASTRIC CANCER: THE CRUDE SURVIVAL RATES CALCULATED FROM THE DATE OF TREATMENT, OF PATIENTS WHO HAD GASTRIC RESECTION DIRECTED AT CURE, RELATED TO THE PATHOLOGICAL STATUS OF THE REGIONAL LYMPH GLANDS. THERE WERE SIXTY PATIENTS SURVIVED FIVE YEARS. IN THIRTY-FOUR THE NODES WERE NOT INVOLVED, IN NINE THE GLANDS WERE POSITIVE AND IN SEVENTEEN THE RELEVANT DATA WERE NOT AVAILABLE.

SASKATCHEWAN 1932-50

TABLE 139

Years	Number Of Patients	Operated Upon		Resected		Causes of Post-Resection Loss of Salvage						Potential Five Year Survival Rate (Per cent)
						Resection Operative Mortality (Per cent)		Lines of Resection Involved (Per cent of Resected Cases)		Omenta Incompletely Removed (Per cent of Resected Cases)		
		Number	%	Number	%	Number	%	Number	%	Number	%	
1932-55	2000	1189	59.5	578	28.9	99	4.5	82	4.1	82	4.1	16.2
1932-36	138	54	39.1	21	15.2	8	5.8	1	0.8	1	0.8	7.8
1937-41	204	94	48.1	42	20.6	19	9.3	1	0.5	8	3.9	6.9
1942-46	395	198	50.1	87	22.1	14	3.8	14	3.5	23	5.8	9.0
1947-51	686	424	61.8	199	29.0	30	4.4	28	4.1	28	4.1	16.4
1952-55	577	419	72.8	229	39.7	28	4.9	38	6.6	22	3.8	24.4

GASTRIC CANCER: THE MANAGEMENT OF GASTRIC CANCER IN SASKATCHEWAN 1932 - 55

TABLE 140

Years	Gastric resection		Loss of salvage in resection cases							
			Operative mortality		Lines of resection		Incomplete removal of omentum		Total loss of salvage from three sources	
	Number	Per cent	Number	per cent	Number	per cent	Number	per cent	Number	per cent
1932-55	578	100.0	99	17.3	82	14.1	82	14.1	263	45.5
1932-36	21	100.0	8	38.0	1	4.8	1	4.8	10	47.6
1937-41	42	100.0	19	45.1	1	2.4	8	19.0	28	66.5
1942-46	87	100.0	14	16.1	14	16.1	23	26.4	51	58.6
1947-51	199	100.0	30	15.1	28	14.1	28	14.1	86	43.3
1952-55	229	100.0	28	12.2	38	16.6	22	9.6	88	38.4

GASTRIC CANCER: THE LOSS OF POTENTIAL SALVAGE IN PATIENTS UPON WHOM GASTRIC RESECTION WAS PERFORMED.

SASKATCHEWAN 1932-55

TABLE 141

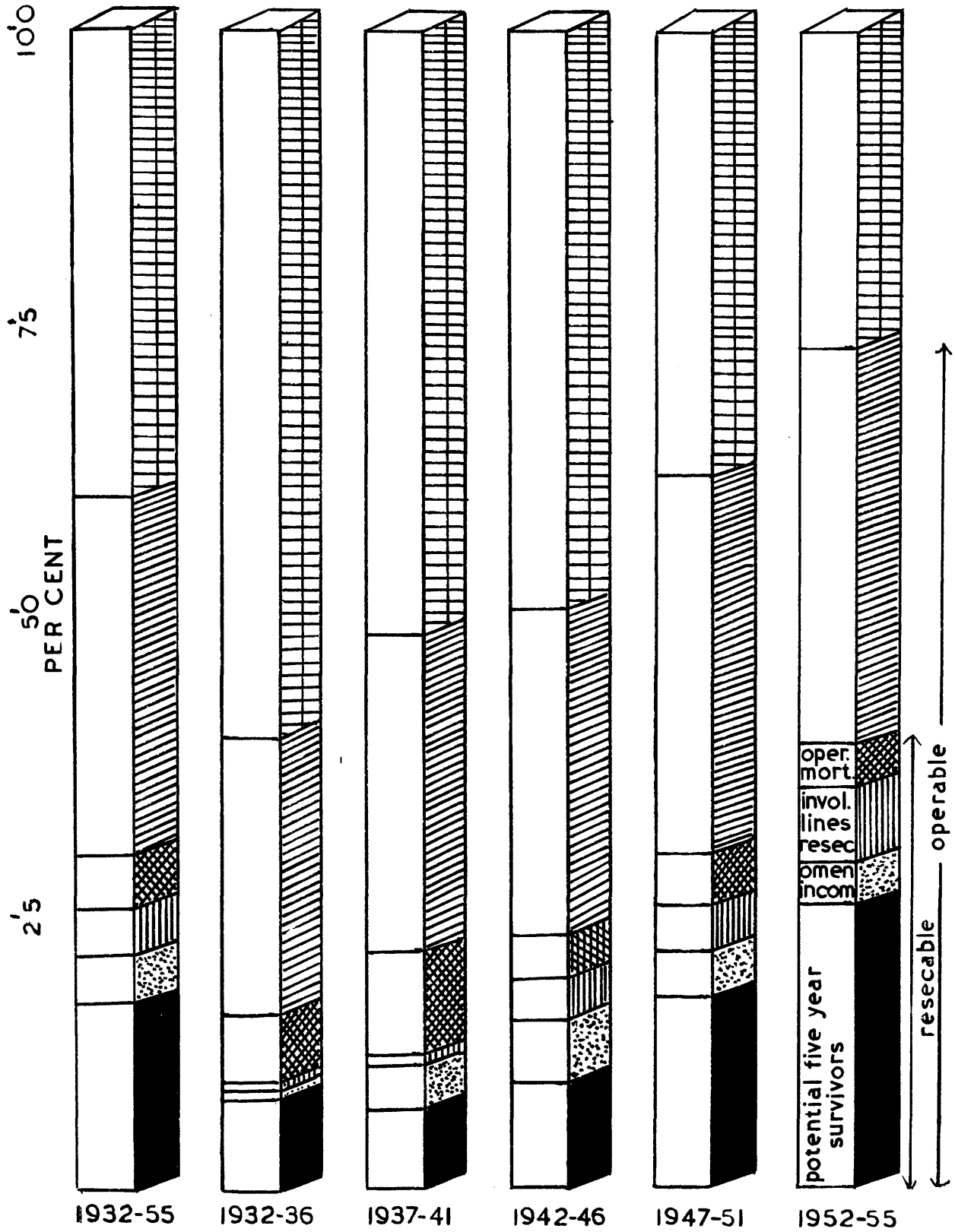


FIGURE 7. THE MANAGEMENT OF GASTRIC CANCER IN SASKATCHEWAN 1932-55.

CHAPTER 11

MISSED OPPORTUNITIES

It is of interest to theorize on what the results of treatment of cancer of the stomach in Saskatchewan might have been if none of the opportunities for cure had been missed. To try to assess this meant reviewing the clinical histories of all those patients who had cancer of the stomach and who were registered in the Cancer Clinics and selecting those in whom salvage appeared to be reasonable, but who, for various reasons, were not cured. The patients registered between the years 1932 and 1950 were taken as the material for examination so that at the end of the study a theoretical but possible five year survival rate could be calculated.

It is realised that the study of clinical histories, the results of investigation and the sequelae of treatment from charts can be very misleading as a means of assessing which

patients could have been salvaged. It is agreed too, that the armchair investigator, by blinding himself to circumstances which can arise to make diagnoses and treatment difficult, can create an entirely erroneous impression and make a black picture blacker or improve it almost at will. An effort has been made, however, to include only those cases in which salvage appeared to be most possible and to present, even at the risk of boring the reader by synopsising the pertinent data, an account which can be assessed by the reader himself.

The reasons for the loss of salvage in this group of patients have been classified under headings which allow an orderly presentation.

UNNECESSARY DELAY IN MAKING DIAGNOSIS

Case 1. A female patient aged 39 years, gave a history of postprandial fullness with epigastric discomfort of eight years' duration. Her symptoms were reputed to have been due to the lack of free hydrochloric acid in her gastric juice. Dilute hydrochloric acid was prescribed and taken, but with only partial resolution of her symptoms. Four years after the onset, she was admitted to hospital with an exacerbation of her symptoms, accompanied by gross melaena. Radiological examination of her stomach revealed no abnormality. No further investigation was done at this time to determine the

source of the gastrointestinal bleeding. She was discharged from hospital, the bleeding under control, but with the dyspeptic symptoms unabated. Four years later she was referred to the Cancer Clinic. She was submitted to total gastrectomy for carcinoma of the lesser curvature. The pathologist demonstrated tumour tissue extending to within one millimeter of the proximal line of resection. The regional lymphatic glands were involved in tumour. The patient died two years later with widespread metastases within the abdominal cavity.

Comment: It is possible, if the patient had been carefully observed because of her achlorhydria or more thoroughly investigated to determine the source of the melaena, that she might have been given surgical treatment when her lesion was small and confined to the stomach. The persistence of her dyspepsia despite the treatment given should have aroused greater suspicion and should have led to more thorough investigation, earlier diagnosis and earlier surgical treatment.

Case 2. A seventy-one year old man was referred to the Cancer Clinic with a history of twelve pounds weight loss over the previous month, epigastric discomfort, flatulence and fat intolerance. There had been no previous dyspepsia. Cholecystogram and barium meal examination were made but both were reported as showing no abnormality. The patient was discharged from the clinic as having no evidence of

malignant disease. Twenty-two months later he was again referred to the Cancer Clinic because of persistence of the same symptomatology but with the addition of anorexia and constipation. He was found to have achlorhydria and x-ray examination revealed carcinoma of the cardiac end of his stomach. His abdomen was explored, but in view of the widespread glandular and liver metastases, excisional surgery was not done. He died seven months later.

Comment: The Cancer Clinic is responsible for the delayed diagnosis in this case. Inadequate investigation following his first referral to the clinic is inexcusable. It is possible that if the patient had been operated upon at this time a resectable non-metastasised lesion would have been found.

Case 3. A fifty-one year old man was referred to the Cancer Clinic with a history of epigastric pain occurring two hours after taking food and postprandial fullness. He had been x-rayed one year previously by his own doctor and told he had a duodenal ulcer. Failure of medical treatment over this year, however, caused concern and his stomach was again x-rayed. On this occasion gastric carcinoma was diagnosed. On account of this he was referred to the Cancer Clinic. There he was found to be achlorhydric and moderately anaemic, but barium meal examination was reported as showing no abnormality. He was discharged from the clinic. Seven month later his

stomach was again x-rayed by his family doctor and a pedunculated gastric tumour reported. He was again referred to the Cancer Clinic but was again discharged because a barium meal conducted there was reported negative for ulcer and tumour. Thirteen months later he was admitted to hospital because of continued symptoms. He was operated upon and a total gastrectomy done. Direct extension of his tumour to transverse colon, jejunum, pancreas and mesenteric lymph nodes was found. He died nine months later.

Comment: Any comment on this case is superfluous. It is difficult to believe that such an error could occur in a well organised Cancer Clinic. For the error to have been repeated is unpardonable.

Case 4. A sixty-four year old man was referred to the Cancer Clinic with a typical history of carcinoma of the stomach of one year's duration. His barium meal was reported as showing only duodenal cap deformity. His free hydrochloric acid was at a normal level and his haemoglobin was ninety-nine percent. There was some clinical suspicion that a mass was palpable in his epigastrium. X-ray investigation was repeated one month later and was again reported as showing only evidence of old duodenal ulcer. Again there was suspicion that a palpable mass was present. The patient was discharged from the clinic with a diagnosis of duodenal ulcer. Thirty months later the patient's abdomen was explored by his own doctor. A large

mass was found in the prepyloric region and metastases had occurred in the regional lymphatic glands and liver. He died nine months later.

Comment: The typical history and the doubt regarding the presence of an epigastric mass in a man of 64 years of age should have caused concern regarding the possibility of gastric cancer. The finding of a normal range of free hydrochloric acid and an x-ray report of duodenal ulcer should not have influenced the clinician unduly. The patient should have been given the benefit of at least one additional clinical assessment before discharge from the clinic or better, been offered the advantage of abdominal exploration.

Case 5. A sixty-four year old man gave a two years' history of nausea, postprandial fullness, loss of weight and weakness. During this time he had had his stomach x-rayed twice, but on neither occasion was any abnormality reported. His free hydrochloric acid level had not been assessed and his stool had never been tested for occult blood. A third x-ray revealed a large filling defect in the fundus of his stomach. He was referred to the Cancer Clinic on this account. Total gastrectomy was performed. Direct extension to his oesophagus and lesser omentum was found. He died eleven months later.

Comment: It is probable that if this patient had been properly investigated when he first consulted his doctor,

suspicion that he had gastric cancer might have been greater and excisional surgery performed two years earlier.

Case 6. A sixty-seven year old man was referred to the Cancer Clinic with a history that he had had epigastric pain for six years. During this time, he had consulted a number of doctors without obtaining relief. His stomach had never been x-rayed. The pain had been worse during the last year, he had progressive weakness of three months' duration, anorexia for two months and for one month he had been vomiting with small haematemeses. On admission to the clinic, his haemoglobin was fifty-four percent, he was found to have hyperchlorhydria and his barium meal was reported as showing no abnormality. He was discharged from the clinic with a diagnosis of bleeding peptic ulcer, although there had been no radiological evidence of an ulcer. He died ten months later with carcinoma of his stomach.

Comment: The culpability for this case probably rests on the shoulders of the Cancer Clinic and upon his own private medical advisor. The latter prescribed treatment for him without investigating the cause of his symptoms. The Cancer Clinic was unjustified in making a diagnosis of peptic ulcer where none was demonstrable in a patient who gave a history of marked change in long-standing gastric symptomatology. It was wrong to accept hyperacidity as the hall-mark of benignity.

Case 7. A fifty-five year old man was referred to the Cancer Clinic with a history of three months' weight loss and one month's history of epigastric pain and postprandial fullness. His haemoglobin was ninety-nine percent, he had hypochlorhydria and his faeces did not contain occult blood. X-ray of his stomach revealed a greater curvature niche, upon which the radiologist gave an equivocal report. The patient was discharged from the clinic as having non-malignant ulceration of his stomach. Twenty-eight months later he was found to have a non-resectable carcinoma of his stomach. He died two months later.

Comment: It would be difficult to excuse the lack of appreciation of the significance of greater curvature ulceration in a man of fifty-five years of age with a short history of dyspepsia. This patient should have had abdominal surgery when he was first seen at the Cancer Clinic.

Case 8. A seventy-two year old man, with a history of three months' epigastric pain, unrelated to meals, and constipation, was referred to the Cancer Clinic. He was found to have achlorhydria, he had no occult blood in his stool, and his barium meal was reported as showing no evidence of ulceration or tumour. He was discharged and advised to take dilute hydrochloric acid. No follow-up was arranged. The patient continued to have gastric symptoms and was referred again to the Cancer

Clinic sixty-five months later. On this occasion there was radiological evidence of cancer of the stomach. The patient elected to seek treatment at another clinic, but was told there that his disease was hopeless. On his return he was operated upon for a prepyloric cancer. Partial gastrectomy was performed. There were no lymphatic glands, no direct extension of tumour and no other evidence of metastatic disease. The pathologist reported however, that there were tumour emboli in the mucosal venules and lymphatic channels. The patient died fifteen months later with widespread abdominal metastases.

Comment: Again, a short history of dyspepsia in a patient of seventy-two years of age should warrant observation, especially when the dyspepsia is accompanied by achlorhydria and when the symptoms are not relieved by the administration of hydrochloric acid. If surgery had been done in this patient even a few months earlier, it is possible that the lymphatic and vascular spread of his tumour (which at the time of his operation appeared to be confined to the gastric wall) would not have been present and the extent of his gastric resection which in this case was probably inadequate to cure most gastric cancers, might have been successful in his case.

Case 9. A fifty-seven year old man, who had a thirty-three years' history of dyspepsia was referred to the Cancer Clinic because of a change in symptoms of one month's

duration. His pain was no longer influenced by taking food, his appetite had become impaired and he had begun to vomit. X-ray revealed a lesser curvature ulcer, and in addition a prepyloric ulcer. He had hypochlorhydria but was not anaemic. Surgery was advised, but the patient showed no enthusiasm for this type of therapy and he was discharged from the Clinic without being given an appointment for review examination. He continued to have symptoms and was hospitalized on several occasions without improvement. He was referred again to the Cancer Clinic thirty months later. Partial gastrectomy was performed. The surgeon was uncertain whether there was cancer present or not. Although there were several palpable glands in the operative field, only one was recovered from the operative specimen. He died seventeen months later with evidence of residual and recurrent cancer in his abdomen.

Comment: This patient was unfortunate thrice over. His discharge from the Clinic was ill-advised. Continued treatment by his own doctor without relief of symptoms and without re-investigation was unwise. The surgeon performed a less than adequate gastric resection and did not remove all those nodes which appeared to him to be involved. It is possible if any one of these errors in judgement had not been committed that the patient would have had a better chance of survival.

Case 10. A sixty-two year old man, with a history of diarrhoea of several months' duration with epigastric discomfort unrelated to food in time or type, was referred to the Cancer Clinic. He was anaemic. His gall bladder and stomach were x-rayed, the former being reported as normal and the latter exhibiting only ulcer deformity of the duodenal cap. No assessment was made of the free hydrochloric acid in his gastric juice and his stool was not tested for occult blood. He was discharged from the Clinic. One year later he was again referred and his gall bladder, stomach and large bowel were investigated radiologically. He was discharged from the Clinic with a diagnosis of duodenal ulcer. Six months later he presented for medical attention at another city, was operated upon, but because of the extensive spread of tumour from a primary lesion of the stomach no curative surgery was possible.

Comment: Perhaps with more thorough investigation at his first visit to the Cancer Clinic, the patient could have been salvaged.

Case 11. A seventy-two year old man had been under continuous care of his own doctor for four years. His major symptom was epigastric discomfort which, at first, occurred fifteen minutes after each meal, but after two years became steady. His gall bladder was x-rayed and he was told

that his symptoms were due to absorption of poisons from that organ. No further investigation was carried out despite the progressive nature of symptoms with weight loss and anorexia. He was referred to the Clinic, with cancer of his stomach arising from the greater curvature. There was a palpable mass. He died without treatment in three months.

Comment: In four years the progression of cancer within and extension beyond the stomach can be considerable. This patient was probably salvagable at the onset of his symptoms, but was denied the chance of cure because of inadequate investigation. The patient was not blameless. With even a minimum of sense he would have changed to another doctor in the hope that he might obtain relief from his symptoms.

Case 12: A seventy-one year old man was referred to the Cancer Clinic and gave a history of epigastric discomfort of eight months' duration, with postprandial fullness, vomiting, anorexia and loss of weight. He was found to have hypochlorhydria. X-ray of his stomach revealed only a duodenal ulcer. Gastroscopy was reported as showing no abnormality. Medical regimen was advised and the patient's progress was followed by the Cancer Clinic staff. His condition was reviewed seven months after his initial investigation, and although his symptoms had not been alleviated and his weight loss was now gross, he was not investigated at this time, but was given an appointment for review examination on a date six months later. When he

reported back to the Clinic at the end of this period, he had a palpable mass in his epigastrium. At this time he was investigated more fully, but was considered too advanced for surgical treatment. He died one year later.

Comment: This case illustrates the error of accepting the evidence of ancillary methods of investigation in preference to the patient's history and clinical findings. It is difficult to excuse the failure to re-investigate the patient at his first review examination despite progression of his symptoms.

Case 13: A sixty-four year old man was referred to the Cancer Clinic complaining of postprandial epigastric pain, fullness, constipation, and increasing weakness of one month's duration and eleven pounds weight loss during the previous two months. He was found to have achlorhydria. Barium meal showed "cup and spill" deformity of his stomach. He was discharged from the Clinic with the advice that he should take dilute hydrochloric acid. He was referred again to the Clinic five months later. His symptoms were still present, but were less severe. He was reassured and discharged from the Clinic again without further investigation. He returned to the Clinic in four months. Repeat x-ray of his stomach showed a large filling defect at the pyloric end of his stomach. His disease was considered too advanced for treatment and he died in six months.

Comment: Abdominal exploration when he was first seen may have proved the diagnosis of cancer of the stomach and may have permitted curative resection.

Case 14. A fifty-six year old man was under the continued medical supervision of his family doctor for a period of four years. His symptoms were those of dyspepsia. He had his stomach x-rayed at the first consultation, but these were reported by his doctor as showing no abnormality. No further investigation was done, but he was given "powders" to relieve his symptoms. Within one year, his appetite had failed and his dyspepsia had increased in severity. Weight loss became marked but no further investigation was conducted. His treatment was changed many times. At the end of four years he consulted another doctor who demonstrated a prepyloric cancer by x-ray. The patient was beyond hope of cure by this time, as evidenced by the large umbilical ulcer which was proved histologically to be secondary spread from a primary carcinoma of the stomach.

Comment: This patient's chance of long term survival was prejudiced by the fact that his doctor remained satisfied with the apparent absence of cancer in one x-ray examination. He failed to realise that subsequent to this radiological investigation gross changes could have occurred in the stomach. It is possible that the inexpert reporting of the x-ray films by the general practitioner or inexperience in

the preliminary fluoroscopy may have been responsible for missing a small lesion. The failure of the patient's symptoms to respond to treatment should have been indication enough to warrant further and perhaps more expert examination.

Case 15. A sixty-nine year old man was referred to the Cancer Clinic with a two years' history of irregular epigastric discomfort, occurring half an hour after food, anorexia and loss of weight. At this time he was well nourished and in good general condition. He was found to have achlorhydria. Barium meal suggested a diagnosis of hypertrophic gastritis, although there was suspicion that there might also be a prepyloric ulcer present. Dietary regime and dilute hydrochloric acid were prescribed and with this the patient improved but his symptoms did not subside completely. He reported to the Clinic at regular intervals for twenty-eight months and was discharged at the end of this time. There were no radiological examinations of his stomach done during this period. Within six months he returned to the clinic because of the onset of diarrhoea, anorexia, loss of weight and epigastric discomfort. Barium meal revealed fixation of the lesser curvature of his stomach. No treatment was advised, but he was given an appointment for a further examination in two months' time. Again there was radiological evidence of gastric cancer, but he was considered to be too advanced for treatment at this time. He died without having had treatment eight months later. Autopsy demonstrated spread of the disease to the oesophagus,

pancreas, regional and distant lymphatic glands.

Comment: Had the patient been given the benefit of surgical therapy when he was first referred to the Cancer Clinic it is likely that his chance of survival would have been considerable. Again the failure to reinvestigate the patient when there was radiological doubt concerning the possibility of a prepyloric ulcer is unpardonable.

Case 16. A fifty-six year old man was under the care of his doctor for a period of sixteen months and received empyrical treatment for dyspeptic symptoms which were said to be due to "ulcer", although the patient had never been investigated. With this treatment the patient's symptoms continued. Not until the patient had lost thirty pounds in weight and an epigastric mass became evident was he referred to the Cancer Clinic. X-ray of his stomach revealed cancer at the pyloric end. The radiologist volunteered the opinion that the lesion was inoperable. No treatment was given to the patient. He died sixteen months later.

Comment: The patient's family doctor was responsible for sixteen months delay in diagnosis. The radiologist cannot determine the operability or resectability of a gastric lesion by his examination. The fact that the patient survived for sixteen months after the diagnosis had been made raises the possibility that the presence of the palpable mass was no indication of non-resectability and that

the patient may have been salvaged if this treatment had been carried out. It is probable that if excisional surgery had been done when the patient first consulted his doctor that reasonably long term survival would have resulted.

INADEQUATE SURGICAL TREATMENT

There were thirteen cases in this group.

Case 1. The distal half of the stomach was removed for a prepyloric lesion. The great omentum was not removed. The patient died postoperatively as a result of peritonitis and pulmonary embolus. Autopsy revealed that the greater omentum had been invaded directly by the gastric tumour and that this extension had not been removed at the time of surgery. There was no other evidence of tumour. The regional lymphatic glands were not involved.

Cases 2 - 5. In this group, the proximal line of resection was involved in tumour and this was reported by the pathologist on the surgical specimen removed. In none of the patients was there any evidence of extra gastric spread at the time of operation. One of these patients died postoperatively and the only evidence of cancer was in the gastric stump. There was no extra gastric, direct or indirect, spread and the regional glands were not involved in tumour. The second patient died nine months after partial gastrectomy and was found to have residual tumour in the gastric remnant, and the regional nodes,

which had not been involved preoperatively were involved at the time of death. The third patient had a second abdominal exploration made twenty-eight months after partial gastrectomy had been performed. The line of anastomosis was involved and there was secondary disease in the regional glands and peritoneum. The fourth case in whom the proximal line of resection was found to be involved after subtotal gastrectomy died two years after his operation. Residual tumour was found involving the anastomosis but no regional lymphatic glands were involved in tumour and there was no evidence of direct spread or metastases found.

Cases 6 - 8. In this group, the distal line of resection was found to be involved in tumour. Partial gastrectomy was performed upon the first patient for a prepyloric tumour. There was no regional glandular involvement and there was no evidence of extra gastric spread. The pathologist did not report on whether the tumour extended to involve the lines of resection. It would appear that the distal line of resection was involved because within four months the patients required abdominal exploration again and the duodenal stump was found to be grossly infiltrated with tumour. The pathologist was at fault in this case for not reporting the involvement of the distal line of resection. The second patient had a partial gastrectomy for a prepyloric lesion with pyloric stenosis. There was no evidence of extra gastric spread and the regional lymphatic glands were not involved. The

pathologist reported involvement at the distal line of resection. No further treatment was given and the patient died six months later with evidence of local recurrence of tumour. The third case had a history of less than one month's duration. He had a partial gastrectomy done. The pathologist found tumour extending to the distal line of resection. There was no lymphatic glandular involvement and no evidence of other extra gastric spread. The patient died postoperatively. There was no autopsy and the reason for the postoperative death was not ascertained.

Cases 9 - 11. These were patients in whom both proximal and distal lines of resection were found to be involved in patients who demonstrated no extra gastric spread or lymph node metastases. The first patient died fourteen months post-operatively and at autopsy the anastomotic line was involved in residual tumour and cancer was also present in the duodenum. The second patient survived only six months. The third patient had partial gastrectomy for a lesser curvature infiltrative lesion. The patient survived eleven months and at death had peritoneal implantation by tumour. One year previous to the gastric surgery the patient had had a cholecystectomy done in an effort to relieve her of symptoms which were more characteristic of cancer of the stomach than cholecystitis or cholelithiasis. It is possible that if the cancer of the stomach had been found at this time, the lesion would have been smaller and the chance of the tumour

extending to the lines of resection would have been less.

Cases 12 and 13. A patient with a long history of dyspepsia, but with change in symptoms one year previously and whose prepyloric region exhibited distortion on x-ray examination was operated upon. The operative report described fibrosis and evidence of a healed ulcer in the prepyloric area. No treatment was directed at the stomach, but the appendix was removed. Two years later, the patient's abdomen was re-explored. On this occasion carcinoma at the prepyloric region was found and a partial gastrectomy was done. No pathological report was made on the regional lymphatic glands because none had been removed with the surgical specimen. The resected portion of stomach measured one inch along the lesser curvature and three inches along the greater curvature. The lesion measured 1.2 cm. in length. The patient remained well for six years and three months, but died of recurrent cancer of the stomach.

The second patient in this group had a resectable lesion at the pyloric end of the stomach with no glandular spread or evidence of metastasis, but had only a gastro-enterostomy performed. He died postoperatively.

Comment: Leaving obvious residual tumour, when the gastric lesion is resectable and there is no direct extension or glandular spread is not likely to encourage long term survival. The pathologist should examine all lines of resection and when these are reported as showing evidence of tumour, the surgeon

should re-operate upon the patient and perform a wider resection. It is easy to misjudge the extent of tumour in the stomach wall and no shame need be felt by the surgeon who finds that he has resected through tissue which contains tumour cells. There should be a feeling of guilt, however, when a patient dies of residual disease arising from the cells at the lines of resection when no effort has been made to resect the involved resection lines.

POSTOPERATIVE DEATHS WITH NO RESIDUAL CANCER

This group of patients constitutes probably the most tragic of all. They are the patients who have been cured, but who have died not from the disease, but as a result of the treatment. There were twenty-eight patients in whom gastric resections of various degrees had been conducted who exhibited at operation no extra gastric extension of tumour, no glandular involvement and no metastases and in whom this freedom from tumour was proved. The causes of death included peritonitis, with and without leaking anastomotic lines, ileus, subphrenic abscess, pulmonary abscess, broncho-pneumonia, massive gastric haemorrhage and cardiac complications.

ERRORS OF CLINICAL OR SURGICAL JUDGEMENT

There are twelve examples of patients who were denied the chance of cure because of errors of judgement on the part of their medical attendants.

Case 1. A sixty-seven year old man, with a five months' history of nausea, vomiting, anorexia, early satiety and loss of weight, was found to have achlorhydria and to be grossly anaemic (haemoglobin 45%). Barium meal demonstrated a prepyloric cancer. The patient had a palpable mass. The radiologist and the surgeon were agreed that the patient's tumour was not resectable. On account of this no abdominal exploration was done. During the next five years the patient's stomach was X-rayed repeatedly and showed slow progression in size of the tumour. He eventually died sixty-four months after being seen for the first time by the surgeon.

Comment: The presence of a palpable mass was sufficient to influence the surgeon to withhold treatment. The radiologist's opinion may have contributed to this decision. The patient survived longer than six years without treatment and this would suggest that his lesion was curable when the patient was first seen.

Case 2. A fifty-nine year old man with a history of postprandial vomiting of six weeks' duration was operated upon and was said by the surgeon to have a non-resectable tumour at the pyloric end of his stomach. A jejunostomy was performed. The patient died postoperatively. An autopsy was performed and two separate cancers were found in the stomach, the first at the pyloric end and the second on the lesser curvature. The former had extended directly into the duodenum and the latter had

remained localised. There was no extra-gastric spread and no lymphatic glandular involvement. The stomach was not fixed and the lesions were very suitable for resection.

Comment: This patient may have been salvaged with adequate excisional surgery.

Case 3. A seventy year old man with one month's history of dyspepsia, vomiting, epigastric pain and constipation was found to have pyloric obstruction. At operation the stomach was grossly distended. Gastrotomy was done and revealed "a quantity of dried up food in the antrum." It was thought that the pyloric stenosis was secondary to benign ulceration and gastro-enterostomy was performed. Fourteen months later he was re-admitted to hospital in poor general condition and with a large palpable epigastric mass. X-ray revealed a filling defect, the presence of which was confirmed at abdominal exploration. Extension of tumour was evident in the gastro-hepatic ligament. The posterior gastroenterostomy done previously was found to be obstructed and anterior gastroenterostomy was performed. The patient lived for seven months.

Comment: The observations of the surgeon at the time of operation were possibly inaccurate. Had gastric resection been done at that time, even as treatment for "benign ulcer" a cure for carcinoma of the stomach might have been accomplished.

Case 4. A seventy-two year old woman was referred to the Cancer Clinic with a typical history of gastric

cancer. The investigations done supported this diagnosis. She was found to have achlorhydria, and X-ray demonstrated a prepyloric cancer. She was referred to a clinic in another city where she was reinvestigated and was sent home with a diagnosis of benign ulcer and advice to keep to a strict diet. She returned to the Cancer Clinic seventeen months afterwards with clinical and radiological evidence of pyloric stenosis. She was referred again to the clinic from which she had been discharged, but on this occasion, abdominal exploration was performed, and an extensive carcinoma involving the greater part of the stomach and with spread to the oesophagus was found. The regional lymphatic glands were involved. Resection was considered impossible. The patient died within two months.

Comment: It is dangerous to assume the benignity of a prepyloric ulcer, especially when other evidence, such as the clinical history and the results of gastric analysis support a diagnosis of gastric cancer. The failure to salvage this patient's life was due to the non-acceptance of such evidence because the barium meal X-ray done at the second clinic did not exhibit the lesion which was evident in the first X-ray series done at the Cancer Clinic.

Case 5. A sixty year old man, with a typical history of cancer of the proximal end of the stomach, was operated upon. The operation note "the lower one third of the stomach is freely movable and no involvement of pancreas is

apparent. It is questionable whether the growth is resectable even by the abdomino-thoracic approach," is self-explanatory. Only jejunostomy was done. The patient died postoperatively of peritonitis. Autopsy was performed and the presence of cancer of the cardiac end of the stomach with involvement of the lower end of the oesophagus was confirmed. There was no spread within the abdomen and all the lymphatic glands examined by the pathologist showed no evidence of tumour. The lesion was mobile and resectable.

Comment: Had the surgeon been more courageous and performed the total gastrectomy or oesophago-gastrectomy which was necessary, the patient may have had an excellent chance of cure. Any surgeon who does not feel competent to deal with any emergency he may meet or to perform the most radical excisional surgery when such is found necessary, should not attempt to operate on any patient for cancer.

Case 6. A sixty-five year old man with a history typical of pyloric stenosis and who when he was seen by the Cancer Clinic staff and the consultant surgeon was emaciated and dehydrated. No effort was made to improve the patient's general condition and the surgeon was satisfied that "nothing could be done." The patient died. Autopsy revealed a complete pyloric obstruction and a small adenocarcinoma involving the lesser curvature of the stomach. The cancer was not responsible for the pyloric obstruction. There was no direct extra gastric spread and the lymph nodes

contained no tumour and there were no metastases.

Comment: Correction of this patient's dehydration and possible electrolyte imbalance, with additional supportive therapy could probably have improved his general condition to such a degree that with proper excisional surgery the patient could have been salvaged.

Case 7. A fifty-seven year old woman, with a history of two haematemeses in successive years, and who had had medical treatment was X-rayed and found to have an ulcer on the lesser curvature of the distal one third of the stomach. Abdominal exploration was done, but no ulcer was found and "slight prepyloric serosal fibrosis" was seen. A cholecystectomy was done. She was seen on several occasions during the next two years, during which time she continued to complain of dyspepsia, but no investigations were conducted. Thirty-eight months after the cholecystectomy she was found to have a large carcinoma in the antrum, which was too advanced for treatment. Autopsy revealed metastases to liver falciform ligament, ovaries, abdominal wall and regional and remote lymphatic glands.

Comment: Ample opportunity was available to treat this patient at an early stage of development of her disease and at a time when cure might have been effected.

Case 8. A seventy-one year old man, with one year's history of epigastric pain, occurring two-and-a-half hours after food, and which was relieved by food and alkali, was given medical

treatment on the basis of a diagnosis of duodenal ulcer. No investigations were conducted. This treatment was continued for eight months, but because of the onset of anorexia, weakness and the loss of forty per cent of the patient's normal body weight, he was referred to the Cancer Clinic. He was found to have hyperacidity, but barium meal appearances were suggestive of a gastric cancer. He was anaemic. Abdominal exploration was done, but the surgeon believed that the disease was too widespread for possible extirpation, only jejunostomy was performed. The patient died three months later. Autopsy revealed an infiltrating lesion of the stomach but no extra gastric spread. All the regional lymphatic glands examined were free of tumour. There was no duodenal or oesophageal spread.

Comment: It is possible that with total gastrectomy this patient could have been salvaged.

Case 9. A seventy-two year old man gave a two years' history of epigastric pain occurring after food, with post-prandial fullness. Anorexia, nausea and vomiting had been symptoms for one year. Weight loss amounted to sixteen per cent of his normal body weight. X-ray showed a prepyloric ulcer, diagnosed by the radiologist as being consistent with carcinoma. Gastric analysis revealed a free hydrochloric level greater than normal. It was assumed at the Cancer Clinic that the ulcer was benign and medical management for the ulcer was advised. The patient was discharged from the clinic without determining whether

the medical therapy had resulted in complete resolution of the ulcer. The patient returned to the clinic two and a half years later, having had a haematemesis and with a haemoglobin value of twenty six percent of normal. The patient died without treatment within six months. Cancer of the stomach with regional glandular metastases was found at autopsy.

Comment: This patient demonstrates again the danger of making an assumption of benignity in gastric ulcers.

Case 10. A sixty-seven year old man was referred to the Cancer Clinic by another clinic outside the province. He had been investigated there and told he had a gastric cancer and had only four months to live. When admitted to the Cancer Clinic he was dehydrated and had clinical signs of pyloric obstruction. The correctness of the diagnosis and the prognosis by the referring clinic was assumed, and little was done to help the patient. He died in three days. Autopsy revealed a Grade II carcinoma of the prepyloric region of the stomach with no direct extra gastric spread, no lymphatic glandular involvement and no evidence of metastases. The lesion would have been readily resectable.

Comment: The first clinic made an error of judgement. The second clinic's error lay in accepting the prognosis of the first clinic. The patient was salvageable.

Case 11. A sixty-one year old woman gave a long history of dyspepsia, but with the onset of anorexia was

investigated at the Cancer Clinic. A palpable mass was found. Gastric analysis revealed achlorhydria. No abnormality was seen on X-ray of her stomach. She was discharged from the clinic, but was advised to take dilute hydrochloric acid. She continued to have slowly progressing symptoms for more than five years and when she died an autopsy revealed a small antral cancer with regional lymphatic glandular metastases.

Comment: This patient's abdomen should have been explored when she was first seen at the Cancer Clinic. Her symptoms, the achlorhydria and the presence of a palpable mass would have justified this action despite the fact that time and an autopsy proved that the finding of a mass was only a clinical impression and not factual. It is possible that the lesion might have been found at that time and the patient salvaged. Certainly the progressive nature of her symptoms should have alerted the medical attendants to the probability of the diagnosis of gastric cancer before spread had occurred.

Case 12. A fifty-nine year old man gave a history of dyspepsia of more than twenty years' duration. A change in his symptomatology occurred with the onset of vomiting, weakness and weight loss. A gastro-enterostomy was done. Three years later he was referred to the Cancer Clinic with widespread metastases from primary gastric cancer. The anastomotic line of the gastro-enterostomy was involved in tumour.

Comment: It is possible that the gastro-enterostomy was done at a time when the lesion was confined to the stomach. No note of glandular involvement was made at the time of this operation. Adequate gastric resection at this time may have given the patient a reasonable chance of cure.

OTHER REASONS

1. Patient refused treatment.

There were four patients all with histories of short duration, with apparent localised disease in the stomach who refused to have treatment and who died at intervals of three days, nineteen months, twenty-nine months and thirty-one months after being seen at the Cancer Clinics. There is evidence that all of these patients could have been salvaged had they permitted the supportive and excisional treatment offered to them. The patient who lived only three days after bein g seen, died as the direct result of a haematemesis. Autopsy revealed cancer at the pyloric end of the stomach with no extra gastric spread, lymph nodeinvolvement or other metastases.

2. Cancer involving the cardia.

There were several patients in the series who had cancer involving the cardia. Many were seen at a time when oesophago-gastrectomy and total gastrectomy were procedures which were not seriously considered in the treatment of gastric neoplasm because they were associated with such a high mortality rate.

Two cases did occur within the period when these procedures were considered applicable and practical in the treatment of gastric cancer. Neither case had evidence of extra gastric spread or lymph gland metastases. One patient died postoperatively after abdominal exploration. Autopsy revealed the cause of death to be perforation of the oesophagus apparently produced by the gastric suction tube. The cancer was confined to the stomach and there was no oesophageal spread. The regional lymph glands were not involved in tumour. The second patient survived fourteen months after exploratory laparotomy.

3. Unwarranted delay in referral for treatment.

Two patients were observed by their doctors over long periods of time after clinical history and X-ray findings pointed to a diagnosis of cancer of the stomach. Neither patient was referred for treatment until there could be no hope of cure.

The first case had a history of dyspepsia for more than thirty years. Change in symptomatology occurred with loss of weight and onset of diarrhea. His stomach was X-rayed at this time and gastric carcinoma diagnosed. One year later X-ray confirmed this diagnosis. During the next four years several additional X-rays of stomach were taken with repeated confirmation, but during this time the patient was given medical treatment only. Autopsy revealed a polypoid cancer of the stomach with liver metastases, but no lymphatic glandular involvement.

The second patient was also inoperable at the time he was referred to the Cancer Clinic. He had had continuous medical treatment for four years, despite the X-ray evidence of a malignant gastric ulcer. It is not known whether there were metastases at the time of his death, but it would appear reasonable that four years prior to his death his gastric lesion would have been resectable and that the patient might have had a chance of cure.

It is not possible to assess to what extent the five year survival rate would have been influenced had these opportunities not been missed.

The gratitude of most men is but a secret desire of receiving greater benefits.

La Rochefoucauld

A survey of the clinical data from a large number of patients such as has been presented in this monograph would not have been possible without the help of many of my friends and colleagues.

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