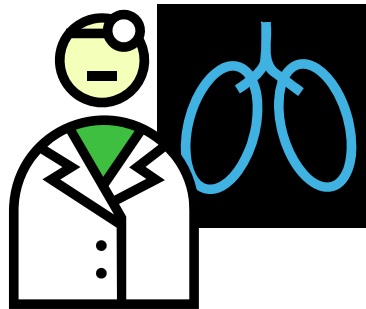


OUTCOME ANALYSIS

2009

LUNG CANCER

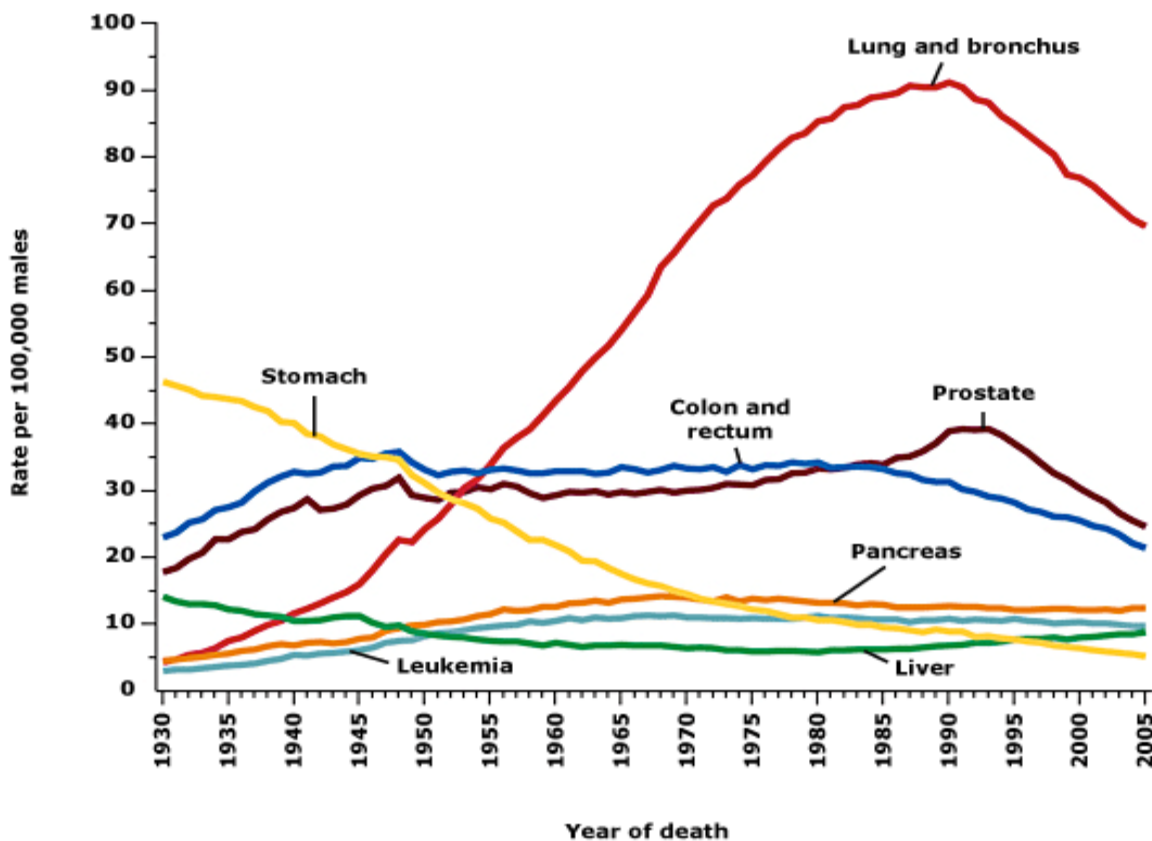


Written By: Aaron P. Scholnik, MD, FACP

Outcome Analysis – 2009 – Lung Cancer

Lung cancer is the most frequently diagnosed and certainly the most frequently fatal of all cancers in the United States and throughout the world. It is estimated that there are 1.2 million deaths per year from lung cancer throughout the world and that in the United States in 2008 there were approximately 215,000 new cases of lung cancer discovered and approximately 162,000 deaths from lung cancer. To put this in perspective, the total number of cancer deaths due to colorectal, breast and prostate cancers combined is responsible for “only” 124,000 deaths ([Figure 1 and 2](#)).

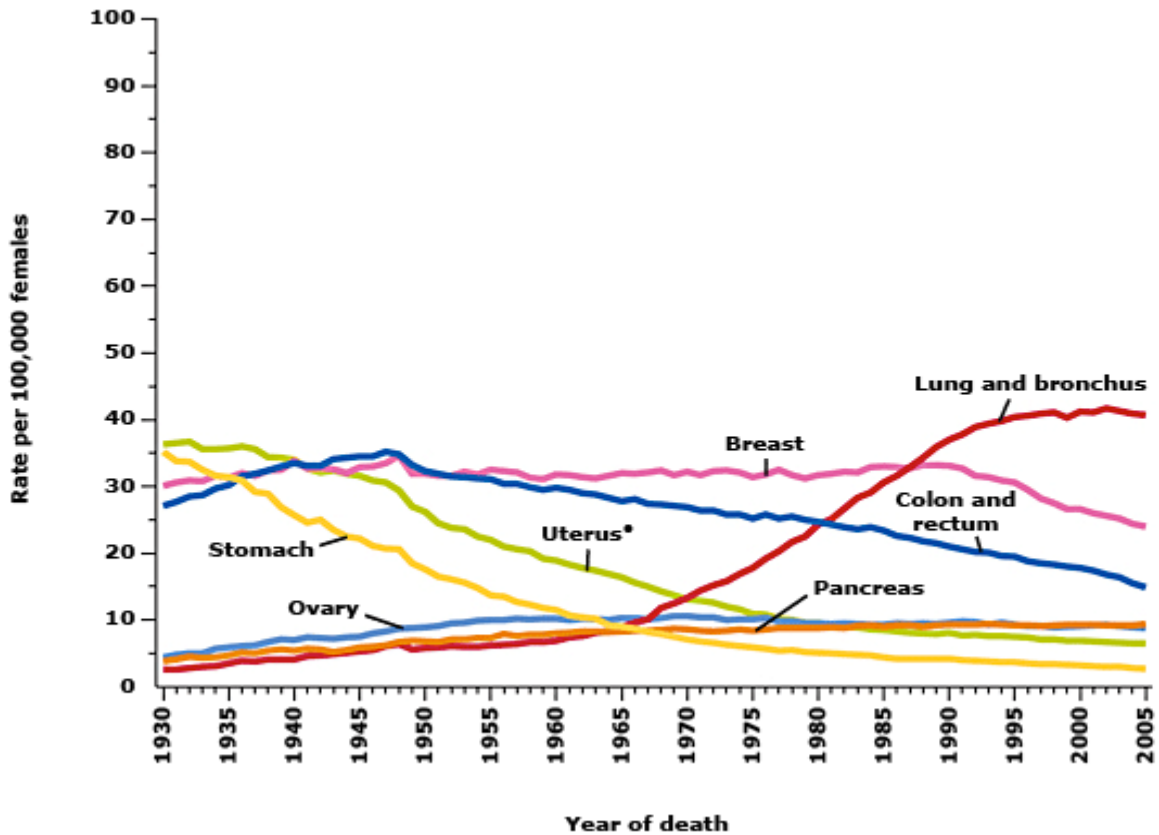
FIGURE 1
ANNUAL AGE-ADJUSTED CANCER DEATH RATES AMONG MALES FOR SELECTED CANCERS, UNITED STATES, 1930-2005



Rates are age adjusted to the 2000 US population. Due to changes in ICD coding, numerator information has changed over time. Rates for cancers of the lung and bronchus, colon and rectum, and liver are affected by these changes. Source: US mortality data, 1960 to 2005, US Mortality Vol. 1930-1959, National Cancer Institute, Centers for Disease Control and Prevention, 2008.

Jemal, A, Siegel, R, Ward, E, et al. *Cancer Statistics, 2009. CA Cancer J Clin* 2009; 59:225. Copyright © 2009 American Cancer Society.

FIGURE 2
ANNUAL AGE-ADJUSTED CANCER DEATH RATES* AMONG FEMALES
FOR SELECTED CANCERS, UNITED STATES, 1930-2005



* Rates are age adjusted to the 2000 US standard population.

■ Uterus includes uterine cervix and uterine corpus. Due to changes in ICD coding, numerator information has changed over time. Rates for cancers of the uterus, ovary, lung and bronchus, and colon and rectum are affected by these changes. Source: US mortality data, 1960 to 2005, US Mortality Vol. 1930 to 1959, National Center for Health Statistics, Centers for Disease Control and Prevention, 2008.

Jemal, A, Siegel, R, Ward, E, et al. *Cancer Statistics, 2009. CA Cancer J Clin* 2009; 59:225. Copyright © 2009 American Cancer Society.

At the Marquette General Cancer Center from 1998 through 2001 we had 338 cases of Non-Small Cell Lung cancer and 104 cases of Small Cell Lung cancer. This report will detail the results of our treatments.

Lung cancer is one of the more preventable diseases because it seems to be highly correlated with environmental toxins. The most frequent environmental toxin that has been linked with lung cancer is, of course, exposure to cigarette smoke.

Smoking itself is the most common cause of cancer however second hand smoke exposure can also be linked to increased risk of developing lung cancer. In addition, exposure to radiation such as radon gas and to asbestos can also be linked to the development of lung cancer but is most important as potentiator of the carcinogenic effect of cigarette smoke.

Lung cancers are generally divided into two broad categories. The first category is that of a small cell anaplastic lung cancer. This is also referred to as small cell lung cancer or oat cell carcinoma or anaplastic neuroendocrine lung cancer. It accounts for about a one quarter of the cases and seems to be linked to smoking and environmental carcinogens such as radioactive gases.

The other larger and broader category of lung cancer is Non-Small Cell Lung cancer. This includes squamous cell lung cancer, adenocarcinoma, large cell anaplastic carcinoma and the broad category generally referred to as Non Small Cell Lung Cancer NOS (not otherwise specified). The NOS category is used when the type of cancer cell cannot be easily categorized as squamous or adeno or large cell neuroendocrine but nonetheless is not a small cell cancer and is definitely a cancer of lung origin. There are also some relatively rare cancers that occur in the lung such as carcinoids and sarcomas, which will not be discussed here since they form a different category of thoracic cancer.

Lung cancers can vary from very rapidly acting aggressive cancers such as small cell anaplastic carcinoma to very slow growing indolent cancers such as bronchioalveolar adenocarcinoma. As we learn more about what causes the lung cells to become malignant and what changes in their DNA cause them to change from normal to cancerous, we are also learning how to better identify what type of

cancer cells they are even when it's not clear under the microscope. The use of various immunohistochemical stains (IHC) and gene microarrays may teach us how to take advantage of some of these differences between individual cancers with treatments that are more specific for one type of Non Small Cell Lung Cancer than another.

Unfortunately, most lung cancers do not cause any symptoms when they are quite small. Thus the presentation of lung cancer often involves symptoms and signs of advanced disease with cancer spread to other organs (metastases) or pressure on organs within the chest due to growth of the tumor or the draining lymph nodes to which the cancer has spread. The most common presenting symptoms of lung cancer are seen in Table 1.

Table 1

LUNG CANCER – PRESENTING SIGNS & SYMPTOMS 2008 172 TOTAL CASES	
Symptoms	Percent Of Total Cases
Dyspnea	(57)53.1%
Persistent Cough	(42)24.4%
Weight Loss	(40)23.2%
Chest Discomfort	(28)16.3%
Hemoptysis	(19)8.1%
Lump or Thickening	(11)6.4%
No Symptoms (Asymptomatic)	(11)6.4%
Loss of Appetite	(10)5.8%
Hoarseness	(7)4.1%
Night Sweats	(3)1.7%
Anemia	(3)1.7%
Vomiting	(2)1.1%
Bone Pain	(2)1.1%
Nausea	(2)1.1%
Other Bleeding	(2)1.1%
Indigestion	(1)0.6%
Unexplained Fever	(1)0.6%

Dyspnea or shortness of breath is the most common presenting complaint followed closely by a persistent cough, weight loss and chest discomfort

Patients who develop lung cancer frequently have underlying lung disease with either emphysema or other forms of chronic obstructive pulmonary disease (COPD). Thus the symptoms of the preexisting lung problems may obscure the added symptoms of the developing lung cancer for variable periods of time.

There is also an increasing body of knowledge that there may be genetic traits that make certain people or certain families more susceptible to the adverse effects of smoking such as chronic bronchitis, emphysema or cancer. Those people who develop benign lung disease secondary to cancer may be at a greater risk for development of lung cancer than those who seem to be more resistant to the effects of tobacco.

In Table 2 one can see some of the contributing risk factors in patients diagnosed with lung cancer at the Marquette General Hospital in 2008. The majority of patients who were smoking at the time their cancer was discovered had anywhere from 25 – 99 pack years history of smoking.

Drug or Alcohol Abuse



BIOHAZARD

Table 2

CONTRIBUTING RISKS FACTORS IN PATIENTS DIAGNOSED WITH LUNG CANCER AT MARQUETTE GENERAL HOSPITAL IN 2008 (172 PATIENTS)	
	Number (Percent of Total)
Patient History of Other Cancer	
Yes	39(22.7%)
No	131(76.2%)
Unknown/Not Stated	2(1.1%)
Family History of Lung Cancer	
Yes	28(16.3%)
No	86(50.0%)
Unknown/Not Stated	58(33.7%)
Patient History of Lung Disorders	
Yes	96(55.8%)
No	69(40.1%)
Unknown/Not Stated	7(4.1%)
Occupational Risks	
Yes	13(7.6%)
No	25(14.5%)
Unknown/Not Stated	134(77.9%)
Alcohol Use	
Yes	68(39.5%)
No	59(34.3%)
Unknown/Not Stated	45(26.2%)
Smoking History	
None, Never Smoked	7(4.1%)
Stopped Smoking More than 5 years previously	54(31.4%)
Less than 25 Pack Years	6(3.5%)
25 – 49 Pack Years	30(17.4%)
50 – 99 Pack Years	37(21.5%)
100 or more Pack Years	5(2.9%)
Cigar or Pipe usage	1(.6%)
Smoker, Unknown # of Pack Years	21(12.2%)
Secondhand Smoke Exposure	1(.6%)
Unknown/Not Stated	10(5.8%)

The cancer risk from smoking unfortunately does not disappear as soon as the smoking is discontinued but continues for up to 20 years although it gradually diminishes by a few percent each year.

This is illustrated in Table 2 by the smoking history section which shows that only 4.1% of our lung cancer patients never smoked and that 31.4% had previously been smokers but had stopped more than 5 years before the discovery of their cancer.

Because lung cancer appears to be due to the cumulative damage to the lungs it is logical for lung cancer to be more frequent with advancing age. None of our patients in 2008 developed their lung cancer prior to age 40. The age range for the highest risk of cancer was between age 50 and age 80.

It can also be seen clearly from Table 3 that the number of cancers occurring in men versus women is essentially equal with 88 women and 84 men having been diagnosed with lung cancer in 2008.

Table 3

TOTAL NUMBER OF LUNG CANCER PATIENTS DIAGNOSED AT MARQUETTE GENERAL HOSPITAL IN 2008 BY AGE & GENDER			
AGE AT DIAGNOSIS	MALES (84 – 49%)	FEMALES (88 – 51%)	TOTAL COMBINED MALE & FEMALE (172 – 100%)
31 – 40 YEARS OF AGE	0(0%)	0(0%)	0(0%)
41 – 50 YEARS OF AGE	8(4.7%)	5(2.9%)	13(7.6%)
51 – 60 YEARS OF AGE	12(7.0%)	22(12.8%)	34(19.8%)
61 – 70 YEARS OF AGE	31(18.0%)	30(17.4%)	61(35.4%)
71 – 80 YEARS OF AGE	23(13.4%)	23(13.4%)	46(26.8%)
81 – 90 YEARS OF AGE	10(5.8%)	8(4.7%)	18(10.5%)
TOTAL	84(49%)	88(51%)	172(100%)

Although the diagnosis of lung cancer may be suspected by the patients' complaints, history and physical examination and radiologic studies, a tissue biopsy must be done in order to confirm that there is indeed a cancer present (since benign conditions can often have some of the same symptoms and the same x-ray

appearance as cancer) and to determine which of the several types of cancer is present. These biopsies can be obtained in various ways depending on the patient and the clinical situation.

Bronchoscopy or mediastinoscopy are frequently utilized to secure tissue and also CT guided biopsy of a chest mass or other abnormality can be used to secure tissue. It may take several days to a week or more to completely evaluate a lung cancer specimen depending on whether the category or type of cancer is immediately apparent under the microscope or whether additional immunohistochemical stains have to be done to help with that process.

In addition to diagnosing what type of cancer is present, we must determine how extensive the cancer is, whether it is localized to where it first originated, whether it has spread to adjacent structures or spread to draining lymph nodes and whether the cancer cells have spread to other areas of the body. The process of determining the extent of the cancer is called “staging”. In general stage 1 disease implies a relatively small cancer, which is localized to one area. Stage 2 cancers may imply a somewhat larger cancer, or a cancer that impinges on the larger airways or involves some of the hilar lymph nodes in the root of the lung. Stage 3 disease implies a more extensive spread of the cancer within the lung in which it originated and/or spread to lymph nodes deeper within the center of the chest. Stage 4 disease generally implies either spread to other organs or spread to the lining of the chest or heart or to the other lung. The newly revised staging schema for non-small cell lung cancers is seen in [Table 4](#). The staging schema for small cell lung cancer is currently much simpler and consists of either localized disease or extensive disease depending on whether the cancer has spread beyond the chest or not.

Table 4

SEVENTH EDITION OF TNM CLASSIFICATION OF LUNG TUMORS: CHANGES FROM SIXTH EDITION

Primary Tumor (T):
T1 lesions are divided based upon size into T1a (≤ 2 cm) and T1b (> 2 cm but ≤ 3 cm)
T2 lesions are divided into T2a (> 3 cm but ≤ 5 cm) and T2b (> 5 cm but ≤ 7 cm)
T2 tumors > 7 cm are reclassified as T3
T4 tumors with separate tumor nodules in the same lobe as the primary tumor are reclassified as T3
Additional nodules in a different lobe of same lung are reclassified as T4 rather than M1
Malignant pleural or pericardial effusions or pleural nodules are now classified as metastasis (M1a) rather than T4
Regional Nodes (N):
No changes
Metastasis (M):
Subdivided into M1a (malignant pleural or pericardial effusion, pleural nodules, nodules in contralateral lung) and M1b (distant metastasis)
Stage Grouping:
T2aN1M0 lesions are classified as IIA, rather than IIB
T2bN0M0 lesions are classified as IIA, rather than IB
T3 (> 7 cm), N0M0 lesions are classified as IIB, rather than IB
T3 (> 7 cm), N1M0 lesions are classified as IIIA, rather than IIB
T3N0M0 (nodules in same lobe) lesions are classified as IIB, rather than IIIB
T3N1M0 or T3N2M0 (nodules in same lobe) are classified as IIIA, rather than IIIB
T4M0 (ipsilateral lung nodules) lesions are classified as IIIA (if N0 or N1) and IIIB (if N2 or N3), rather than stage IV
T4M0 (direct extension) lesions are classified as IIIA (if N0 or N1), rather than IIIB
Malignant pleural effusions (M1a) are classified as IV, rather than IIIB

Groome, PA, Bolejack, V, Crowley, JJ, et al. J Thorac Oncol 2007; 2:694.

Goldstraw, P, et al. J Thorac Oncol 2007; 2:706.

Staging and determination of cell type are the main determinants of the outcome of treatment of lung cancer. Table 5 shows the 5-year survival for patients with various stages of non-small cell lung cancer. Since these statistics in Table 5 were gathered between 1975 and 1988 the outlook may well have improved with better staging techniques and better treatments, however the survival outlook for even Stage 1A disease was only 61% in 1989 and has improved slowly over time.

Table 5

PROGNOSIS OF NON-SMALL CELL LUNG CANCER BY STAGE

Clinical Stage	N	Five-Year Survival (Percent)
IA	687	61
IB	1189	38
IIA	29	34
IIB	357	24
IIIA	511	13
IIIB	1030	5
IV	1427	1

Adapted from Mountain, CF, Chest 1997; 11:1710. Data are from 4351 patients treated at the University of Texas MD Anderson Cancer Center and 968 patients treated by the National Cancer Institute cooperative Lung Cancer Study Group between 1975 and 1988.

We have seen significant improvements in surgical techniques, which allow people with relatively poor lung function or other co-morbid medical problems to undergo surgeries that previously would not have been tolerated. Radiation therapy, which is a mainstay of treatment in cancers which cannot be surgically removed, has improved markedly in technique since 1988 and continues to improve with new innovations. Chemotherapy has also improved considerably since 1988 with many new drugs and combinations introduced. We have also learned better how to combine the various modalities of surgery, radiation and chemotherapy to get better effect. In addition, new non-chemotherapy biological agents and targeted therapies are being developed which will eventually help to improve not only the tolerability of treatment and response rate of lung cancers but eventually to improve overall survival as well.

In recent years we have learned that giving chemotherapy after resection of a lung cancer can increase the cure rate. This is known as “adjuvant” therapy. We have also learned that some types of lung cancer such as squamous cell cancer should not be treated with drugs such as Avastin because of increased risk of certain side

effects. Other cancers such as adenocarcinomas in non-smokers may respond to an oral targeted agent such as Tarceva.

The potential combinations of surgery, radiation therapy and systemic therapy (chemotherapy and targeted agents) are further complimented by the use of interventional radiology techniques such as radio-frequency ablation (RFA). In RFA a microwave-emitting probe can be inserted much like a biopsy needle into a localized cancer and can destroy the tumor with minimal damage to surrounding tissue.

Tables 6, 7, 8, 9 show an attempt to categorize the various combinations of treatments that are used in various stages of non-small cell lung cancer and its sub-groups of squamous cell cancer and adenocarcinoma and also in small cell anaplastic carcinoma.



TABLE 6

NON-SMALL CELL LUNG CANCER (includes Carcinoma undifferentiated, Non Small cell carcinoma, Neuroendocrine carcinoma & Carcinoid histologies) DIAGNOSED AT MARQUETTE GENERAL HOSPITAL IN 2008 BY STAGE & TREATMENT (69 CASES)

TREATMENT	STAGE AT DIAGNOSIS			
	LOCAL	REGIONAL	DISTANT	UNKNOWN
CARBOPLATIN WITH RT TO LUNG				
	0(0%)	1(1.4%)	0(0%)	0(0%)
CARBOPLATIN & OTHER AGENTS:				
TAXOL	0(0%)	3(4.3%)	1(1.4%)	OTHER SITE 0(0%)
ETOPOSIDE	0(0%)	1(1.4%)	0(0%)	0(0%)
ALIMTA	0(0%)	1(1.4%)	0(0%)	0(0%)
TAXOL & GEMCITIBINE	0(0%)	1(1.4%)	0(0%)	2(2.8%) 1 LIVER, 1 BONE
TAXOL & AVASTIN	0(0%)	0(0%)	1(1.4%)	LUNG 0(0%)
TAXOTERE & AVASTIN	0(0%)	2(2.8%)	0(0%)	0(0%)
TAXOL & AVASTIN WITH LOBECTOMY	0(0%)	0(0%)	1(1.4%)	1 BONE
CARBOPLATIN & OTHER AGENTS WITH RADIATION				
TAXOL & RT TO LUNG	0(0%)	5(7.2%)	2(2.8%)	OTHER SITES 0(0%)
TAXOTERE & RT TO DISTANT METS	0(0%)	0(0%)	2(2.8%)	1 LUNG 1 BRAIN 0(0%)
TAXOL & RT TO DISTANT METS	0(0%)	0(0%)	5(7.2%)	2 BRAIN 1 BRAIN/BONE 1 LIVER/BONE 1 BONE/SPINE 0(0%)
CARBOPLATIN WITH OTHER AGENT(S) & SURGERY				
TAXOL/AVASTIN WITH LOBECTOMY	1(1.4%)	0(0%)	0(0%)	0(0%)
TAXOL & LOBECTOMY	0(0%)	0(0%)	1(1.4%)	1 BRAIN 0(0%)
CARBOPLATIN WITH OTHER AGENT(S) WITH SURGERY & RADIATION TO DISTANT METS				
GEMCITIBINE/ALIMTA	0(0%)	0(0%)	1(1.4%)	BRAIN (CRANIOTOMY & RT TO BRAIN) 0(0%)
ALIMTA ONLY				
	0(0%)	1(1.4%)	0(0%)	0(0%)
ALIMTA WITH RT TO DISTANT METS & LUNG				
	0(0%)	0(0%)	1(1.4%)	LUNG/BRAIN 0(0%)
CISPLTAIN WITH OTHER AGENT(S) WITH RT TO LUNG				
ETOPOSIDE	0(0%)	1(1.4%)	0(0%)	0(0%)
TAXOTERE	0(0%)	1(1.4%)	0(0%)	0(0%)
ETOPOSIDE/TAXOTERE	0(0%)	1(1.4%)	0(0%)	0(0%)
RADIATION ONLY				
LUNG	0(0%)	1(1.4%)	0(0%)	0(0%)
DISTANT SITE(S) ONLY	0(0%)	0(0%)	6(8.6%)	5 BRAIN 1 BONE 0(0%)
RADIOFREQUENCY ABLATION				
	3(4.3%)	1(1.4%)	1(1.4%)	1 DISTANT LYMPH AND OTHER 0(0%)
PNEUMECTOMY WITH RADIATION CHEST WALL				
	0(0%)	1(1.4%)	0(0%)	0(0%)
LOBECTOMY ONLY				
	0(0%)	3(4.3%)	0(0%)	0(0%)
WEDGE RESECTION				
	1(1.4%)	1(1.4%)	0(0%)	0(0%)
NO TREATMENT				
	5(7.2%)	2(2.8%)	6(8.6%)	2 BONE 1 DISTANT LYMPH NODES (DLN) 1 DLN & OTHER SITE 1 DLN & LIVER 1 OTHER SITE 2(2.8%)
TOTAL	10(14.4%)	27(39.1%)	28(40.5%)	4(5.7%)

TABLE 7

SQUAMOUS CELL LUNG CANCER DIAGNOSED AT MARQUETTE GENERAL HOSPITAL IN 2008 BY STAGE & TREATMENT (35 CASES)				
TREATMENT	STAGE AT DIAGNOSIS			
	LOCAL	REGIONAL	DISTANT	UNKNOWN
CARBOPLATIN WITH OTHER AGENT(S)				
TAXOTERE	0(0%)	1(2.8%)	0(0%)	0(0%)
GEMZAR	0(0%)	0(0%)	1(2.8%)	1 LUNG 0(0%)
TAXOL	0(0%)	0(0%)	1(2.8%)	1 LUNG 0(0%)
CARBOPLATIN WITH OTHER AGENT(S) & RADIATION TO LUNG				
NAVELBINE/GEMCITIBIN E	0(0%)	1(2.8%)	0(0%)	0(0%)
TAXOTERE	0(0%)	2(5.7%)	0(0%)	0(0%)
TAXOL	0(0%)	2(5.7%)	2(5.7%)	1 LIVER 1 BONE/OTHER 0(0%)
ETOPOSIDE	0(0%)	1(2.8%)	0(0%)	0(0%)
CARBOPLATIN WITH OTHER AGENT(S) WITH RADIATION TO DISTANT METS				
	0(0%)	0(0%)	2(5.7%)	1 LUNG/BRAIN /BONE 1 BONE 0(0%)
CARBOPLATIN WITH OTHER AGENT(S) & RADIOFREQUENCY ABLATION OF LUNG PRIMARY				
	0(0%)	0(0%)	0 (0%)	1(2.8%)
CISPLATIN WITH OTHER AGENT(S)				
ETOPOSIDE/TAXOTERE/ NAVELBINE	0(0%)	1(2.8%)	0(0%)	0(0%)
ETOPOSIDE	0(0%)	1(2.8%)	0(0%)	0(0%)
CISPLATIN WITH OTHER AGENT(S) WITH RADIATION TO LUNG				
TAXOTERE	0(0%)	0(0%)	1(2.8%)	1 BONE 0(0%)
CISPLATIN WITH OTHER AGENT(S) WITH SURGERY TO LUNG				
TAXOTERE	0(0%)	1(2.8%)	0(0%)	0(0%)
LOBECTOMY				
	2(5.7%)	0(0%)	0(0%)	0(0%)
LOBECTOMY WITH RADIATION TO LUNG				
	0(0%)	1(2.8%)	0(0%)	0(0%)
WEDGE RESECTION				
	0(0%)	0(0%)	0(0%)	1(2.8%)
PNEUMONECTOMY				
	0(0%)	1(2.8%)	0(0%)	0(0%)
RADIOFREQUENCY ABLATION				
	0(0%)	1(2.8%)	0(0%)	0(0%)
SURGERY TO DISTANT SITE				
	0(0%)	0(0%)	1(2.8%)	1 LUNG/OTHER 1(2.8%)
NO TREATMENT				
	1(2.8%)	3(8.5%)	3(8.5%)	1 BRAIN 1 LIVER/BONE 1 OTHER 2(5.7%)
TOTAL	3(8.5%)	16(45.7%)	11(31.4%)	5(14.2%)

TABLE 8

ADENOCARCINOMA CELL LUNG CANCER DIAGNOSED AT MARQUETTE GENERAL HOSPITAL IN 2008 BY STAGE & TREATMENT (46 CASES)					
TREATMENT	STAGE AT DIAGNOSIS				
	LOCAL	REGIONAL	DISTANT		UNKNOWN
CARBOPLATIN WITH OTHER AGENT(S)					
TAXOL	0(0%)	1(2.1%)	2(4.3%)	2 BONE	0(0%)
TAXOL/AVASTIN	0(0%)	1(2.1%)	3(6.5%)	1 LUNG 1 BONE/OTHER 1 OTHER	1(2.1%)
TAXOTERE/AVASTIN/ TARCEVA	1(2.1%)	0(0%)	0(0%)		0(0%)
CARBOPLATIN WITH OTHER AGENT(S) & SURGERY TO LUNG(LOBECTOMY)					
GEMCITIBINE	0(0%)	1(2.1%)	0(0%)		0(0%)
ALIMTA	0(0%)	1(2.1%)	0(0%)		0(0%)
CARBOPLATIN WITH OTHER AGENT(S) & RT TO LUNG					
TAXOTERE	0(0%)	1(2.1%)	0(0%)		0(0%)
TAXOL	0(0%)	2(4.3%)	0(0%)		0(0%)
CARBOPLATIN WITH OTHER AGENT(S) WITH RT TO LUNG & SURGERY TO DISTANT METS					
TAXOL	0(0%)	0(0%)	1(2.1%)	1 DISTANT LYMPH NODES/OTHER RADIOFREQUENCY ABLATION TO METS	0(0%)
TARCEVA ONLY					
	0(0%)	0(0%)	1(2.1%)	1 BONE	1(2.1%)
CISPLATIN WITH OTHER AGENT(S) & SURGERY TO LUNG					
TAXOTERE	0(0%)	2(4.3%) 2 LOBECTOMY	0(0%)		0(0%)
CISPLATIN WITH OTHER AGENT(S) & RT LUNG					
ETOPOSIDE	0(0%)	1(2.1%)	0(0%)		0(0%)
CISPLATIN WITH OTHER AGENT(S) WITH RT LUNG & DISTANT METS					
ETOPOSIDE	0(0%)	0(0%)	1(2.1%)	1 BRAIN	0(0%)
RADIATION TO SITE DISTANT METS ONLY					
	0(0%)	0(0%)	2(4.3%)	1 BRAIN 1 LIVER/BONE	0(0%)
WEDGE RESECTION					
	3(6.5%)	0(0%)	0(0%)		1(2.1%)
LOBECTOMY					
	6(13.0%)	3(6.5%)	0(0%)		0(0%)
PNEUMONECTOMY					
	0(0%)	1(2.1%)	0(0%)		0(0%)
RADIOFREQUENCY ABLATION					
	2(4.3%)	1(2.1%)	0(0%)		1(2.1%)
TOTAL	12(26.0%)	18(39.1%)	10(21.7%)		6(13.0%)

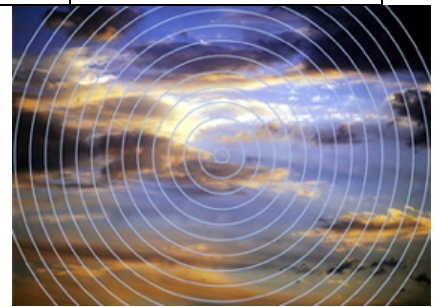
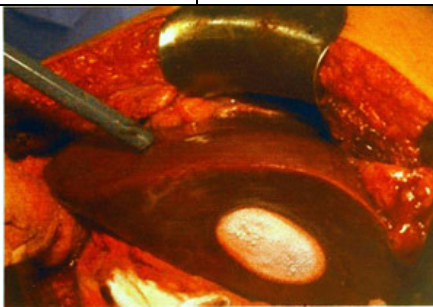


TABLE 9

SMALL CELL LUNG CANCER DIAGNOSED AT MARQUETTE GENERAL HOSPITAL IN 2008 BY STAGE & TREATMENT (22 CASES)					
TREATMENT	STAGE AT DIAGNOSIS				
	LOCAL	REGIONAL	DISTANT	UNKNOWN	
CARBOPLATIN WITH OTHER AGENT(S)					
ETOPOSIDE	0(0%)	1(4.5%)	1(4.5%)	0(0%)	
CARBOPLATIN WITH OTHER AGENT(S) WITH RADIATION TO LUNG					
ETOPOSIDE	0(0%)	2(9.0%)	0(0%)	1(4.0%)	
CARBOPLATIN WITH OTHER AGENT(S) WITH RADIATION TO LUNG WITH PROPHYLACTIC BRAIN RADIATION					
ETOPOSIDE	0(0%)	2(9.0%)	0(0%)	0(0%)	
CARBOPLATIN WITH OTHER AGENT(S) WITH RADIATION TO DISTANT METS					
ETOPOSIDE	0(0%)	0(0%)	1(4.5%)	1 BRAIN	0(0%)
CISPLATIN WITH OTHER AGENT(S)					
ETOPOSIDE	0(0%)	0(0%)	4(18.0%)	1 BONE 1 LIVER 1 LIVER/BONE 1 DISTANT LYMPH NODES/OTHER	1(4.5%)
CISPLATIN WITH OTHER AGENT(S) WITH RADIATION TO LUNG					
ETOPOSIDE	0(0%)	0(0%)	0(0%)	1(4.5%)	
CISPLATIN WITH OTHER AGENT(S) WITH RADIOFREQUENCY ABLATION OF LUNG PRIMARY					
ETOPOSIDE	1(4.5%)	0(0%)	0(0%)	0(0%)	
CISPLATIN WITH OTHER AGENT(S) WITH RADIATION TO LUNG & OROPHYLACTIC BRAIN RADIATION					
ETOPOSIDE	0(0%)	3(13.6%)	0(0%)	0(0%)	
CHEMO NOS					
	0(0%)	2(9.0%)	0(0%)	0(0%)	
RADIATION TO DISTANT METS					
	0(0%)	0(0%)	1(4.5%)	1 BRAIN	0(0)
NO TREATMENT					
	0(0%)	0(0%)	1(4.5%)	1 BRAIN	0(0%)
TOTAL	1(4.5%)	10 (45.4%)	8(36.3%)	3(13.6.%)	

While relief of symptoms and other forms of palliation is extremely important, improvement of survival is an equally important goal. To ensure that the quality of our diagnosis, staging and treatment is comparable to national standards, we routinely compare our 5-year results with those of the combined National Cancer DataBase. As shown in Figures, 3, 4 and 5 our survival rates compare quite favorably with those of the National Cancer DataBase (NCDB).

FIGURE 3

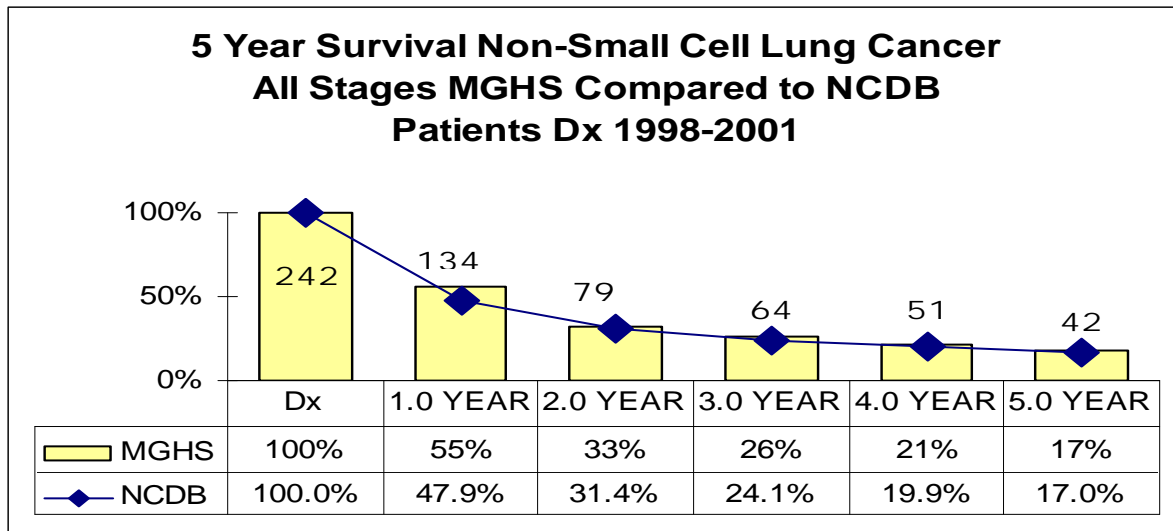


FIGURE 4

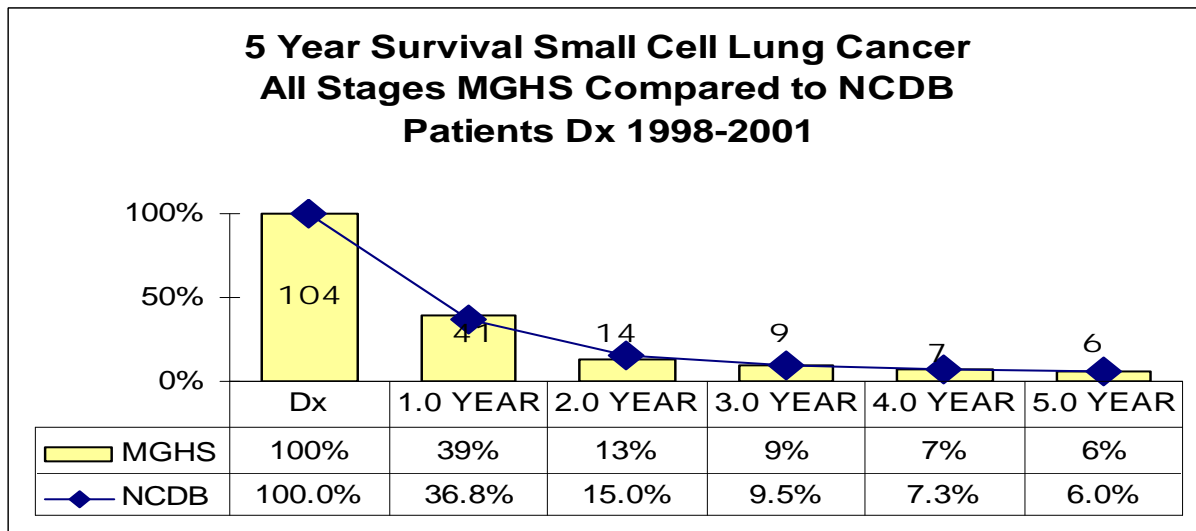
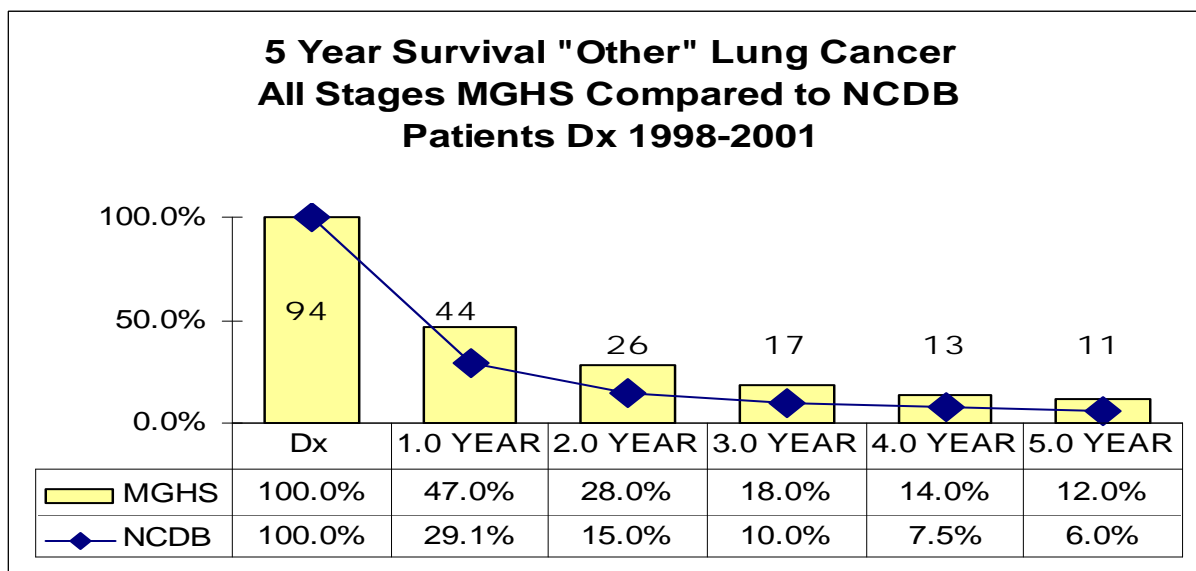


FIGURE 5



We have chosen the years 1998 thru 2001 because in order to have 5-year survival data, one must have at least a 5-year span after the last patient is treated and then several years to ensure that all of the data is collected and collated. Thus our 2007 and 2008 patients cannot be used for this type analysis since many of them are still undergoing treatment or have only recently finished treatment. For the same reasons, the NCDB chose the years 1998 thru 2001 which enables us to compare contemporaneous data.

Figures 6 through 10 show the survival statistics for our patients with squamous cell lung cancer. Going from Stage 1 squamous cell lung cancer (Figure 6) to Stage IV squamous cell lung cancer (Figure 9) the effect of increasing stage on survival can definitely be seen. Figure 10 shows the combined 5-year survival for all of our squamous cell lung cancer patients from 1998 to 2001.

FIGURE 6

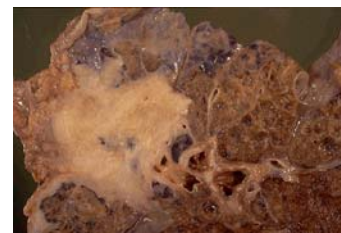
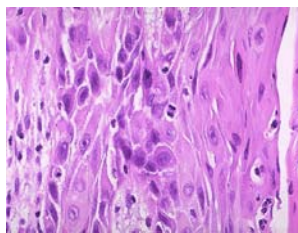
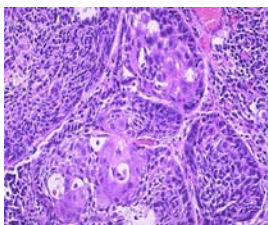
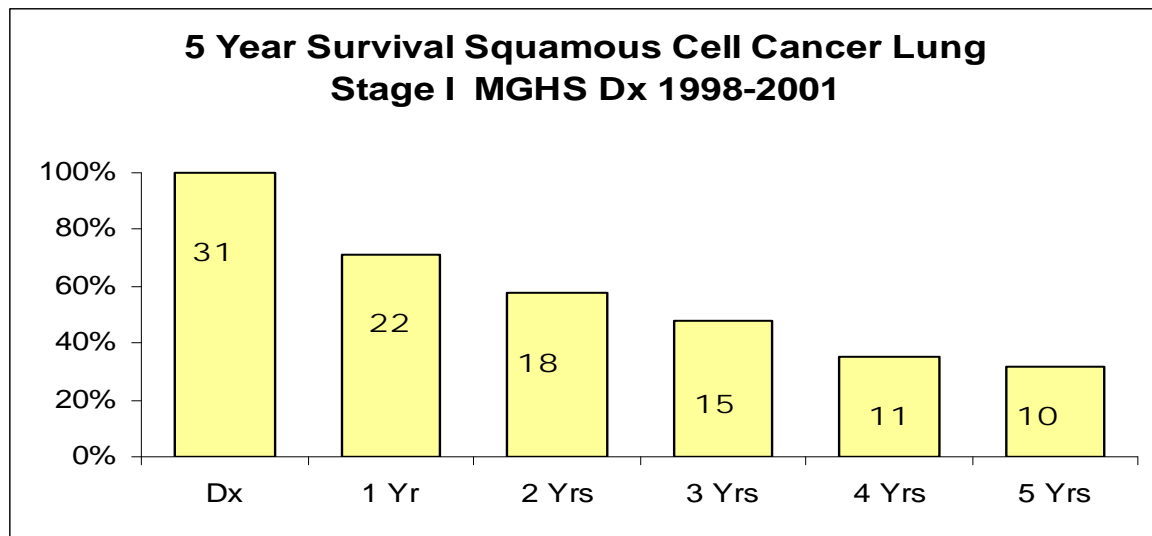


FIGURE 7

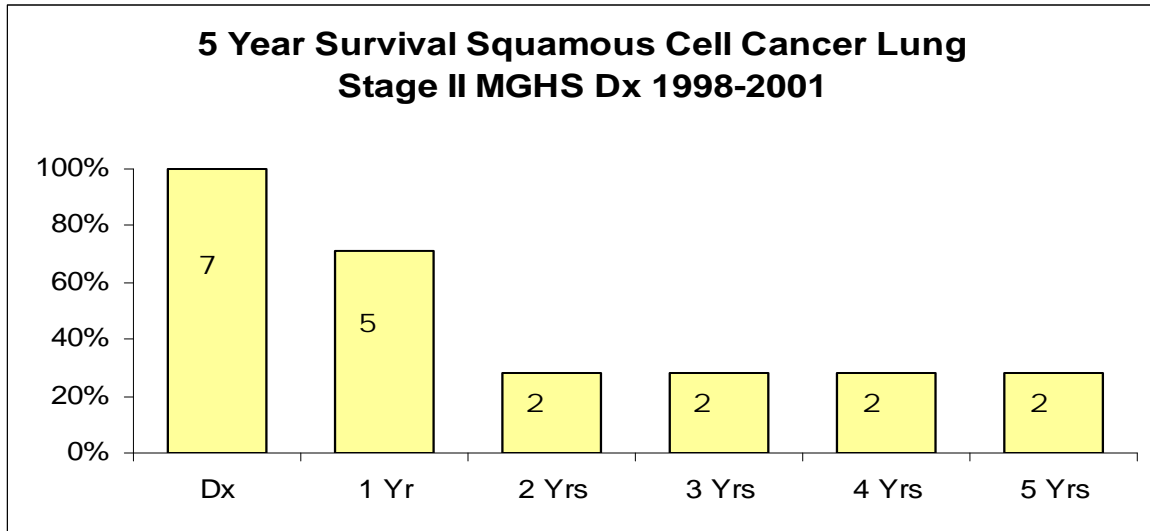


FIGURE 8

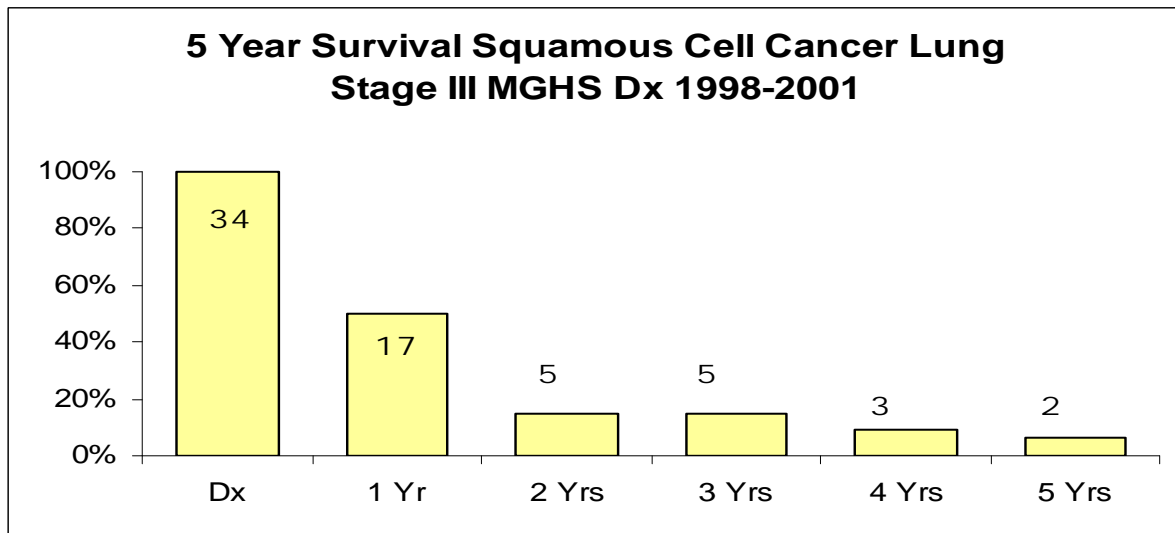


FIGURE 9

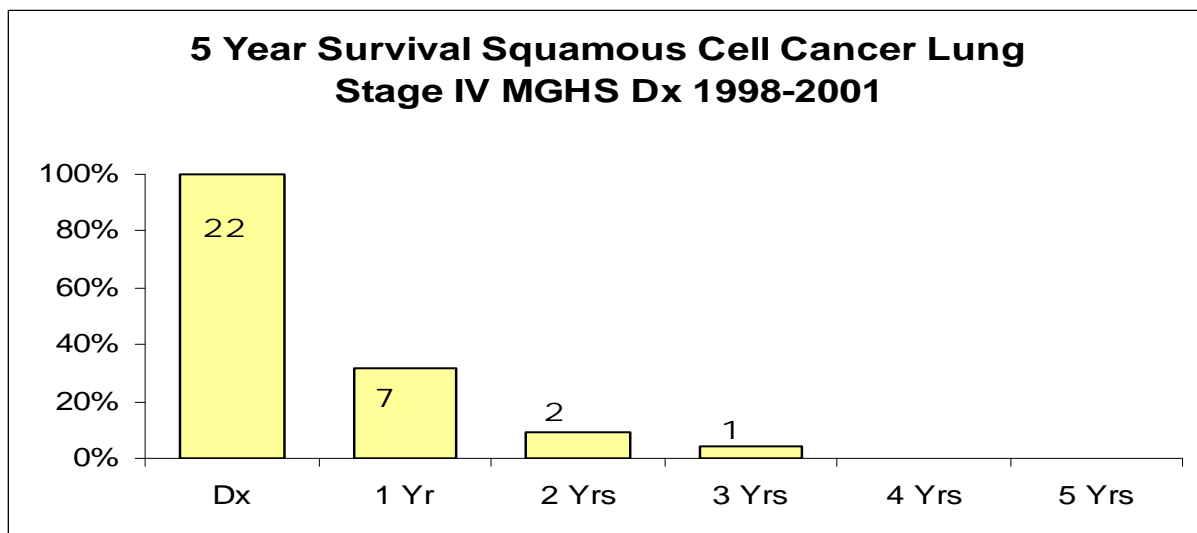
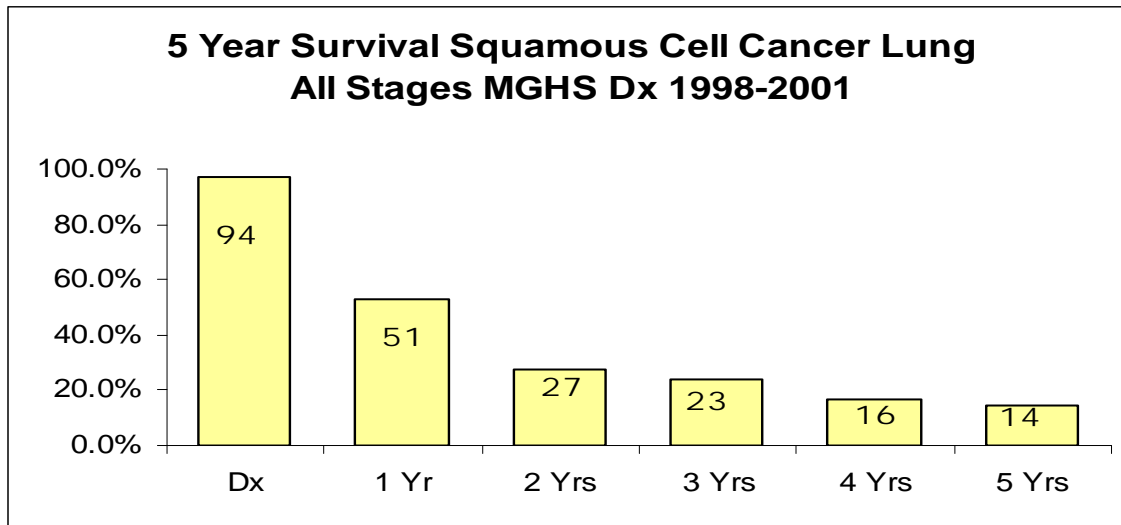


FIGURE 10



Similarly, Figures 11 through 15 show the survival characteristics of our patients with adenocarcinoma and again the effect of stage can be seen on their survival. The results show very little significant survival difference between squamous cell carcinoma and adenocarcinoma although when all stages are combined the overall survival of patients with adenocarcinoma is slightly better than that of squamous carcinoma.

FIGURE 11

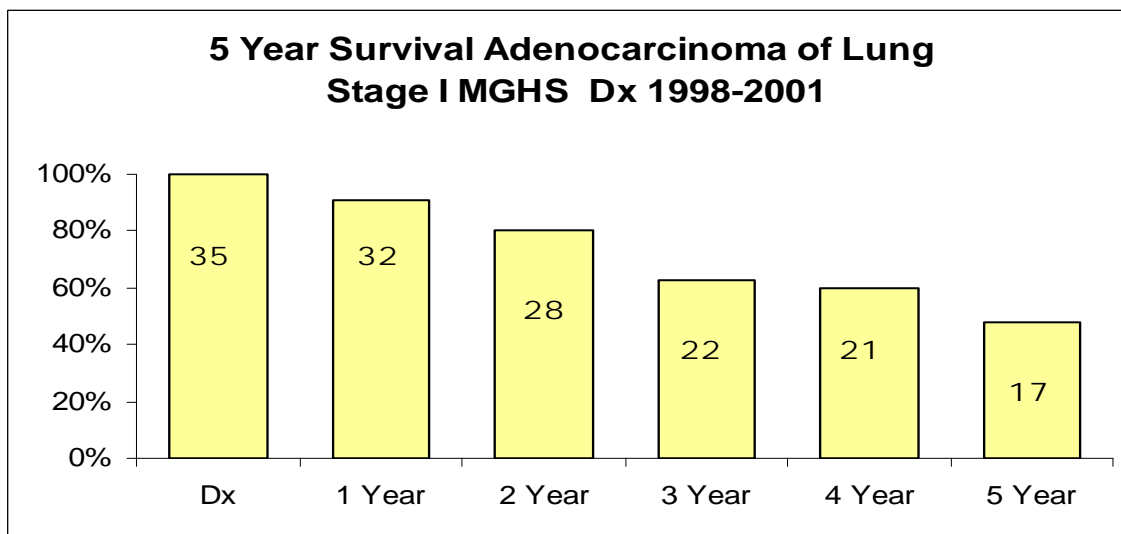


FIGURE 12

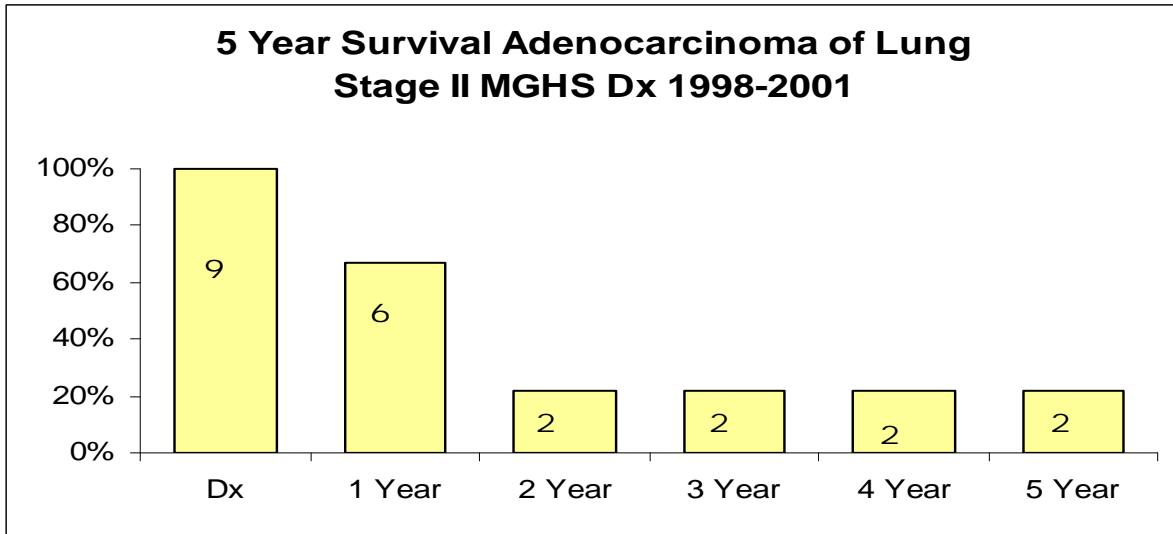


FIGURE 13

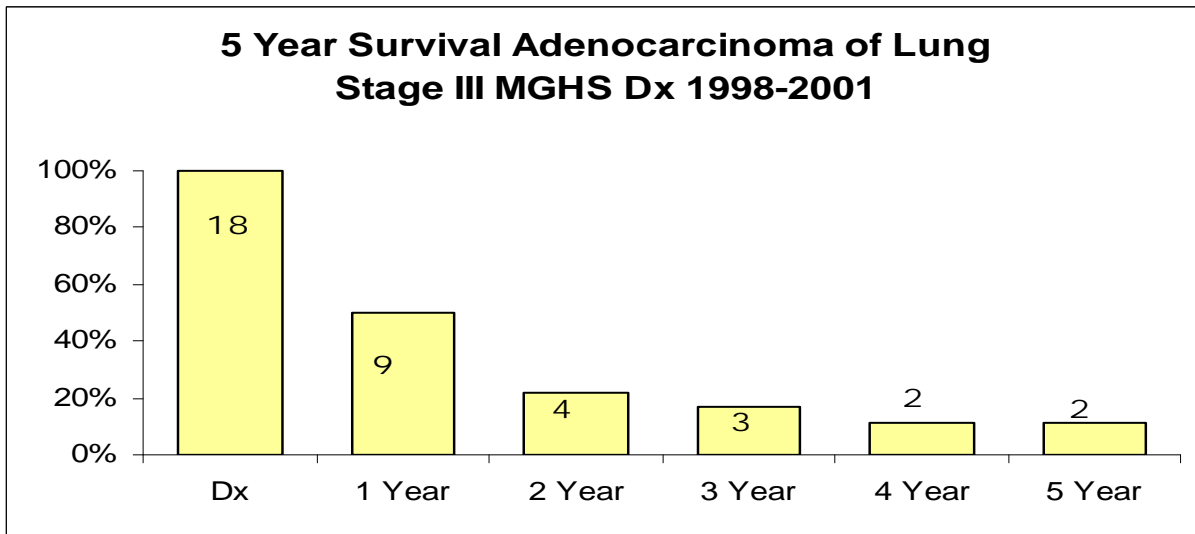


FIGURE 14

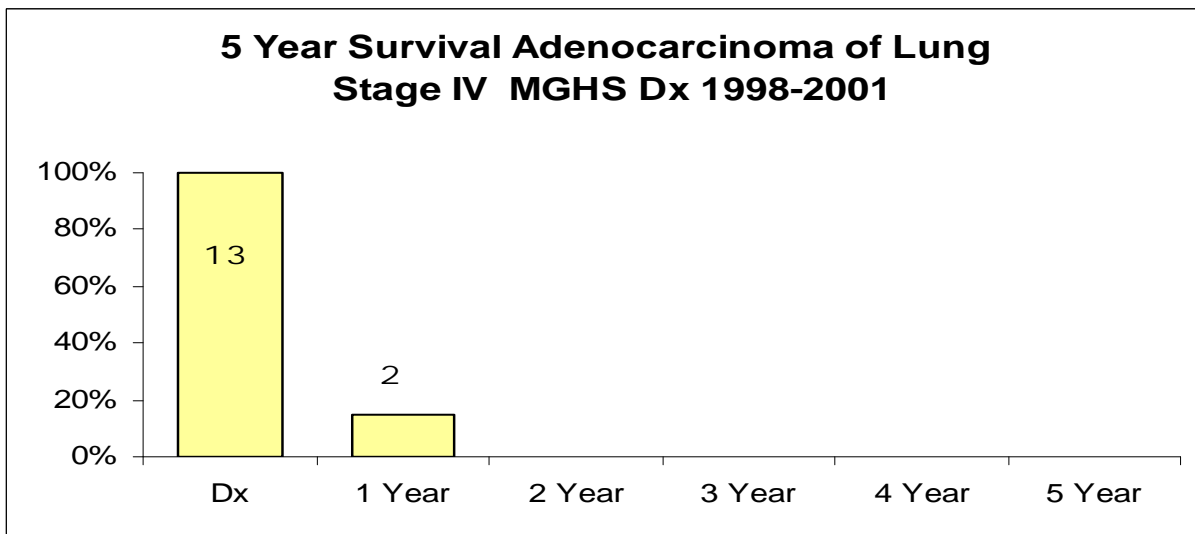
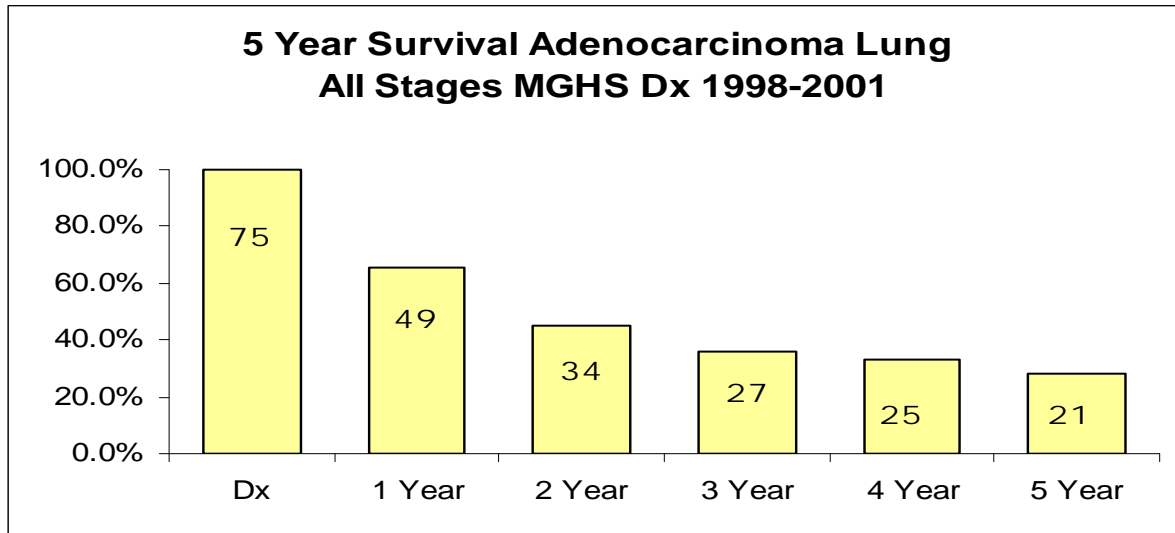


FIGURE 15



Figures 16 through 20 show the 5-year survival for a “catch-all” diagnostic category, which includes multiple poorly defined types of cancer. The combined survival statistics for this cancer diagnostic group are actually somewhat better than for adenocarcinoma or squamous.

FIGURE 16

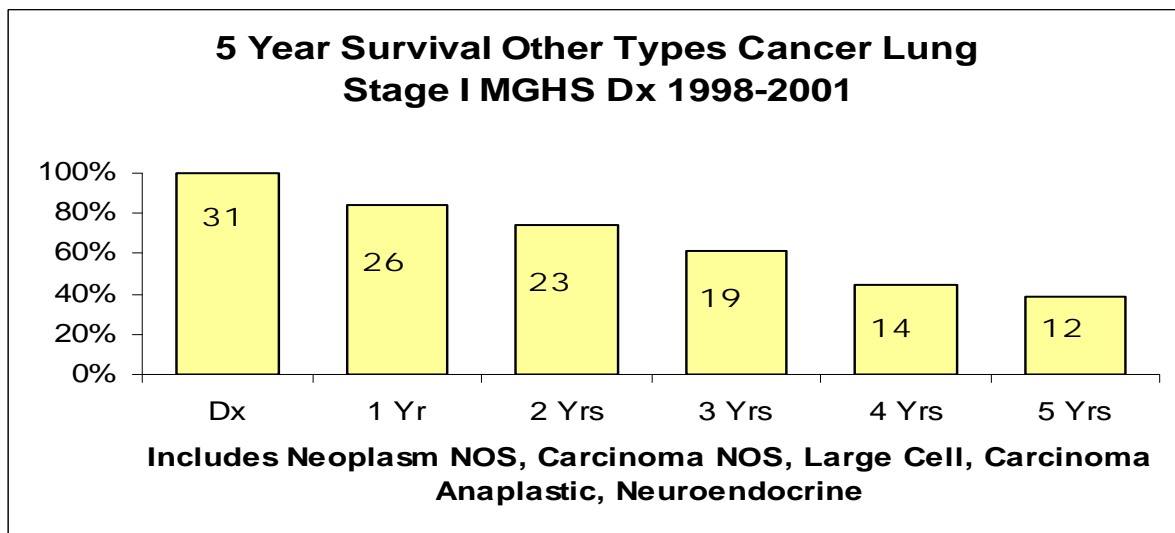


FIGURE 17

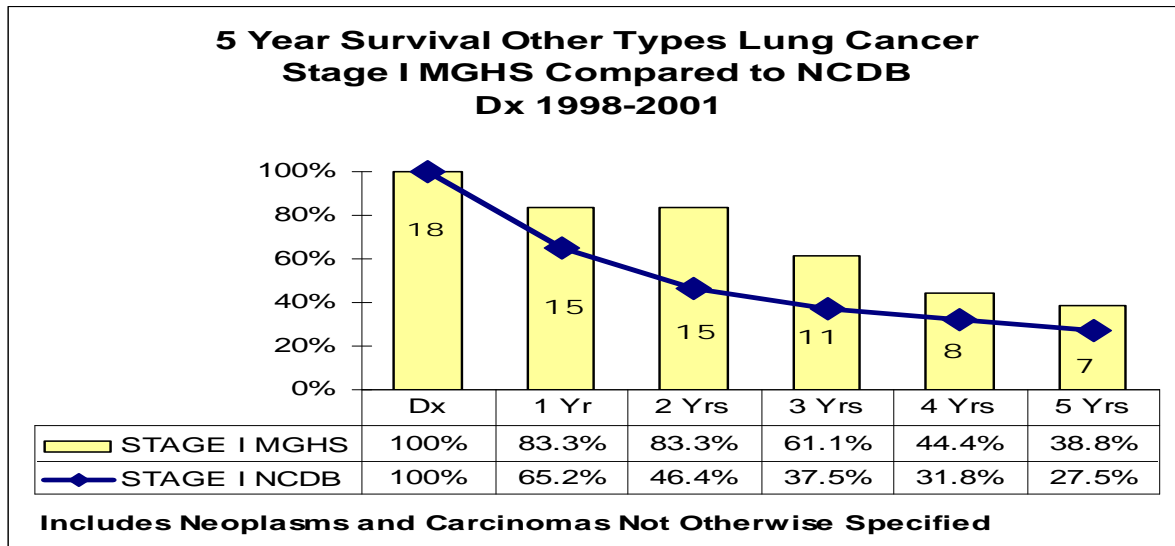


FIGURE 18

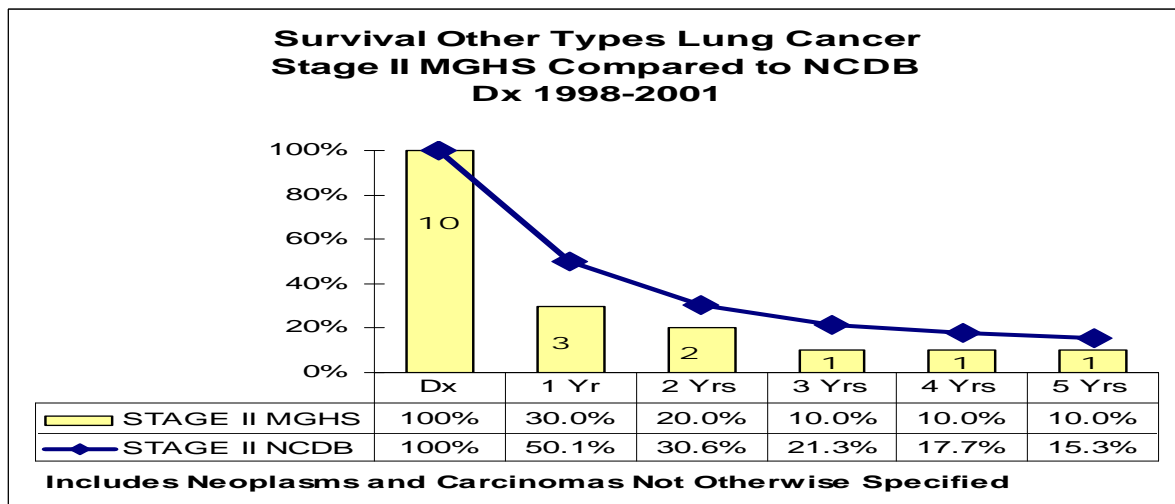


FIGURE 19

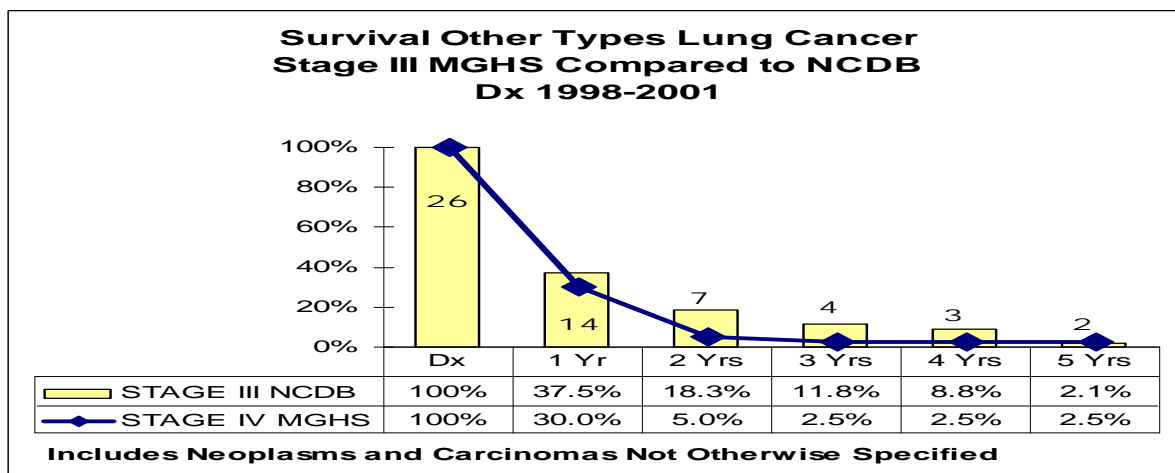
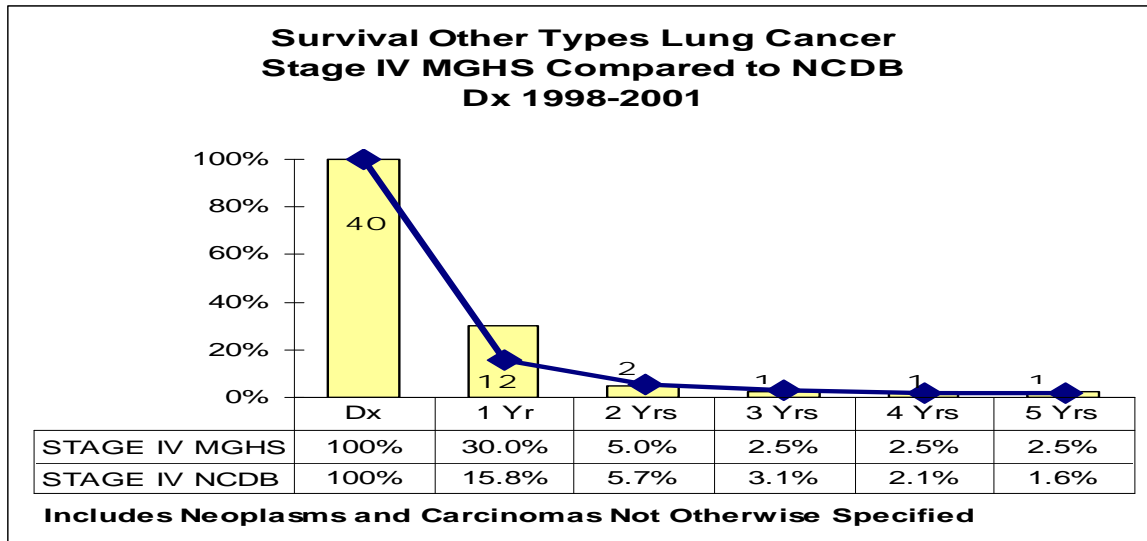


FIGURE 20



Again the effect of stage at presentation is seen very clearly if the various non-small cell neuroendocrine cancers are eliminated as shown in [Figure 21](#). When all of the non-small lung cancers are combined for survival statistics according to stage in [Figures 21 thru 24](#) we can see clearly that our overall results are very compatible with those of the NCDB. (The yellow vertical bars represent Marquette General Cancer Center's numbers and the dark blue line with the diamond icons represents the NCDB statistics.)

FIGURE 21

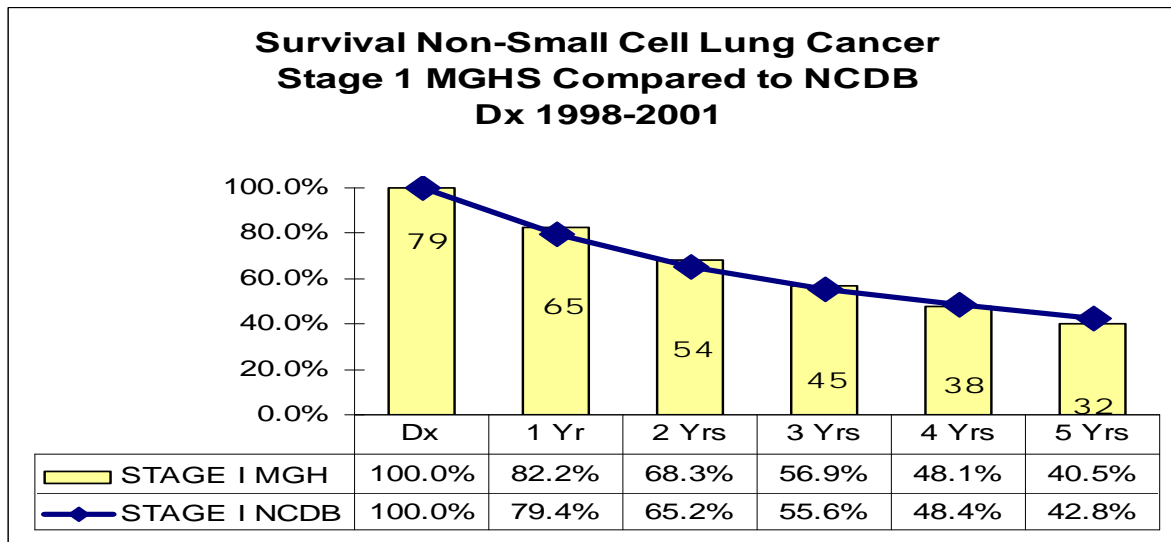


FIGURE 22

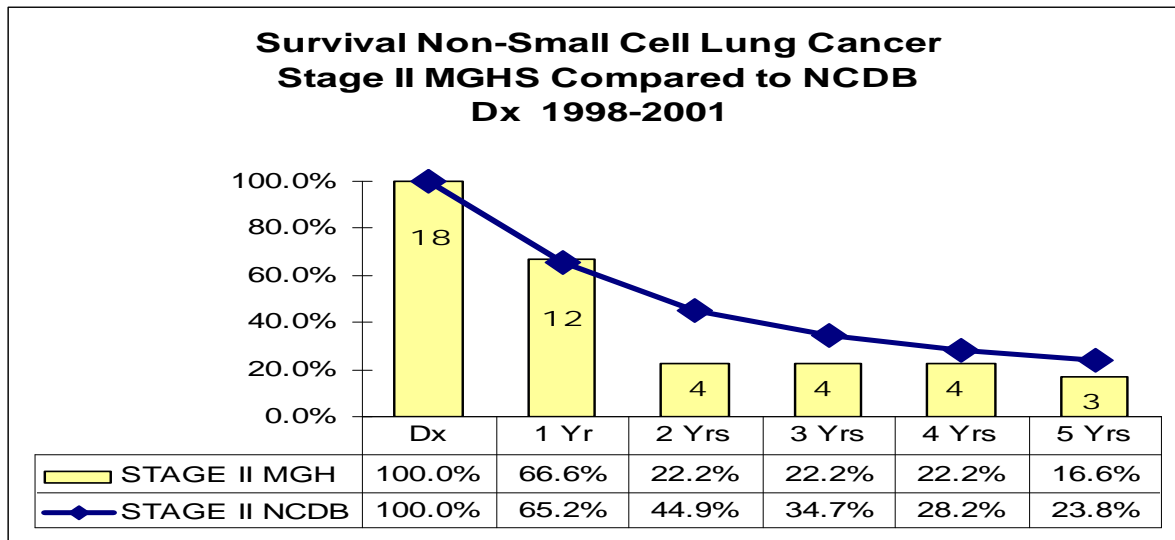


FIGURE 23

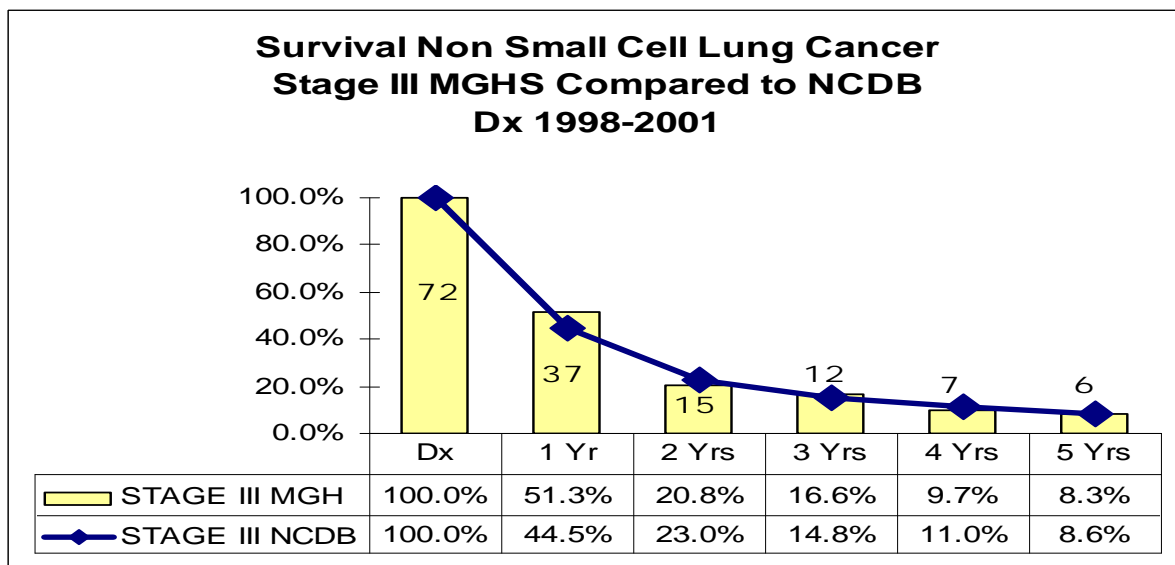
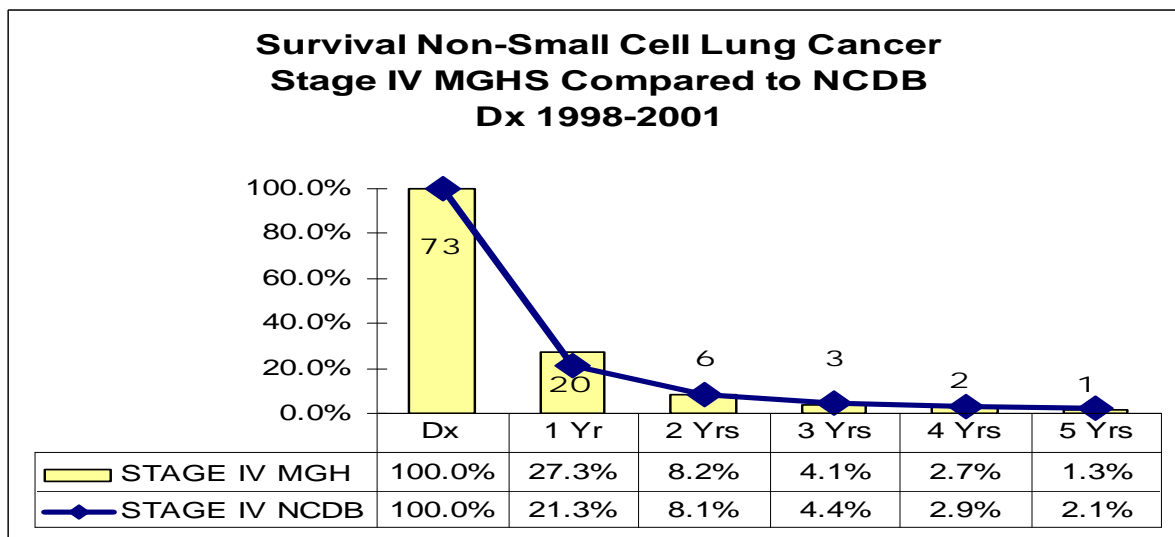


FIGURE 24



Our survival data for small cell anaplastic lung cancer by stage is represented in Figures 25 through 28. Again the survival statistics of our patients and the NCDB are quite comparable. One can also see that the survival statistics even in Stage 1 and II small cell lung cancer are worse than for non-small cell lung cancer. Nonetheless, there are a few long-term survivors even for those patients with Stage III small cell lung cancer.

FIGURE 25

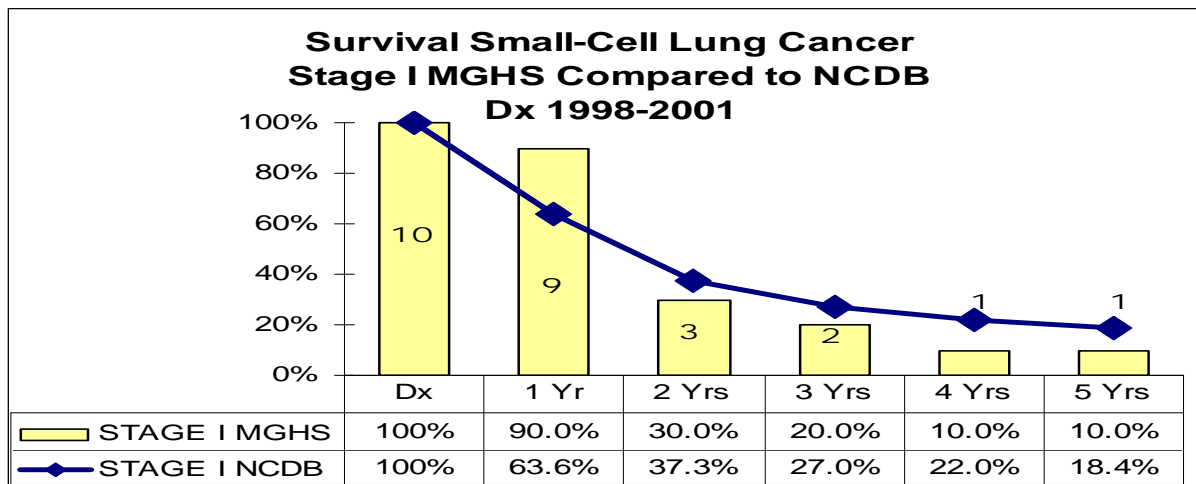


FIGURE 26

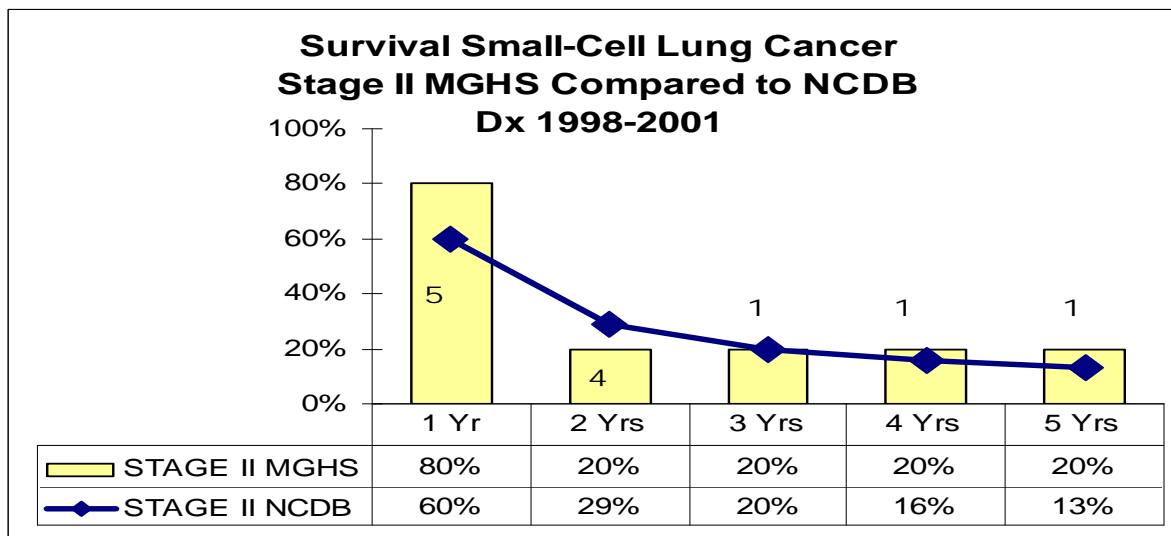


FIGURE 27

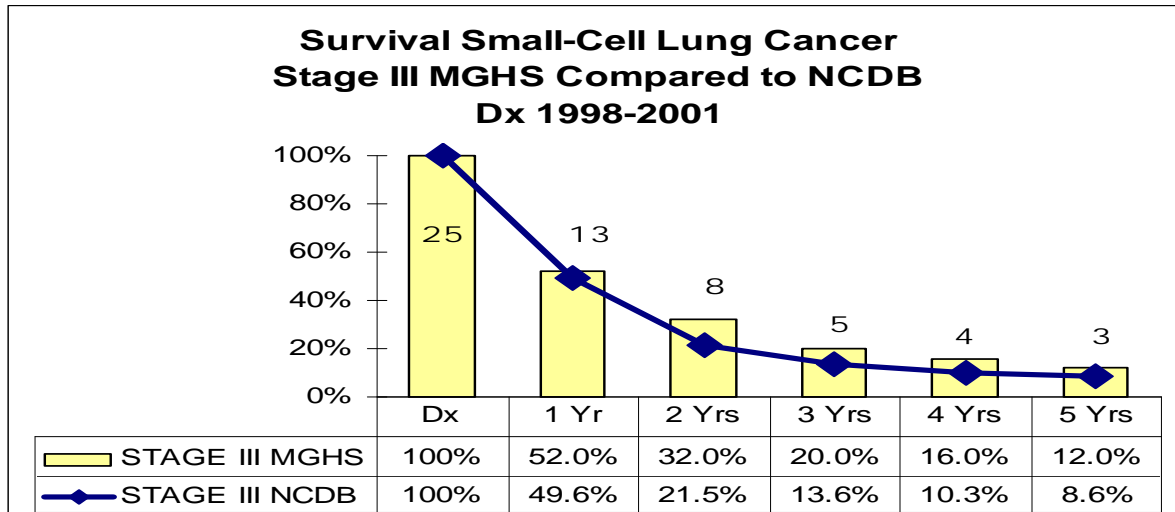
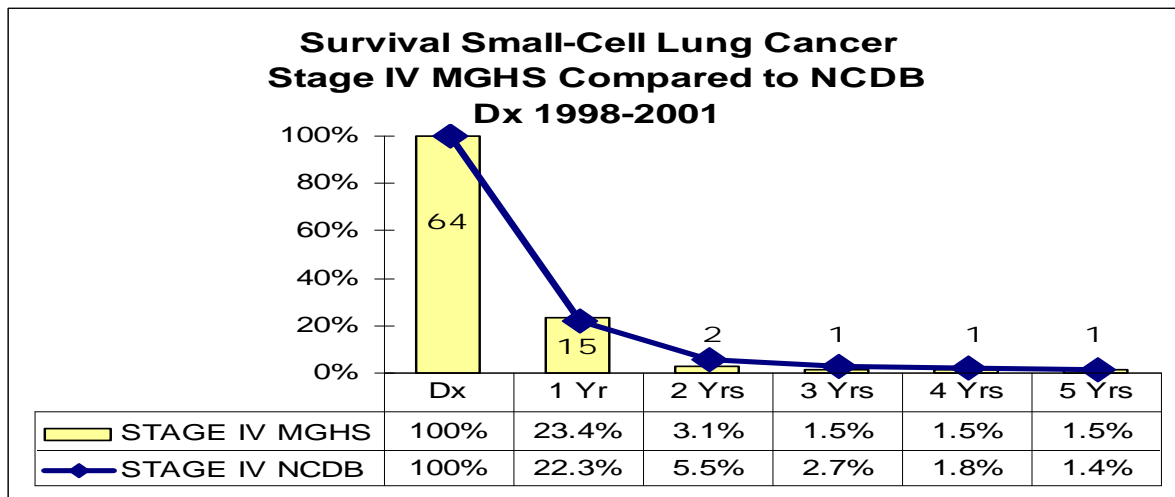


FIGURE 28



Hopefully, the treatment techniques and the development of screening and early detection algorithms will improve the statistics for lung cancer throughout the country including Marquette General Cancer Center the next time we examine this area of thoracic oncology. Similarly, the improvement in understanding of the basic mechanisms that cause cancer, development of new and more specific techniques for treatment, and especially the prevention of lung cancer, principally by smoking cessation and prevention, should give us a much brighter hope for the

future. In the meantime we can be assured that patients treated for their lung cancer at the Marquette General Cancer Center receive the state-of-the-art treatment with state-of-the-art results comparable to the national statistics. The Marquette General Cancer Center will continue to keep abreast of all the improvements in lung cancer care and with the improvement of state-of-the-art; the results of treatment should continue to improve at MGH and throughout the country.