Bronchogenic Carcinoma

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Lung cancer rates in the United States continue to rise and if present trends persist there will be nearly 300,000 new cases annually by the year 2000.[1] Added to the well established hazards of cigarette smoking[2] is burgeoning evidence of the harmful effects of many commonly encountered environmental agents and occupational risk factors.[3] Other predisposing elements have been observed and are under investigation—e.g., host determinants[4] and genetic propensities.[5-7]

Treatment advances are slow; survival rates remain low. At present, the most effective means of controlling lung cancer may be close surveillance of the high risk patient. The Mayo Lung Project of the Mayo Foundation[8] is conducting a controlled, prospective lung cancer screening program in males 45 years and older who smoke at least 20 cigarettes per day. The study group has a chest x-ray and pooled three-day sputum analysis every four months, and the controls are urged to have the same tests yearly. The Johns Hopkins University and the Memorial Sloan-Kettering Cancer Center are carrying out similar projects. Firm conclusions cannot be drawn yet, but compared to controls, considerably more of the lung cancers found in the study group are diagnosed early, while the prognosis is hopeful.

Histology and Embryology

Table 1 lists the World Health Organization classification of malignant pleuropulmonary neoplasms.[9] Embryologically, the tracheobronchial tree is a ventral entodermal foregut derivative, lined by at least five types of epithelial cells forming a pseudostratified mucosal sheath resting on a basement membrane. Three types are columnar cells and include mucus-secreting goblet cells, ciliated cells, and brush cells. The two basal types are small, multi-potential cells (probably reserve columnar cells) that lie parallel to the basement membrane and give the mucosa its pseudostratified appearance. In addition to being in reserve for mature columnar cells, they probably harbor the potential for metaplastic squamous epithelial cells, which form in response to chronic irritation and are not a normal finding. Another basal cell, identified by electron microscopy, is the Kulchitsky or K-type cell. It is postulated that the

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K-type cells may be neuroectodermal in origin, having endocrine or chemoreceptor functions and possessing the capability of secreting amine compounds such as serotonin, kinin activators, and others. The K cell may be the source of small cell carcinoma and may account for the wide spectrum of paraneoplastic syndromes associated with this type of bronchogenic carcinoma. Adenocarcinoma likely arises from the mucin-secreting cell.

Table 2 lists the prevalence of the five most common lung cancers in men and women, smokers and never-smokers, as they have presented at the Mayo Clinic. For reasons unknown, the incidence of adenocarcinoma is rising faster than that of the other types.

Epidermoid carcinoma, also called squamous cell epithelioma (SCE) or squamous cell carcinoma, is the most common bronchogenic carcinoma, and its pathogenesis is fairly well understood.[10] Fifty to 60 percent present as a proximal or hilar lesion (Table 3),[11] and it is uncommon for them to metastasize early. Epidermoid carcinomas tend to grow into the lumen of bronchi and obstruct, and so they often present first as an obstructive pneumonitis (Fig. 1). Endoscopically, they are often covered by a necrotic mass. The rest of the time they are more peripheral and 10 percent will cavitate. (Epidermoid carcinoma is the most common cavitating lung neoplasm.)

Epidermoid carcinomas are the easiest to diagnose by exfoliative sputum cytology, if for no other reason than the fact that they occur in proximal bronchi, and tend to grow into the lumen where the surface cells are easily sloughed.

Small cell carcinoma (also called oat cell carcinoma and anaplastic small cell carcinoma) is almost always inoperable, i.e., even though the lesion can be surgically resected without apparent involvement of hilar or mediastinal nodes, distant metastasis has usually already occurred. Seventy-five percent present as proximal lesions (Fig. 2) but in contrast to epidermoid carcinoma, the lumen is usually not filled [illegible]

I. Epidermoid carcinomas

II. Small cell anaplastic carcinomas

III. Adenocarcinomas
  1. Bronchogenic
    a. acinar
    b. papillary
    } with or without mucin formation
  2. Bronchioloalveolar

IV. Large cell carcinomas

V. Combined epidermoid and adenocarcinomas

VI. Carcinoid tumors

VII. Bronchial gland tumors
  1. Cylindromas
  2. Mucoepidermoid tumors

VIII. Papillary tumors of the surface epithelium

IX. "Mixed" tumors and carcinosarcomas

X. Sarcomas

XI. Unclassified

XII. Melanoma

spread tends to occur through the submucosa and compromises the lumen in this way. In spite of a large hilar mass, the endoscopic picture may consist merely of distortion of the bronchus with narrowing of the lumen, plus submucosal abnormalities. The mucosa itself may be normal.

Of the four commonest of lung cancers, small cell carcinoma is by far the one most often associated with the para-

Type	Total	Male		Female	
		Smoker	Never-smoker	Smoker	Never-smoker
Epidermoid	992	892	7	80	13
Small cell	640	533	4	100	3
Adenocarcinoma	760	492	39	128	101
Large cell	466	389	16	46	15
Bronchioloalveolar cell	68	35	4	13	16
TOTAL	2,926	2,341	70	367	148

Radiographic Findings	Squamous cell carcinoma (253 cases)			Adenocarcinoma (126 cases)			Large-cell carcinoma (97 cases)			Small-cell carcinoma (114 cases)		
	S	M	%	S	M	%	S	M	%	S	M	%
Hilar or perihilar mass or prominence	34	70	40	6	16	17	10	21	32	23	66	78
Parenchymal pulmonary lesion												
Small mass (<4.0 cm)	22	2	9	49	8	46	12	6	18	8	16	21
Large mass (>4.0 cm)	40	9	19	22	11	26	27	13	41	5	4	8
Apical mass	7	1	3	1	0	1	1	3	4	2	1	3
Multiple masses	1	0	0.5	3	0	2	1	1	2	1	0	1
Obstructive pneumonitis, collapse, or consolidation	59	80	53	7	25	25	2	30	33	5	38	38
Intrathoracic extrapulmonary involvement												
Mediastinal mass or widening	1	1	1	2	1	2	4	6	10	0	15	13
Chest wall lesion	0	0	0	0	0	0	0	0	0	0	0	0
Pleural effusion	1	8	3	1	5	5	0	2	2	0	6	5
Elevation of hemidiaphragm	1	2	1	0	5	4	0	1	1	0	4	4

S = single abnormality; M = multiple abnormalities

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Fig. 1 Squamous cell carcinoma of the right upper lobe bronchus causing a right hilar enlargement and partial collapse of the right upper lobe.

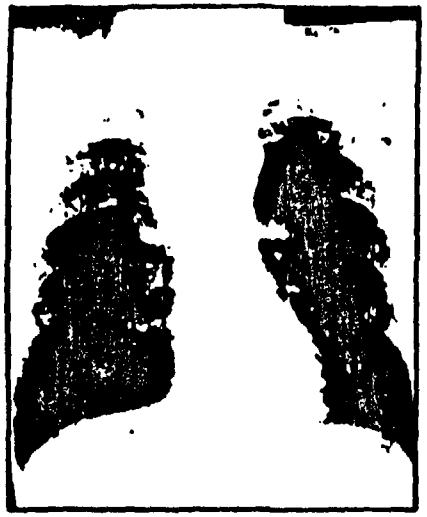


Fig. 2 Small cell carcinoma involving the proximal portion of the right lung. At bronchoscopy only minimal submucosal abnormalities were seen in the right main stem and right upper lobe bronchus.

neoplastic syndromes (Table 4), which can precede the chest roentgenographic appearance of the neoplasm by six to 12 months.[12,13] Yet the most common paraneoplastic syndrome — clubbing of the digits — does not, with very rare exception, occur with small cell cancer. (See also Ruckdeschel JC, et al: Small cell anaplastic carcinoma of the lung: changing concepts and emerging problems. Ca 29:84-95, 1979.)

Adenocarcinoma is peripherally located in 70 percent of cases, and may be quite large when discovered (Fig. 3). Because of peripheral location, these tumors are not likely to obstruct a bronchus, with resulting obstructive pneumonitis, or to directly invade the mediastinum. Ten percent present as a pleural effusion. Histologically, it may be very difficult to determine whether the adenocarcinoma in a pleural effusion is a primary or metastatic lesion. When the cancer is metastatic to the lung, the history, physical examination and routine laboratory studies will locate the primary tumor 80 percent of the time.

Large cell carcinoma (also called undifferentiated or anaplastic carcinoma) tends to be the "wastebasket" category for neoplasms that show no evidence of maturation or differentiation. Not uncommonly, the diagnosis of large cell carcinoma is made largely on the basis of exclusion of other cell types. The histology may show squamatoid features or patterns seen in poorly differentiated adenocarcinomas; however, in these cases a pathologist would label the tumor squamous cell carcinoma or adenocarcinoma, respectively. Any bronchogenic carcinoma may have features typical of any one or more of the four common types; exact designation to one particular class may be difficult in some situations. This is especially so when the pathologist has only cytology or a small piece of biopsy tissue. On chest x-ray a large cell

PARANEOPLASTIC SYNDROMES ASSOCIATED WITH BRONCHOGENIC CARCINOMA

Skeletal
Clubbing
Hypertrophic pulmonary osteoarthropathy
Endocrine
Cushing's syndrome
Hypercalcemia
Syndrome inappropriate antidiuretic hormone secretion (SIADH)
Gynecomastia
Carcinoid syndrome
Hypoglycemia
Hypercalcitonemia
Elevated human growth hormone
Elevated prolactin, follicle stimulating hormone, leutinizing hormone
Neurologic
Myasthenic syndrome
Peripheral neuropathy
Subacute cerebellar degeneration
Cortical degeneration
Polymyositis
Progressive multifocal leukoencephalopathy
Transverse myelitis
Cutaneous
Dermatomyositis
Acanthosis nigricans
Other
Cardiovascular
Thrombophlebitis
Marantic endocarditis
Hematologic
Anemia
Dysproteinemia
Disseminated intravascular coagulation
Granulocytosis
Eosinophilia
Hypoalbuminemia
Other: Plasmacytosis of marrow; leukoerythroblastosis; pure red cell aplasia; thrombocytopenia
Suppressed Immunity
Other
Nephrotic syndrome
Hyperuricemia
Hypersecretion of vasoactive intestinal polypeptide, with diarrhea
Hyperamylasemia
Anorexia-cachexia

carcinoma can present like epidermoid carcinoma, or small cell carcinoma as a hilar lesion or a peripheral lesion (Fig. 4).

Bronchioloalveolar cell carcinoma (also known as alveolar cell carcinoma and terminal bronchiolar carcinoma) is considered by some pathologists to be a variety of adenocarcinoma.

The fifth commonest bronchogenic carcinoma, this type is the least associated with tobacco carcinogens. It probably originates from the type II alveolar cell (surfactant producing cell). In contrast to the four types already discussed, which are almost always high grade (grades 3 or 4) neoplasms, the bronchioloalveolar cell carcinoma is usually a low grade 1 or 2 neoplasm. It has the greatest variety of presentations: a solitary pulmonary nodule; a localized infiltrate; a more diffuse but still localized infiltrate of segmental or lobar distribution; and finally, a diffuse disease involving all five lobes of the lungs. The latter may be associated with the production of copious amounts of thin mucoid secretions.

Signs and Symptoms of Bronchogenic Carcinoma

Local Effects of the Primary Tumor

About 25 percent of patients with bronchogenic carcinoma are asymptomatic at the time of diagnosis. The presence of any symptoms worsens the prognosis, since the first symptom is often from a metastasis.

Localized symptoms of a bronchogenic carcinoma are cough or change in a smoker's cough, dyspnea, hemoptysis, signs of obstructive pneumonitis, or a stridor or wheeze. Any of these conditions should alert the clinician to the possibility of lung cancer, especially in a smoker or a former smoker. Any new abnormality on the chest x-ray warrants further investigation, possibly including an eventual thoracotomy.

Fiberbronchoscopy has revolutionized our approach to the patient with possible lung cancer. It more than doubles the range of visibility over that of rigid bronchoscopy in the tracheobronchial tree, where a neoplasm may not be evident on x-ray. With the use of fluoroscopy, it also allows us to obtain material, with a brush or small biopsy forceps, from more peripheral lesions. Its use should be considered in every individual with the aforementioned symptoms. Persistent hemoptysis, regardless of a negative fiberbronchoscopy, requires close followup to be certain an occult carcinoma was not overlooked. Though the most common cause of hemoptysis in a smoker is bronchitis, the risk of bronchogenic carcinoma is high enough to warrant aggressive monitoring.

Effects of Spread to Adjacent Thoracic Structures

The first symptoms of a bronchogenic carcinoma may be related to invasion of adjacent structures, which almost always implies unresectability. Pain in the chest can come from the tumor impinging on nerves or from invasion of the chest wall or mediastinum.

Left vocal cord paralysis from involvement of the left recurrent laryngeal nerve is not an uncommon event when a bronchogenic carcinoma involves the left hilus or mediastinal lymph nodes.

A pleural effusion may hide a bronchogenic carcinoma, and this should be considered whenever there is an unexplained pleural effusion in an adult. There are at least six mechanisms by which bronchogenic carcinoma can be associated with a pleural effusion. Direct involvement of the pleura by tumor cells, obstruction of mediastinal lymphatics, and thoracic duct obstruction with resultant chylous effusion definitely portend unresectability; obstruction of pulmonary lymphatics with poor resorption of pleural fluid, obstructive atelectasis with pneumonitis, and pulmonary embolus with pleural effusion generally do not indicate unresectability.

Direct involvement of the brachial plexus by a bronchogenic carcinoma produces the Pancoast syndrome, also known as superior sulcus tumor of the lung [illegible]

Fig. 3 Three centimeter peripherally located adenocarcinoma.

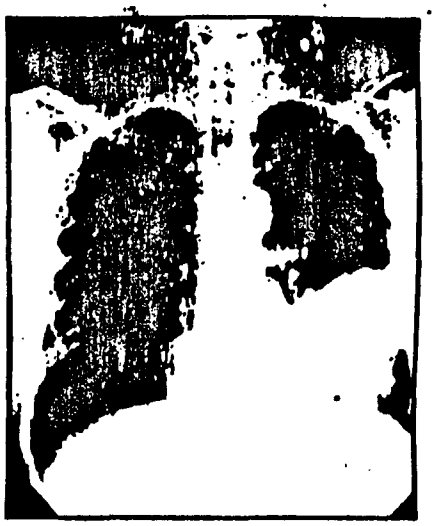


Fig. 4. Large cell carcinoma of the lung. It is over 4 cm in diameter so it is called a mass lesion. This patient was asymptomatic.

tumor. It is invariably associated with pain, which may precede the roentgenographic abnormality by many months (Fig. 5). Over 80 percent of these patients are smokers, but small cell carcinoma very rarely occurs in this area.

Invasion of the mediastinum may cause obstruction of the superior vena cava or the esophagus. This occurs in fewer than two percent of patients with bronchogenic carcinoma.

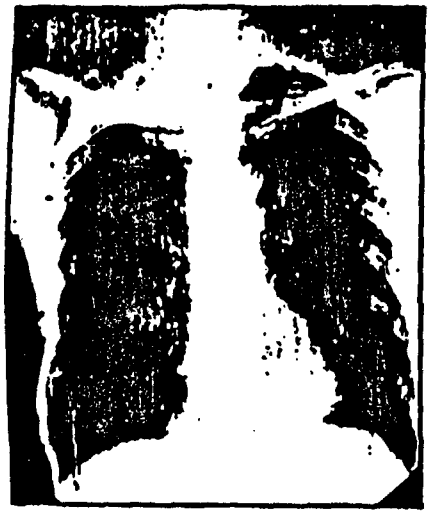
Manifestations of Distant Metastasis

The most common sites of metastasis of bronchogenic carcinoma are the lymph nodes, liver, brain, bone, adrenals, and lung. The incidence varies considerably from one report to the next, and depends to a great extent on the tumor cell type. It can be assumed that most small cell carcinomas have metastasized by the time of diagnosis. There isn't a good answer at this time as to how far to go to look for these metastases before con-

Systemic Symptoms Unrelated to Tumor Location

Non-specific symptoms—unrelated to obvious metastasis—of anorexia, fatigue, malaise, and fever are worrisome. They may be hormone- or peptide-mediated, like those of the paraneoplastic syndromes, but we know very little about them. A good deal more is known about the paraneoplastic syndromes, particularly the endocrine ones (Table 4).[12,13] It is estimated that 10 to 20 percent of patients with bronchogenic carcinoma

sidering mediastinoscopy/thoracotomy. Certainly a thorough history and physical examination are mandatory, and liver and bone enzyme studies may be of help, though the latter can be deceptively negative. At present, isotope scanning of liver, bone, and brain in the asymptomatic patient with bronchogenic carcinoma are not of sufficient value to be used routinely.



ig. 5 Epidermoid carcinoma in the thoacic outlet, opacifying the right apex. Pain n the upper extremity or shoulder is present n most of these patients.

ave one or more of these syndromes, nd if suppressed immunity is considered s a paraneoplastic syndrome, then the ncidence is considerably higher.

There have been numerous studies of ie immunologic characteristics of paents with lung cancer,[14] showing that any of these patients have various imunodeficiencies. The percentage of deiencies increases from stage I to stage and the prognosis is worse in patients ith the deficiency. However, there is proof that the immunodeficiency conbutes to the initiation of the developent of the cancer. In fact, it seems ely that the immunodeficiency may be e of the cancer's effects on the host.

aging

aging a lung cancer aids in the selection optimal treatment and the estimation the prognosis, enhances communican about a specific patient's cancer, and allows clearer reporting on groups of patients. A lung cancer may be staged a number of times during the course of the disease:

- A *Clinical-Diagnostic Stage* should be recorded after a thorough diagnostic examination that may include a medical history, physical examination, routine and special roentgenograms, scans of various organs, endoscopic examinations such as bronchoscopy, esophagoscopy, mediastinoscopy, and thoracoscopy, and other procedures such as mediastinotomy and lymph node biopsies. Exploratory thoracotomy is not included.
- All the data for the Clinical-Diagnostic Stage, plus the surgeon's findings at an exploratory thoracotomy, may be used to establish a *Surgical-Evaluative Stage.*
- A *Post Surgical Treatment-Pathologic Stage*, based on all the above data plus the pathologist's report, follows examination of the resected specimen.
- Post-treatment followup examinations may reveal recurrence of the cancer, at which time a *Retreatment Stage* should be recorded.
- Finally, in the event of death and postmortem examination of a lung cancer patient, an *Autopsy Stage* may be recorded.

The stage of a lung cancer may be described in a narrative statement or by a formal staging system. The American Joint Committee for Cancer Staging and End Results Reporting has published such a staging system for lung cancer[15] and it or similar systems are now used all over the world. This system uses the TNM nomenclature (including appropriate subscripts) in which the letter T describes the extent of the primary tumor, the letter N describes the absence or presence and extent of metastases to regional lymph nodes and the letter M describes the absence or presence of more distant metastases. Certain TNM groups with comparable treatment and prognosis may be combined into specific stages (Table 5). The relationship be-

TABLE 5
STAGING OF LUNG CANCER

TNM Classification	Definition
Primary Tumor (T)	
TX	Tumor proven by the presence of malignant cells in bronchopulmonary secretions but not visualized roentgenographically or bronchoscopically; or any tumor that cannot be assessed
T0	No evidence of primary tumor
TIS	Carcinoma in situ
T1	Tumor 3.0 cm or less in greatest diameter, surrounded by lung or visceral pleura, and without evidence of invasion proximal to a lobar bronchus at bronchoscopy
T2	Tumor more than 3.0 cm in greatest diameter, or a tumor of any size that either invades the visceral pleura or has associated atelectasis or obstructive pneumonitis extending to the hilar region. At bronchoscopy, the proximal extent of demonstrable tumor must be within a lobar bronchus or at least 2.0 cm distal to the carina. Any associated atelectasis or obstructive pneumonitis must involve less than an entire lung and there must be no pleural effusion.
T3	Tumor of any size with direct extension into an adjacent structure such as the parietal pleura or the chest wall, the diaphragm, or the mediastinum and its contents; or a tumor demonstrated bronchoscopically to involve a main bronchus less than 2.0 cm distal to the carina; or any tumor associated with atelectasis or obstructive pneumonitis of an entire lung or pleural effusion
Nodal Involvement (N)	
N0	No demonstrable metastasis to regional lymph nodes
N1	Metastasis to lymph nodes in the peribronchial or the ipsilateral hilar region, or both, including direct extension
N2	Metastasis to lymph nodes in the mediastinum
Distant Metastasis (M)	
MX	Not assessed
M0	No (known) distant metastasis
M1	Distant metastasis present

Stage Grouping

Occult Carcinoma	Stage 1	Stage 2	Stage 3
TX N0 M0	T1S N0 M0	T2 N1 M0	T3; any N or M
	T1 N0 M0		N2; any T or M
	T1 N1 M0		M1; any T or N
	T2 N0 M0		

Cell Type	Clinical-Diagnostic Stage 1	2	3
Squamous	46.6%	39.8%	11.6%
Adenocarcinoma	45.9%	14.3%	7.9%
Large	42.8%	12.9%	12.9%
Small	6.0%	5.0%	3.8%

Adapted from the American Joint Committee for Cancer Staging and End Results Reporting booklet "Clinical Staging System for Carcinoma of the Lung."

veen the Clinical-Diagnostic Stage and rognosis for each of the major cell pes is shown in Table 6. The Post Surcal Treatment-Pathologic Stage is a ore accurate estimation of the extent lung cancer than are Clinical-Diagstic Staging or Surgical-Evaluative aging, as the latter two tend to underimate the extent of the T and N comtments.[16] Therefore, the Post Surgical eatment-Pathologic Stage should be ed in reporting surgically treated cases. l staging systems, however, tend to derestimate the extent of the M comtment, as we have no reliable clinical thods of detecting the microscopic tastases that are so common in lung ncer.

atment

ng cancer treatment varies, depend- on the cell type, the stage of the can- , and multiple host factors such as general condition, and the presence concomitant diseases, particularly onic obstructive lung disease with airment of respiratory function and coronary heart disease. Current modalities, used in various combinations, are surgical-resection, radiation therapy, chemotherapy and immunotherapy. Despite their aggressive application, the five-year survival of large groups of patients with lung cancer remains in the range of 10 to 15 percent. Obviously, improvement is needed in the management of this disease.

Most authorities agree that surgical resection is the proper treatment of lung cancer whenever there is a reasonable chance of removing all the cancer with an acceptable risk of morbidity and mortality.[16-18] Thus Stages 1 and 2 non-small cell lung cancers are usually resected, and for bronchioloalveolar cell carcinoma, there is no effective non-surgical treatment. (The therapy of small cell lung cancer is a special problem and will be discussed later.[19,20]) Some Stage 3 non-small cell lung cancers are resected if there is a reasonable probability that all of the cancer can be removed, e.g., peripheral carcinomas with direct invasion of the chest wall without metastases to lymph nodes or T1 or T2 lesions with metastases to subcarinal or distal ipsilateral paratracheal lymph nodes. The recurrence rate in such cases is high and postoperative radiation therapy[21] or chemotherapy or a combination of both may be used in an effort to improve the treatment results of these patients. Similar postoperative adjuvant therapy is also being evaluated in Stage 2 cases. Postoperative immunotherapy using BCG vaccine intrapleurally has been reported to be beneficial in Stage 1 cases.[22] An international cooperative study is now underway to determine the value of BCG vaccine used in this way.

Over one-half of all bronchogenic carcinomas are found to be inoperable at the time of initial workup, including mediastinoscopy; only 35 to 40 percent of patients have resectable lesions. This group has a 25 to 35 percent five-year survival, and the overall five-year survival is only 10 to 15 percent; thus, for the unresectable group, the five-year survival is very small. Palliative resections are seldom advisable, as the symptoms due to the cancer usually can be alleviated by other treatment, with less risk of mortality.

Definitive radiotherapy is frequently recommended for Stages 1 and 2 patients for whom surgery is contraindicated. It is also widely used for patients with Stage 3 lesions if the disease is limited to one hemithorax and the ipsilateral supraclavicular lymph nodes. Such therapy may completely destroy the cancer in the field of treatment in about 35 percent of cases.[23] However, it frequently fails to cure these patients because of occult distant metastases that are out of the field of therapy. Radiation pneumonitis and permanently impaired lung function commonly complicates such therapy, so it may be an unacceptable alternative to surgery for patients with poor respiratory function.

The frequent failure of radiotherapy due to persistent thoracic cancer or to occult distant metastases has induced numerous studies of adjuvant chemotherapy using single drugs or combinations of drugs. Some of these studies are promising but none has yet proved effective enough to be recommended as routine therapy.[24] The same may be said for various types of immunotherapy given along with radiotherapy.

Palliative radiotherapy usually succeeds when given for the relief of hemoptysis, superior vena caval obstruction, bone pain due to osseous metastases, and various symptoms due to metastases to the brain.

Administering anti-cancer drugs along with radiotherapy is supported by evidence that the drugs suppress the growth of cancer cells and cause objective regression of measurable lesions in a significant percentage of cases. Because no single drug has been outstandingly effective, many combinations are being tested and some have produced regression of the disease in about 50 percent of cases of non-small cell lung cancer. However, to date there is little evidence of any improvement in either median survival rates or the quality of life of these patients.

Small Cell Lung Cancer

The treatment of small cell lung cancer has become more effective in recent years.[19,20] Multiple drug therapy produces objective regression of the disease in almost all of these patients, with complete regression of demonstrable lesions in more than one-half of them. When such therapy is combined with radiotherapy to the thorax in cases wherein disease seems confined to the involved hemithorax and ipsilateral supraclavicular space, the median survival time approaches two years. When distant metastases are demonstrable at the time of diagnosis, the response rates to multiple drug chemotherapy are less impressive, but even in these patients the median survival time may approach one year, compared to about three months when treatment consists of a placebo or of a single anti-cancer drug.

Combination chemotherapy does not control CNS metastases, an unfortunate fact since they develop in one-third to one-half of all patients with small cell

cancer. Many advocate prophylactic radiotherapy to the head, but the efficacy of this approach has not been proved.

Prognosis and Followup

Survival rates of one group of patients classified by cell type and Clinical-Diagnostic Stage are shown in Table 6. Even better survival rates have been reported more recently, especially for non-small cell cancer patients with a Post Surgical Treatment-Pathologic Stage I classification.[23]

In addition to cell type and stage, other factors that influence prognosis are performance index, nature and duration of symptoms, concomitant disease and the patient's age.

Doubling time (DT), which is the rate of growth of a tumor, is of relative prognostic value when it is known. The DT can vary from as little as 10 days to over two years. Generally, the shorter the doubling time, the worse the prognosis. However, it seems that the longer the DT the less responsive is the neoplasm to chemotherapy.

Followup examinations should be performed at least three or four times a year for all patients who have completed treatment for lung cancer. Local recurrence, distant metastases, or second primary cancers are not uncommon, especially in patients who continue to smoke. Although complete cure is usually out of the question when cancer is detected on followup, much can be done to suppress the growth of the lesions, to delay the development of debilitating symptoms, to prevent complications and, sometimes, to prolong the lives of these patients.

In the course of followup, numerous questions and psychosocial needs arise on the part of the patient and his or her family and friends. The physician must meet these needs, time-consuming as this may be. For the interested doctor, several good texts on lung cancer are available;[26-28] we have found *Living with Lung Cancer: a reference book for people with lung cancer and their families*[29] to be valuable in our effort to educate patients and their loved ones and to ease the difficulties they encounter.

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BEYOND STATISTICS

Many of the most difficult medical decisions depend mainly on character, the slowly matured power of judgment, and a grasp of fundamental principle, and detailed knowledge and technical skill are not as helpful as we sometimes would like to think. In making such decisions, statistics are useful but secondary. They save us from having ignorance foisted on us. But they are relegated to a position below empathy and compassion aided and abetted by experience. In trying situations one assembles all the help he can muster.

From: Airing CD: Beyond Statistics. JAMA 241:2193-2194, 1979.

TABLE 2
OCCURRENCE OF FIVE MAJOR TYPES OF LUNG CANCERS IN SMOKERS AND NONSMOKERS

Type	Total	Male		Female	
		Smoker	Never-smoker	Smoker	Never-smoker
Epidermoid	992	892	7	80	13
Small cell	640	533	4	100	3
Adenocarcinoma	780	492	39	128	101
Large cell	486	389	16	46	15
Bronchioloalveolar cell	68	35	4	13	16
TOTAL	2,926	2,341	70	367	148

TABLE 3
ABNORMAL RADIOGRAPHIC PATTERNS IN 600 CASES OF CARCINOMA OF THE LUNG AS RELATED TO HISTOLOGICAL CELL TYPE

Radiographic Findings	Squamous cell carcinoma (263 cases)			Adeno-carcinoma (126 cases)			Large-cell carcinoma (97 cases)			Small-cell carcinoma (114 cases)		
	S	M	%	S	M	%	S	M	%	S	M	%
Hilar or perihilar mass or prominence	34	70	40	8	16	17	10	21	32	23	66	78
Parenchymal pulmonary lesion												
Small mass (<4.0 cm)	22	2	9	49	8	45	12	5	18	6	16	21
Large mass (>4.0 cm)	40	8	19	22	11	28	27	13	41	8	4	8
Apical mass	7	1	3	1	0	1	1	3	4	2	1	3
Multiple masses	1	0	0.5	3	0	2	1	1	2	1	0	1
Obstructive pneumonitis, collapse, or consolidation	59	80	53	7	25	26	2	30	33	5	38	38
Intrathoracic extrapulmonary involvement												
Mediastinal mass or widening	1	1	1	2	1	2	4	6	10	0	15	13
Chest wall lesion	0	0	0	0	0	0	0	0	0	0	0	0
Pleural effusion	1	8	3	1	5	5	0	2	2	0	6	5
Elevation of hemidiaphragm	1	2	1	0	5	4	0	1	1	0	4	4

S = single abnormality; M = multiple abnormalities

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