unimportant because pulmonary function studies do not show evidence of obstructive lung disease is to ignore the patient's complaints. And if such symptoms fail to respond to medical therapy for reflux, an anti-reflux operation may be indicated even in the absence of esophagitis. The failure to demonstrate a significant association between obstructive lung disease and gastroesophageal reflux in no way justifies the conclusion that respiratory symptoms and reflux are not related. The respiratory complications of esophageal disease remain poorly understood and recognized by many physicians, and it seems inappropriate for physicians whose primary interest is chest disease to minimize this relationship even further.

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Sequential Staging of Bronchogenic Carcinoma

The paper by Mintz and his colleagues in this issue of Chest (see page 653) is an important contribution to the literature on staging of bronchogenic carcinoma. Their most striking finding is the effective use of the gallium scan in the first phase of staging beyond the initial assessment (history, physical examination, chest X-ray film and routine blood tests). In 29 patients who were advanced in stage to III M1 as a result of this "clinical staging," 72 percent had metastatic disease detected by gallium scan, and 10 percent had metastatic disease detected only by gallium scan; it was missed by bone, liver and brain scans. These were, however, metastases to the kidney or the adrenal glands, which could have been detected by routine intravenous pyelography. In terms of cost benefit, the authors' findings make an excellent case for the following algorithm in the staging of patients with lung cancer:

Initial Staging: History, physical exam, CBC and chemistries
  negative for metastases
  positive for metastases

Clinical Staging:
  Gallium scan
  negative for metastases
  liver, bone and brain scans

Final Staging:
  negative for metastases
  invasive procedure
  (mediastinoscopy and/or thoracotomy)

Also of considerable interest is the fact that uptake was demonstrated in the primary tumor on gallium scan in 96 percent of patients, irrespective of histology. The authors do not comment on the value of the gallium scan when assessing the presence or absence of nodal involvement in the hilum, in the mediastinum, or in the supraclavicular areas. They do point out, however, that five patients were lowered in clinical stage by the final (presumably invasive) procedure: four from clinical stage II to stage I, and one from clinical stage III M1 to stage II. These apparently represent "false positives" for the gallium scan and/or the chest X-ray examination.

Several precautions should be noted in the clinical application of the staging sequence herein suggested. First, although initial staging (IS) was highly specific for stage III M1 disease, this was because "our criteria for IS III M1 were very strict." For instance, an elevated serum level of alkaline phosphatase alone was not sufficient (it would be of interest to know how many patients with an elevated level of alkaline phosphatase did not have evidence of extensive disease). Second, the gallium scan may be "false positive" in areas of inflammatory disease, abscess, or because of retained radioisotope in the gut.

Also, other investigators have not obtained such encouraging results with the gallium scan as a substitute for other scanning procedures, at least for small (oat)–cell carcinoma of the lung. Finally, it may be seriously questioned whether routine liver and brain scans are justified in patients with negative initial staging and a negative gallium scan. Of 50 patients with metastatic disease in the present series, only eight were so staged by these procedures, and the liver scan alone was positive in only one. Other authors have reported a lower yield with brain scans in neurologically unremarkable patients (possibly because CT scanning was not used), and little additional yield from liver or...
brain scans in patients with normal findings on blood chemistry and physical examination.

The survival data in this series bear emphasis for several reasons. It is surprising that patients with final clinical stage II disease have survival at 124 weeks (about 29 months) almost as good as those with final stage I disease (90 vs 83 percent). This may be explained by the small numbers involved, or by the additional radiation and chemotherapy which patients in stage 2 received. The median survival of 45 weeks for patients in clinical stage III M<sub>0</sub> is about what one would expect with surgery alone in resectable stage III patients,<sup>4</sup> or what may be achieved with radiation therapy alone in patients who are considered candidates for "definitive" treatment using this modality.<sup>6</sup><sup>–</sup><sup>7</sup> The median survival of 17 weeks for patients in clinical stage III M<sub>1</sub> and with extensive disease is sobering, especially in an institution where a median survival of 35 weeks has been reported for patients in this category receiving combination chemotherapy (CAMP).<sup>6</sup><sup>–</sup><sup>7</sup> This apparent discrepancy, as the authors correctly point out, is due to the criteria for selection applied to patients receiving chemotherapy: only a third of the patients with extensive disease in this series met the conditions for a trial of CAMP.

What the criteria for selection are (or should be) for patients with extensive, non-small cell bronchogenic carcinoma to receive chemotherapy is a fit topic for further consideration and discussion among clinicians.

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REFERENCES


Procainamide and the Sinus Node

Procainamide is most frequently used for the control of ventricular dysrhythmia. Recently, this agent has been shown to be effective against some paroxysmal supraventricular tachycardias utilizing dual A-V nodal pathways (A-V nodal re-entry) or anomalous pathways. Procainamide may be associated with cardiac or extracardiac toxicity, namely: Q-T prolongation with and without ventricular fibrillation, negative inotropy, hypotension, fever, rash, and a lupuslike syndrome. In this issue of Chest (see page 699), Kim and Friedman describe the occurrence of severe sinus node dysfunction following low dose (1.5-2 gm/daily) oral procainamide therapy in two patients with chronic renal failure. They support their conclusions by noting the disappearance of severe sinus brady-cardia and sinus pauses following discontinuance of procainamide therapy in their patients.

Although the observation of sinus nodal dysfunction with procainamide therapy has some clinical implications, the present paper sheds little light on the mechanism or mechanisms involved in this unexpected response. Procainamide possesses sympathomimetic and vagolytic properties and has been reported to enhance sinus nodal activity rather than depressing it.<sup>8</sup> In a recent study on the electrophysiologic effects of procainamide, Wyse and co-workers reported a significant enhancement of sinus nodal automaticity in 16 patients following acute intravenous administration of this agent. Procainamide was administered at two different dose regimens: a) 8 mg/kg as bolus and 0.05 mg/kg per min infusion, and b) 12 mg/kg bolus and 0.075 mg/kg/min as infusion. Sinus cycle length decreased significantly from 848 ± 43 msec to 799 ± 42 msec at plasma procainamide level ranging from 6 to 7.4 mg/ml. Increase in sinus nodal automaticity was also suggested by significant decrease in sinus node recovery time. Procainamide also facilitated perinodal conduction as suggested by a significant shortening of sinoatrial conduction time from 105 ± 11 to 90 ± 9 msec. At a higher dose (plasma levels of 10-11 mg/ml) the effects on sinoatrial function were similar, but of greater magnitude.

In view of the above findings by Wyse and co-workers,<sup>4</sup> if we analyze the data presented by Kim and Friedman, it appears that in certain circum-

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