equated with the presence of brain metastases, at least in the case of solitary brain lesions. In a previous study 11% of such lesions were histologically proven to represent various non-metastatic processes.2

Mitchell L. Margolis, MD, FCCP, Pulmonary and Critical Care, Philadelphia VA Medical Center, Philadelphia

REFERENCES

To the Editor:

We have read with interest the letter by Dr. Margolis who commented on our article (Chest 1994; 106:1025-29). We agree that patients with non-small cell lung cancer, with only non-organ specific clinical signs, have increased risk of metastatic disease, and therefore, of brain metastasis. In our experience, 13 of the 16 patients with neurologically asymptomatic brain metastases had non-organ specific clinical signs (weight loss, 9 patients; bone pain, 2; increased transaminases, 2, and abnormal level of alkaline phosphatase, 2). Ten of them were eventually shown to have metastases in other organs (skeleton, 4 patients; suprarenal glands, 2; chest and suprarenal glands, 1; liver, 1; liver and suprarenal glands, 1, and lung, 1). The only three patients who showed neither symptoms and signs of neurologic involvement nor non-organs specific signs, however, were precisely those patients who had an unnecessary intervention because of a brain computed tomography (CT) (see Table 6, CHEST 1994; 106:1025-29). A policy of checking for brain metastasis only in subjects with non-organ specific findings would have failed in 3 of the 31 otherwise resectable cancers (10%). On this basis, we still recommend obtaining a brain CT in all surgical candidates for lung cancer resection, even when they are totally asymptomatic.

We thank Dr. Margolis for having pointed out that a positive brain CT is not synonymous with metastasis. This possibility was taken into consideration in our study, where the reference criteria for brain metastases were a positive CT scan and neurologic symptoms appearing within 6 months from the initial diagnostic evaluation.

Domenico Ferrigno, MD, and Gianfranco Buccheri, MD, FCCP, 2nd Pulmonary Division, A Carle Hospital of Chest Diseases, Cuneo, Italy

Redundant Aryepiglottic Folds May Require Surgical Removal

To the Editor:

I read with interest the article, “Abnormal Movement of the Arytenoid Region During Exercise Presenting as Exercise-induced Asthma in an Adolescent Athlete,” by Bittleman and colleagues (CHEST 1994; 106:615-16), which appeared in the August issue of CHEST. I believe the report describes a patient with redundant aryepiglottic folds as depicted in the illustration of the fiberoptic image. It appears to be a typical example of redundant folds that are flabby, edematous excrences, histologically showing edematous mucosa overlying fibrocollagenous stroma (C.T.R., unpublished report, 1958). Such folds require surgical removal.

In the early part of my practice of thoracic surgery in Phoenix, I performed operations on some 25 patients with severe inspiratory stridor due to redundant aryepiglottic folds. With the patient under general anesthesia the operation was carried out with a biopsy forcep through a laryngoscope. The ages of the patients ranged from 8 days to 8 years. Most were less than 2 years old. All were successfully relieved of the stridor and severe respiratory difficulty without any complications with resection of the folds.

At laryngoscopy, if the blade of the scope was positioned in the customary manner behind the epiglottis, the redundant aryepiglottic folds were stretched and thinned to a more flattened appearance. However, with the blade positioned anterior to the epiglottis, the redundant folds were seen to fall dramatically into the glottic chink blocking the airway. After resecting the redundant folds a normal airway becomes visible with the blade either anterior or posterior to the epiglottis.

Resection of the redundant aryepiglottic folds was reported by Hasslinger in 19281 as quoted in a thesis, “Congenital Laryngeal Stridor,” by Kelemen.2 He gives a detailed erudite discussion of laryngeal stridor, which includes redundant aryepiglottic folds as one of the causes. This prompted me to search for such findings as early as 1955.

Before the proper diagnoses were made, several patients were referred to competent well-qualified specialists at recognized centers wherein a number of examinations including angiograms were performed on the patients to no avail, although specious diagnoses including asthma were offered (written communication, Sinai Hospital of Baltimore, Oct 1956). Nagai et al3 attributed laryngeal stridor to abnormal motion of the arytenoid region with no abnormalities of the pharynx, vocal cords, or trachea and no laryngeal spasm, although a photograph made at bronchoscopy appears to reveal redundant aryepiglottic folds. It was concluded in the report by Nagai et al3 that the cause was emotional stress, and treatment with sedation was recommended. Functional airway obstruction with stridor was reported by Appleblatt and Baker4 wherein no abnormal physical findings were apparent. Their management of three patients included tracheostomy and psychiatric evaluation.4

A prominent cause of inspiratory stridor especially in the pediatric group can be overlooked during endoscopic examination by inserting the blade of the laryngoscope behind the epiglottis. The true cause of the stridor, however, may well be elicited when the blade is positioned anteriorly as described.

C. Thomas Read, MD, FCCP, Sun City, Arizona

Reprint requests: Dr. Read, 9325 Briarwood Circle, Sun City, AZ 85351

REFERENCES
2 Kelemen G. Congenital laryngeal stridor. Arch Otolaryngol 1953; 58:245-68

Communications to the Editor