views in our study. Our pooled analysis failed to show the efficacy of consolidation chemotherapy (CCT) in terms of survival prolongation for patients with locally advanced non-small-cell cancer (LA-NSCLC). This negative result should carefully be assessed because, as Dr. Jermic suggested, our study has several limitations which might affect our study result. In particular, as our study was performed on a publication basis, we could not assess the heterogeneity at the individual patient level. It is important to identify the characteristics of patients who could benefit from CCT. However, we could not carry out subgroup analyses based on the patterns of treatment failure or responses to initial chemo-radiotherapies as our analysis did not use individual data of each trial.

Another important factor that may affect our study result is the diversity of CCT regimens among trials. We evaluated the effectiveness of CCT by dividing it into two patterns: continuous CCT, which continues chemotherapy with agents given in the induction therapy, and switch CCT (SCCT), which switches chemotherapy to different agent(s) in the consolidation phase. SCCT might be more promising than continuous CCT because it is expected to effectively eradicate tumor cells resistant to the induction chemo-radiotherapy. Although our analyses failed to show the efficacy of SCCT, it was probably because of the small number of trials: only four trials were designed for SCCT. Further clinical trials on SCCT will be warranted to answer these queries.

Finally, our pooled analysis failed to provide evidence that CCT yields significant survival benefit for LA-NSCLC patients. However, we believe that the findings of this study are relevant because it reminds us that there is currently no sufficient evidence to support CCT for LA-NSCLC patients, and that current recommended treatment for LA-NSCLC patients remains concurrent chemo-radiotherapy. Little progress in treatment strategies for LA-NSCLC patients has been observed in the last 20 years, and it is urgent to seek new treatment options/strategies to improve this. Further studies, for example, individual patient-based meta-analyses or prospective studies focusing on patterns of treatment failure or responses to initial chemo-radiotherapy are needed to establish how to use CCT appropriately to improve survival of LA-NSCLC patients.

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Lung Adenocarcinoma with Ipsilateral Pleural and Breast Metastases

To the Editor:

We congratulate Huang et al.1 for their elegant description on how lung adenocarcinoma cells may metastasize to the ipsilateral parietal pleura, invade the chest wall lymphatic vessels which drain to the ipsilateral axillary lymph nodes, retrogradely spread to the intramammary lymphatics, and finally establish ipsilateral breast metastasis. It is plausible that the presence of the clinical triad of ipsilateral pleural effusion or thickness, enlarged ipsilateral axillary lymph nodes that are palpable or evident on computed tomography (CT), and ipsilateral breast metastasis provides support for this proposed mechanism for ipsilateral breast metastasis from lung cancer. In addition, the presence of intact fat planes between the chest wall and breast tissue on CT scan excludes direct tumor invasion of the breast from the ipsilateral parietal pleural metastasis as a less likely mechanism.1

We also like to add that the absence of enlarged mediastinal (N2 or N3 disease) and ipsilateral supraclavicular (N3 disease) lymph nodes on CT scan and the latter also on palpation is needed to discount the other possible mechanism of lymphatic spread to ipsilateral axillary lymph nodes from mediastinal lymph nodes, through intercostal lymphatics2 or retrogradely through supraclavicular nodes.3

Whatever the mechanism of spread to the axillary lymph nodes, breast metastasis is an infrequent manifestation of advanced disseminated lung cancer and is associated with an extremely poor prognosis and a short survival.4,5

As Huang et al.1 has stated, differentiating primary from metastatic breast carcinoma is of great clinical importance.

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because of the different clinical implication and management options. Metastatic breast lesions, which are very rare compared with primary breast carcinoma, tend to be found in the subcutaneous fat, whereas primary breast cancers develop in the glandular tissue. As opposed to primary breast cancers, metastatic tumors to the breast are characteristically superficially located, poorly defined, irregular nodules or masses without calcification on mammography and ultrasound examination.5

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