drome who have micrognathia, growth hormone can help the mandible catch up to the maxilla, whereas in girls who do not have micrognathia, increased mandibular growth can lead to an underbite, mainly due to vertical growth of the ramus of the mandible. This is quite different from acromegaly, of course, in which the entire mandible enlarges and protrudes.

I agree with Dr. Taback and colleagues that we really do not know yet how best to introduce estrogen therapy in short patients with Turner's syndrome. Estrogen should probably be introduced only when patients are near their final height. In a study that combined very low doses of estrogen (25 ng per kilogram of body weight per day)[3] with growth hormone, there was a slight additive effect on growth velocity but also an accelerated increase in bone-age maturation in girls with bone ages of less than 11 years and chronological ages between 13 and 14 years. Hence, I recommend 14 years as a rational age for starting estrogen supplementation. The low doses Taback et al. recommend have not yet been tested in Turner's syndrome. Despite the variable height outcomes after growth hormone therapy in patients with Turner's syndrome, a Food and Drug Administration (FDA) advisory committee recommended this therapy nearly unanimously at its meeting on December 10, 1996. The FDA has since granted marketing clearance, as have authorities in 27 other countries.

Although the issue of adequate bone mineral density is difficult to assess in Turner's syndrome,[4] because of a frequent intrinsic "osteopenic" appearance of the bone, we should take heart from the report by Neely et al., which demonstrates that adolescents with Turner's syndrome who receive growth hormone are not osteopenic and have normal bone mineral density. The report concludes that early estrogen replacement cannot be justified on the basis of bone mineral status.[5]

As Gargan and Peerzada point out, we need a national data base. The U.S. Turner Syndrome Society (1313 S.E. 5th Street, Minneapolis, MN 55414; fax: [612] 379-3619) is developing one and welcomes data submissions.

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## Management of Venous Thromboembolism

*To the Editor:* In his review of the management of venous thromboembolism (Dec. 12 issue),[1] Ginsberg failed to mention the role of echocardiography in the diagnosis of this disorder. Despite the importance of ventilation–perfusion lung scanning, it has its limitations. It usually necessitates the transfer of the patient, who may be acutely dyspneic, to the radiology department and occasionally to a different hospital. On the other hand, echocardiography, whether transthoracic or transesophageal, is widely available, carries a minimal risk or none, and can help in making the diagnosis at the bedside. In the absence of chronic pulmonary disease, echocardiographic features that suggest pulmonary embolism include a dilated hypokinetic right ventricle and the presence of tricuspid regurgitation with increased velocity, suggesting an elevation of the pulmonary arterial systolic pressure. The absence of left ventricular dysfunction helps rule out the cardiac disease as a cause of acute dyspnea. Visualization of a right atrial thrombus confirms the diagnosis.[2]

Bedside echocardiography is a practical diagnostic procedure that in some patients eliminates the need for further studies.

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*To the Editor:* We think Ginsberg failed to emphasize the importance of recurrent embolism. All patients with pulmonary embolism are at risk for further embolism, and therefore, diagnosing pulmonary embolism is not enough. Instead, every patient should be evaluated for residual large thrombi, from the popliteal vein to the vena cava, by Doppler ultrasonography or venography. Patients with large residual thrombi may die from recurrent embolism, whereas patients without thrombi will not. Whether thrombolytic therapy prevents recurrent embolism is unknown. Vena caval filters are effective in preventing pulmonary embolism.[1] Placement of a vena caval filter may be the pivotal step to prevent death — by preventing further embolism in patients with pulmonary embolism who have residual venous thrombi.

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*To the Editor:* We agree with Ginsberg that the duration of anticoagulant therapy in patients who have venous thromboembolism should be determined by balancing the risks of continuing therapy against the risk of recurrent thrombosis and the complications of therapy. He points out that there are no data to assist us in making decisions about the use of anticoagulation therapy in asymptomatic

carriers of a thrombophilic defect (i.e., anticoagulation treatment restricted to high-risk situations vs. life-long prophylaxis) or in these same patients after a first thrombotic event (i.e., treatment for three to six months vs. life-long treatment). However, there is evidence that overall mortality in families with antithrombin deficiency and protein C deficiency does not differ from that in the general population.[1,2] The mortality rates in these families were calculated back into the previous century, before thrombophilia was recognized and before anticoagulation therapy existed. These results also hold true for the most severe type of thrombophilia, type I antithrombin deficiency.[3]

Although these results are general and cannot guide management in the case of patients with recurrent thrombosis or families that appear to be extremely prone to thrombosis, they are reassuring and do not support the use of prophylactic anticoagulation solely on the basis of the presence of the biochemical defect, especially in the light of the known risks of the therapy (a 1 to 3 percent risk of major hemorrhage per year and a 0.3 to 0.6 percent risk of fatal hemorrhage per year).[4,5]

On the basis of these retrospective data, we believe that asymptomatic patients with heritable thrombophilia should not receive anticoagulant agents except in situations in which the risk of thrombosis is increased — for example, after surgery and during immobilization.

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Dr. Ginsberg replies:

*To the Editor:* Ibrahim suggests that echocardiography is a practical procedure that eliminates the need for further studies in some patients with suspected pulmonary embolism. In a patient with suspected pulmonary embolism and a clear-cut right atrial thrombus, a diagnosis of pulmonary embolism can be made. However, the frequency of this finding is probably very low, since the source of pulmonary embolism in most patients is leg-vein thrombosis.[1] Although in the absence of chronic pulmonary disease, a dilated, hypokinetic right ventricle with or without tricuspid insufficiency may be diagnostic of pulmonary embolism, the accuracy of this finding has not been established. Overall, the clinical utility of echocardiography in the evaluation of patients with suspected pulmonary embolism is not known. Furthermore, clinicians should not be distracted from performing tests that have a clear-cut utility in diagnosing pulmonary embolism, such as lung scanning, venous ultrasonography, or pulmonary angiography.

Pechlaner and colleagues suggest that all patients with established pulmonary embolism should undergo extensive evaluation to determine the presence (and extent) of deep-vein thrombosis, with the placement of a vena caval filter if large residual thrombi are present. Such an approach is costly and associated with side effects (of venography, cavography, and insertion of the filter) and in view of the clear-cut efficacy of anticoagulant therapy, cannot be justified. Although some experts recommend insertion of a filter in patients with free-floating thrombi, the necessity for this measure (above and beyond anticoagulant therapy) has never been demonstrated.

I agree with Rosendaal and colleagues that routine anticoagulant therapy is not indicated in asymptomatic patients with thrombophilia,[2] but the data are retrospective, and a small but clinically important increase in mortality in these patients cannot be ruled out. Furthermore, although mortality is the most important complication of venous thromboembolism, there is substantial associated morbidity (the post-thrombotic syndrome and chronic thromboembolic pulmonary hypertension), which may be reduced by long-term anticoagulant therapy.

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## A Pitfall in Assessing Gastric Lymphoma after Eradication of *Helicobacter pylori*

*To the Editor:* Various studies have suggested an important role for *Helicobacter pylori* infection in the pathogenesis of low-grade gastric mucosa-associated lymphoid tissue (MALT) lymphoma. These lymphomas may regress after eradication of *H. pylori* by antibiotics.[1-3] We describe a patient in whom regression of a large infiltrating low-grade gastric lymphoma after antibiotic treatment was documented pathologically, although the macroscopic appearance of the tumor had not changed.

A 39-year-old woman with a short history of epigastric pain was admitted to our clinic in September 1993. Endoscopy revealed a 3-cm-by-2-cm polypoid lesion in the gastric fundus (Fig. 1A). Biopsy specimens showed *H. pylori*–associated gastritis and features of a low-grade B-cell MALT lymphoma. Staging procedures, including endoscopic ultrasonography, demonstrated that the lymphoma was confined to the gastric wall (stage EI2 according to the modified Musshoff staging system). After a two-week course of omeprazole (20 mg twice daily) and amoxicillin (750 mg three times daily), eradication of *H. pylori* was documented histologically and by means of a rapid urease test of biopsy specimens from the gastric antrum and corpus. Careful evaluation of repeated biopsies gave no evidence of persisting lymphoma, but the macroscopic appearance of the tumor was unchanged. Five months later