Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Specific permission to publish should be cited in a covering letter or appended as a postscript.

Iatrogenic or Patient-induced?

To the Editor:

In his letter to the editor (Chest 1988; 93:670), Dr. Forester refers to pneumothorax induced by drug-abusing patients as iatrogenically-created. I would like to point out that the word iatrogenic, based on the Greek word ιατρικος (physician), means "induced by a physician." If all the patients referred to by Dr. Forester happened to be M.D.'s, then perhaps the term iatrogenic does apply. However, pneumothorax induced by an addict that injects drugs into his own jugular vein should be referred to as patient-induced, not iatrogenic.

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Coronary Artery Dissection Following tPA Administration

To the Editor:

I would like to comment on the informative paper by Goulah et al., "Coronary Dissection Following Chest Trauma with Systemic Emboli," (Chest 1988; 93:887-88). In addition to the associated clinical entities, comment should be made with regard to tissue plasminogen activator and acute myocardial infarction.

I recently admitted a 53-year-old man to the hospital with an acute inferior myocardial infarction. There was no previous cardiac history. Physical examination of the cardiovascular system demonstrated an S4 gallop. Electrocardiogram revealed an acute inferior/posterior lateral myocardial infarction. Cardiac isoenzymes, including creatinine phosphatase, were elevated. He received intravenous tissue plasminogen activator with resolution of the chest pain and stabilization of electrocardiographic pattern. The following day he was taken to the cardiac catheterization laboratory where coronary angiography was performed. Coronary arteries were patent with normal left ventricular function. The obtuse marginal circumflex artery demonstrated a localized 2 cm thin radiolucent line consistent with dissection (Fig 1). The patient was subsequently discharged on a calcium channel blocker, nitrate, aspirin and dipyridamole therapy. He returned with recurrent angina two weeks post-discharge and was re-evaluated by coronary angiography and ergonovine maleate testing for coronary artery spasm. Interestingly, the previous area of circumflex artery dissection was completely resolved. The artery appeared to be normal angiographically, with excellent distal flow. Furthermore, there was no evidence of coronary artery spasm at ergonovine induction.

This case, therefore, has several interesting facets. It is associated with the administration of tissue plasminogen activator during acute myocardial infarction. The etiology of the myocardial infarction may have been a primary coronary dissection and subsequent complete resolution of the dissection similar to the author's observation. Furthermore, following the re-establishment of flow with a lytic agent, the patient continued to have angina suggestive of coronary artery spasm.

I am not aware of any previous communication regarding primary coronary artery dissection and tissue plasminogen activator and would appreciate the author's comments.

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Balloons

To the Editor:

I read with great interest the article "Double vs Single Balloon Technique for Aortic Balloon Valvuloplasty" by Midley and associates (Chest 1988; 94:245-50). In this article, the following reference was