Transbronchial Needle Biopsy for Histology Specimens

One of the more exciting recent developments in fiberoptic bronchoscopy has been transbronchial needle aspiration (TBNA). Not surprisingly, the evaluation of this procedure has had two distinct phases which parallel the rigid and fiberoptic eras of bronchoscopy. During the early expansion of diagnostic applications of rigid bronchoscopy, needle puncture of the trachea and proximal bronchi became a valuable adjunct in the diagnosis of bronchogenic carcinoma and in the evaluation of hemodynamic measurements.1 Reasons for this lessenng of interest in the technique, as reflected by published reports if not by numbers of procedures, are unclear. It is likely that Carlens’ introduction of mediastinoscopy in 1959 and the prompt acceptance of this safe and effective approach to diagnosis and staging of mediastinal disease is the most probable contributing factor. There has been no account of untoward outcome or problem with the procedure.

During the past five years, the design of an array of flexible needles has extended these procedures to fiberoptic bronchoscopy, making it more generally available to chest clinicians and their patients.2,3 As with other biopsy techniques, initial reports emphasizing the safety and diagnostic efficacy of the procedure have yielded to investigations extending its applications, addressing limitations of technique identified in early studies, and critically reappraising its worth in patient management. In this regard, phases of enthusiastic introduction, critical re-evaluation, modification, and acceptance of TBNA are representative of those seen in the growth and acceptance of other lung biopsy procedures.

After the successful adaptation of TBNA to the flexible bronchoscope by cytology needle, another major innovation has been achieved through the design of a flexible cutting needle which permitted histologic specimens. In 25 patients, Wang obtained cores of tissue from the mediastinum and hilar areas. This biopsy was diagnostic in 18 patients, and included five patients with sarcomatoid. There were no complications, and in particular, bleeding was not increased despite the use of the 18 gauge needle.4 Preliminary experience with the same prototype needle at other institutions has supported the feasibility and safety of this histologic procedure. Mehta et al5 found that a diagnosis was established in two of five patients with extrinsic compression due to lymphadenopathy.6 Schenk et al (in this issue, see page 272) reported diagnostic histologic specimens in 20 of 29 patients undergoing flexible needle biopsies. There were no complications. These authors found this new approach enhanced the sensitivity of TBNA by cytology and concluded that all patients with bronchogenic carcinoma with a radiologically apparent mediastinal lymphadenopathy should undergo this histologic staging procedure.

While we are stressing that transbronchial needle aspiration represents one exciting example of a new or more properly, rediscovered and refined development of modern bronchology, this procedure itself should not receive disproportionate emphasis. Rather, TBNA is merely one of the many tools available to the chest clinician, better adapting the use of bronchoscopy to overall diagnostic and management strategies, and affording greater latitude in meeting the needs of individual patients. It is in these latter areas, rather than as a technologic advance, that the true value of TBNA resides.7

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REFERENCES

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Coronary Steal
Is It Clinically Important?

Coronary steal is conventionally defined as a fall in absolute coronary perfusion (ml/min/g) of collateralized myocardium after coronary arterial vasodilation, usually after IV administration of dipyridamole. It has been studied experimentally,1-6 modeled theoretically,6 demonstrated in humans,7 and occurs in 10 to 30 percent of patients with coronary artery disease undergoing dipyridamole perfusion imaging, as evidenced by chest pain, ECG changes, and abnormal perfusion scans.6,8 The mechanism is a fall in perfusion pressure at the origin of collateral vessels due to proximal stenoses or to proximal viscous friction developing at high flow rates even in normal arteries from which the collaterals arise. Expressed in terms of circuit models, decreased collateral flow (steal) is due to proportionately greater increase in conductance of the normal vascular bed in parallel with the relatively low, fixed conductance of the collateral bed, which cannot compensate further for the fall in pressure at their origin.6

In view of these mechanisms, the term "steal" is a misnomer, since blood is not "stolen" from the collateralized bed by backward flow through collateral channels to the normal vascular bed. It merely reflects a fall in collateral flow during arteriolar vasodilation below resting control levels, thereby producing ischemia. While developing only in the presence of severe coronary artery stenosis, collaterals in humans protect the myocardium from necrosis and deteriorating contractile function if sufficiently developed over prolonged periods before occlusion occurs.9,10 Although the degree of stenosis and the length of time it is present prior to occlusion are recognized factors in collateral development, genetically mediated angioneogenesis may also be important.18

Coronary subendocardial steal is defined as a fall in absolute subendocardial perfusion with a rise or no change in subepicardial perfusion after coronary arterial vasodilation following IV dipyridamole. As nicely demonstrated in the article by Meerdink et al in this issue of Chest (see p 400), subendocardial steal occurs with severe coronary artery stenosis in the absence of collaterals. Although the effects of low perfusion pressure on transmural flow distribution have been described,1-8,13 this study definitively documents the occurrence and conditions for subendocardial steal associated with severe stenoses in which collaterals play no role. The mechanisms are the same as outlined previously. At normal resting conditions with severe stenosis, resting flow and/or distal coronary pressure are reduced enough to stimulate compensatory subendocardial vasodilation, thereby using up its limited flow reserve. Subendocardial conductance is therefore relatively fixed. In these circumstances, IV dipyridamole then causes subepicardial arterioles to vasodilate proportionately more than subendocardial arterioles. Consequently, absolute perfusion falls in the subendocardium due to greater increase in conductance of subepicardial vessels that are in parallel with relatively fixed conductance vessels of the subendocardium, which cannot compensate for the fall in distal pressure. The necessary conditions for subendocardial steal are a severe stenosis, which produces maximally vasodilated, fixed conductance, subendocardial arterioles, and a fall in distal pressure after dipyridamole administration, which causes lower subendocardial perfusion. Thus, the mechanisms for coronary collateral steal and subendocardial steal are the same, but the anatomy producing them is different.

Clinically, coronary steal, as manifested by chest pain and ST depression after IV dipyridamole, is usually a sign of severe coronary artery disease with viable myocardium. In my experience, steal in the absence of collaterals, ie, subendocardial steal, is not commonly seen clinically, since these patients often have severe or unstable angina at rest and are therefore likely to be excluded from dipyridamole stress. Consequently, in the majority of patients undergoing appropriate dipyridamole perfusion imaging, coronary steal is a sign of collaterals providing significant resting flow rather than subendocardial steal without collaterals. Figure 1 illustrates a clinical example of coronary steal demonstrated by positron emission tomography (PET) as a fall in stress activity below resting levels. PET also provides the percent of the heart outside of 2.5 SD from normal which, for the polar map display of the ratio of absolute counts stress/rest, indicates the percent of the heart that is collateralized (Fig 2).

A rare patient with the hyperadrenergic syndrome of mitral valve prolapse may have angina and ST depression after IV dipyridamole relieved with amrinophylline but no regional perfusion defect on PET scanning. These patients may have a form of sympathetically driven "fixed" subendocardial conductance