DISCUSSION

Mycobacterial disease has been found to be common in patients infected with HIV, often presenting as extrapulmonary or disseminated disease. Although intrathoracic mediastinal lymphadenopathy is more usually found in HIV-associated Mycobacterium tuberculosis, the finding of MAI in mediastinal nodes has been observed. Given the rising number of cases of tuberculosis being reported and its strong association with HIV infection, it can be expected that increasing numbers of HIV-infected patients with mediastinal mycobacterial lymphadenopathy will be seen. In this light, the potential application of TBNA for diagnosing mycobacterial mediastinal lymphadenopathy can be appreciated.

Prior to the development of TBNA and its adaptation for use with the fiberoptic bronchoscope, mediastinal lymphadenopathy required more invasive diagnostic procedures such as mediastinoscopy, anterior mediastinotomy, or thoracotomy. A number of studies have now confirmed the utility of TBNA for diagnosing mediastinal cancer, thus often obviating the need for surgical intervention. In addition, these studies have confirmed the safety of TBNA with most investigators not encountering complications of clinical significance. Nevertheless, despite the well-established role of fine-gauge TBNA for diagnosing mediastinal carcinoma, its usefulness in making benign diagnoses has been limited. To overcome this limitation, Wang introduced 18-gauge flexible transbronchial needle aspiration biopsy to obtain histologic specimens of mediastinal lymph nodes. Benign diagnoses have been made using 18-gauge TBNA, but to our knowledge a TBNA diagnosis of mycobacterial disease involving the mediastinum has not been previously reported. It is quite possible that since most patients have had TBNA performed for the express purpose of diagnosing mediastinal cancer, acid-fast smear has not been routinely performed.

This report, however, highlights the potential role of TBNA in diagnosing intrathoracic lymphadenopathy resulting from mycobacterial infection, hopefully sparing such patients more invasive operative procedures. Given the safety and technical ease of TBNA, it is our recommendation that during TBNA, at least one aspirate should be obtained for acid-fast smear and culture if a diagnosis of mycobacterial disease is being entertained.

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The Use of Oral Labetalol in the Treatment of Arrhythmias Associated with the Long QT Syndrome*

Blair P. Grubb, M.D.

A 7-year-old white boy with the long QT syndrome began to experience recurrent syncope associated with torsade de pointes ventricular tachycardia in spite of beta-blocker therapy. The patient was therefore given a combined alpha-and beta-blocking agent (labetalol) with complete suppression of the syncope episodes. This suggests a role for combined alpha- and beta-blocking agents in the therapy of arrhythmias associated with the long QT syndrome.

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The use of oral beta blockers has long been the principal therapy for the prevention of the arrhythmias associated with the congenital long QT syndrome. A growing body of evidence, however, has suggested that alpha-receptor stimulation also may be important in provoking the rhythm disorders associated with this condition and thus alpha-blocking agents may potentially provide an additional therapeutic benefit. Labetalol hydrochloride is a nonselective beta blocker with alpha-1 blocking properties. We describe the successful use of oral labetalol therapy in a patient with the congenital long QT syndrome whose arrhythmias proved resistant to beta-blocker therapy alone.

CASE REPORT

A 7-year-old white boy was brought to the hospital following multiple syncopal episodes.

The child had been diagnosed as having the long QT syndrome at age 5 following multiple syncopal episodes associated with torsade de pointes ventricular tachycardia. The child's mother had a known history of long QT syndrome and was receiving oral beta-blocker therapy. The child had a male sibling who had been diagnosed as having the long QT syndrome and who had died suddenly at age 10 years after having been called to the school principal's office.

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1724

Oral Labetalol for Arrhythmia Treatment (Blair P. Grubb)
Following his initial diagnosis at age 5 years the patient was placed on oral propranolol therapy at a dosage of 20 mg orally every 8 h. He did well until age 7 years, when he again began to experience syncopal episodes following exertion. During these episodes the child would suddenly lose consciousness and become cyanotic and incontinent. The dose of propranolol was increased to 40 mg orally every 6 h; however, the episodes continued. A Holter monitor demonstrated torsade de pointes ventricular tachycardia associated with one of his episodes of syncope (Fig 1). The patient was admitted to the hospital and the propranolol dose was increased to 50 mg.

An exercise stress test was performed with a modified Bruce protocol. In stage 2, the patient began to experience frequent ventricular premature beats, followed by two- and three-beat runs. In stage 3, the test was terminated due to the development of nonsustained runs of ventricular tachycardia.

The patient and his family were offered left stellate ganglion resection; however, they were reluctant to pursue surgery. Thereafter, therapy was started with oral labetalol and titrated to a dosage of 75 mg orally every 12 h. A repeat stress test was performed and the patient was able to exercise into stage 4 of a Bruce protocol without the development of ventricular ectopy. An epinephrine infusion test was performed per the protocol of Jackman et al. The patient was able to tolerate an infusion of up to 0.6 µg/kg before single and double premature ectopic complexes appeared. The patient has been symptom-free for a period of seven months with a normal activity level.

**Discussion**

The congenital long QT syndrome is a heritable electrical disorder of the heart that principally affects young women and is associated with a propensity to syncope and sudden death. These events are often brought on by intense emotions, vigorous physical activity, or an auditory startle stimulus. It has been noted that patients with the long QT syndrome tend to have reduced resting heart rates that do not increase normally with exercise. Thus, since heart rate control depends almost exclusively on the right stellate ganglion, it has been suggested that the primary abnormality might be diminished right cardiac sympathetic activity reflexly resulting in abnormally increased left cardiac sympathetic activity. Recognition of the role of sympathetic tone in the genesis of the arrhythmias associated with the long QT syndrome has led to the use of beta-adrenergic blocking agents as primary therapy. Several investigators, however, have noted that surgical ablation of the left stellate ganglion can reduce the incidence of both syncope and sudden death in patients who have long QT syndrome who have not responded to beta-blocking agents alone. This seems to indicate an important role for alpha-receptor stimulation in the genesis of these arrhythmias, since stellate ganglion ablation results in alpha- as well as beta-adrenoceptor reduction. Thus, in this patient who did not want to pursue stellate ganglionectomy or permanent pacing it seemed logical to attempt to try combined alpha and beta blockage. Although the dose of propranolol may have been increased further, it seemed quite unlikely that larger doses would have proven successful.

Labetalol hydrochloride is a nonselective beta blocker as well as an alpha-1-adrenergic blocking agent. When given orally the beta to alpha blocking ratio is 3:1. We chose to use it because it is an easy, convenient, and well-tolerated agent.

Thus far, the patient has done remarkably well on labetalol and has had no further episodes of syncope. We conclude that combined alpha and beta blockage may be an acceptable alternative in controlling the arrhythmias associated with the long QT syndrome in patients who are unresponsive to beta blockage alone. Further studies will be necessary, however, to determine the safety and efficacy of labetalol in the control of arrhythmias associated with the long QT syndrome.

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