Figure 3. Drawing of outflow portion of right ventricle. Fibrous continuity between pulmonary valve and mitral valve; no subpulmonary conus.

Preoperatively, ventricular septal defect was closed with a Dacron patch, assuming the conduction tissue to be posterior margin of the defect. There was a cleft in the anterior leaflet of the mitral valve, which was repaired by suturing. At the end of the operation, the pulmonary artery pressure was 55/30 mm Hg, and the aorta, 85/55 mm Hg. The postoperative course was uneventful, without any rhythm disturbances, and he was discharged from the hospital.

Discussion

From the review of the literature, there are ten successful surgical cases of the anatomically corrected malposition of the great arteries, all of which have bilateral conus. Van Praagh et al reported two autopsy cases of anatomically corrected malposition of the great arteries without subpulmonary conus. To our knowledge, our patient is the first reported case of successful surgical repair of anatomically corrected malposition of the great arteries without subpulmonary conus and with ventricular septal defect, mitral regurgitation, pulmonary hypertension, and single coronary artery. The abnormal relation of the great arteries without subpulmonary conus can be most frequently seen in corrected transposition of the great arteries, in which the aorta arises from the morphologic right ventricle and the pulmonary artery from the morphologic left ventricle. Because of the technical problem of the ventricular septal defect closure to avoid the conduction pathway, anatomically corrected malposition and corrected transposition must be differentiated precisely. In the case of corrected transposition, the conduction tissue runs in the anterior aspect of ventricular septal defect. By contrast, in the anatomically corrected malposition, the conduction tissue runs in the posterior aspect of the defect, and consequently the defect can be patch-closed in a usual fashion as described here. Absence of subpulmonary conus does not necessarily mean that corrected transposition of the great arteries is present. As our case illustrates, absence of the subpulmonary conus can and does occur with anatomically corrected malposition. We would like to emphasize the surgical importance of the differentiation of these two congenital cardiac malformations.

Further Observations on the Electrophysiologic Effects of Oral Amiodarone Therapy*

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A case is presented of a reversible intra-Hisian block occurring under amiodarone treatment for atrial tachycardia in a patient without clear intraventricular conduction abnormalities. His bundle recordings showed an atrial tachycardia with intermittent exit block and greatly prolonged BH and HV intervals (40 and 100 msec, respectively). Thirty days after amiodarone discontinuation, his bundle electrograms showed atrial flutter without intra-Hisian or infra-Hisian delay. Amiodarone should be used with caution during long-term oral therapy in patients with or without clear intraventricular conduction defects.

Amiodarone has been shown to be effective in the therapy of various atrial and ventricular arrhythmias. The presence of intraventricular or atrioventricular block is considered a relative contraindication. Rosenbaum et al considered the presence of intraventricular block or AV block as a contraindication to oral therapy with amiodarone, even though no adverse symptom due to this complication was mentioned. This report describes an intermittent exit block from an atrial tachycardia and a reversible infra-Hisian delay under amiodarone treatment in a patient without clear intraventricular conduction abnormalities.

Case Report

A 38-year-old man was admitted to our department because of dyspnea and palpitations with recurrent paroxysmal atrial flutter dating from 1970. In 1971 he underwent commissurotomy for rheumatic mitral valve stenosis.

Previous treatment with digitalis, diuretics, quinidine, and disopyramide in various doses and combinations failed or was minimally effective in the prevention of the arrhythmias.

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References


Chest 82/1 July 1982

117
rhythm. Fifteen days before the present admission, use of all drugs was discontinued, and amiodarone therapy was started at a dose of 600 mg/day orally.

On admission, the physical examination revealed signs of mitral stenosis without congestive heart failure. Routine ECGs showed an atrial tachycardia with various degrees of AV block and normal QRS duration.

Results of blood tests to evaluate hepatic and renal function and serum electrolyte levels were within normal limits. Cardiac catheterization showed mild mitral stenosis and minimal mitral regurgitation. Coronary arteriography showed a 90 percent stenosis of the ostium of the right coronary artery.

His bundle electrograms, performed before the cardiac catheterization, were recorded according to the method of Scherlag et al. The recordings demonstrated an atrial tachycardia with an atrial rate of 188/min and varying degrees of intranodal block. Intermittently, the atrial tachycardia abruptly slowed to an atrial rate of exactly one half of the previous rate (Fig 1 and 2). The P wave morphology and axis were identical at 94 beats/min and 188 beats/min, but different from sinus P wave morphology, suggesting the diagnosis of atrial tachycardia with intermittent 2:1 exit block from an ectopic focus. Progression of the atrial tachycardia from a rate of 188/min to a rate of 94/min and vice versa occurred spontaneously and after carotid sinus massage. The atrial tachyarrhythmia with a rate of 188/min was brought out by carotid sinus massage, suggesting that the maneuver would somehow decrease the degree of exit block from the ectopic focus (Fig 2). This phenomenon was reproducible. The His bundle electrograms revealed a widened BH deflection (40 msec) and a prolonged HV interval (100 msec) both at atrial rate of 188/min and 94/min (Fig 1, and 2). Validation of the BH deflection by stimulation through the electrodes from which this deflection was obtained failed because intervening ventricular muscle was stimulated simultaneously. Incremental atrial pacing was attempted when the atrial rate was 94/min.

![Figure 1](http://journal.publications.chestnet.org/pdfs/access.ashx?url=/data/journals/chest/21300/)

**Figure 1.** His bundle recordings (HBE) under amiodarone treatment show atrial rhythm at rate of 94/min (cycle length, 640 msec) with 1:1 AV conduction. AH interval is 90 msec, whereas BH and HV intervals are 40 and 100 msec, respectively. Paper speed is 100 mm/sec, and time lines are at 1-sec intervals.

![Figure 2](http://journal.publications.chestnet.org/pdfs/access.ashx?url=/data/journals/chest/21300/)

**Figure 2.** Left-sided carotid sinus massage (CSM) during atrial rhythm at rate of 94/min results in atrial tachycardia at rate of 188/min with varying degree of intranodal block and 3:1, 2:1 AV conduction. Note prolongation of AH interval from 90 to 210 msec immediately after CSM; BH and HV intervals remain unchanged (40 and 100 msec, respectively). Paper speed is 100 mm/sec, and time lines are at 1-sec intervals.
but was unsuccessful because the atrial tachycardia at a rate of 188/min reappeared and preempted atrial pacing. However, both spontaneously and during carotid sinus massage, high degree AV block occurred without alteration of the underlying atrial mechanism. During high degree AV block nonconducted atrial complexes failed to produce H, while the A-H intervals for conducted beats were prolonged and each H was followed by a QRS complex with a constant H-V interval.

Since amiodarone may cause HPS disturbance, and the drug therapy was discontinued. Thirty days later His bundle electrograms revealed atrial flutter and sporadic spontaneous conversion of atrial flutter to sinus rhythm which lasted several beats to seconds. The BH and HV intervals during atrial flutter were 15 and 40 msec, respectively (Fig 3). During sinus rhythm, the PA, AH, and HV intervals were 35, 90, and 40 msec, respectively. The frequency of recurrent paroxysmal atrial flutter was reduced by propranolol, and the patient was discharged in stable sinus rhythm.

**Discussion**

Exit block has been defined as failure of an ectopic impulse to propagate to the adjacent myocardium. Its occurrence in atrial tachycardia is rare. Drug-related exit block from an atrial tachycardia has never been reported. Lidocaine administration has been reported to both slow and increase the degree of exit block from a parasystolic ventricular focus.

In the case reported herein, it is likely that amiodarone increased the refractory period of the atrium so that fewer impulses were able to reach the atrial myocardium. After amiodarone therapy was discontinued, an atrial flutter at rate of 280/min occurred, and exit block disappeared.

Intra-Hisian block under chronic amiodarone administration has never been described. However, ample evidence exists that chronic therapy with amiodarone may slow conduction in the human and animal intraventricular conduction system. Indeed, Rosenbaum et al reported a further deterioration of conduction in some patients (8.8 percent of 65 patients) with preexisting intraventricular conduction disturbance, during chronic oral treatment with this drug.

Our case clearly indicates that therapy with amiodarone is capable of inducing HPS conduction delay even in patients without surface ECG defects and that this delay can be completely reversible after drug discontinuation. Actually, the HPS delay was mainly seen as a remarkable prolongation of the HV interval of which the BH deflection was a significant part. There was HV prolongation distal to the recording site of the BH deflection. Since the QRS width remained unchanged, it is likely that this prolongation in the HV interval represented an intra-Hisian delay. Heger et al recently reported some increase in the HV interval in four of five patients during amiodarone therapy. However, there was no mention of intraventricular conduction abnormalities or widening of the BH deflection.

It is not improbable that in our patient there was a latent His bundle delay induced by chronic ischemia. Under such conditions, it is conceivable that long-term amiodarone administration uncovered the intra-Hisian delay.

In conclusion, amiodarone-induced intermittent exit block may prevent capture of the atrium by a rapidly discharging ectopic atrial pacemaker; however, chronic treatment with this drug may result in HPS conduction abnormality even in patients without conduction defects seen on surface electrocardiograms. Although the intra-Hisian delay appears to be reversible, caution should be used in chronic therapy with this drug especially in consideration that the half-life of amiodarone, its therapeutic blood concentration and its metabolism still need to be better determined.
Cardiac Tamponade as a Complication of Thin-needle Aspiration Lung Biopsy

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Thin-needle aspiration lung biopsy has become an important diagnostic technique in chest disease. Complications, other than pneumothorax, are infrequent. We report a case of acute cardiac tamponade complicating biopsy of a lesion near the mediastinum. Caution should be exercised in selecting patients for, and in performing biopsies in, or close to the mediastinum.

Thin-needle aspiration biopsy performed under fluoroscopic guidance is an accepted and effective diagnostic technique. It has been shown to have a low morbidity and rare mortality. Complications include pneumothorax, hemoptysis, hemothorax, air embolism, pulmonary hemorrhage, myocardial infarction, and infection.1–4 There is a single report of malignant seeding through the needle tract.5 Of more than 2,500 procedures performed at our institution, there have been no fatalities, and the only serious complications, or those requiring immediate attention, have been tension pneumothorax, one case of massive hemoptysis, and one case of myocardial infarction. None of these had serious sequelae.

This report describes a healthy, middle-aged woman who had a hemopericardium resulting in acute cardiac tamponade after thin-needle aspiration biopsy of a lung nodule. To our knowledge this complication has not been previously reported.

CASE REPORT

A 43-year-old woman had a solitary noncalcified nodule of undetermined cause noted incidentally on a routine chest roentgenogram. The differential diagnosis included malignancy. It was decided that a tissue diagnosis was required. The large majority of such patients at our institution undergo needle aspiration biopsy rather than thoracotomy, since needle biopsy has proven to be very effective and accurate.

The aspiration biopsy showed that the nodule was 1.8 cm in diameter, well-defined, noncalcified, and located in the right upper lobe adjacent to the mediastinum (Fig 1). One month previously an aspiration biopsy of the same nodule had been performed without complication. Cytopathologic examination revealed inflammatory cells. The patient returned for repeated biopsy.

The technique used was that described by Sanders et al.6 With the patient supine, the nodule was visualized on the image intensifier. With use of local anesthesia, a 10-cm disposable 20-gauge needle was introduced through the anterior chest wall. Three passes were needed to achieve optimum position of the needle tip. The stylet was then withdrawn and the nodule aspirated. The patient was perfectly well throughout the procedure. Following aspiration the needle was withdrawn from the chest and the aspirate plated on slides. Within seconds the patient became obtunded, and respiratory distress and cyanosis developed. Spontaneous respiration ceased. The cardiac arrest team was called.

The patient had a weak but regular carotid pulse. A peripheral blood pressure could not be obtained. There was no evidence of pneumothorax on physical examination or on a portable chest roentgenogram, although the cardiac silhouette and superior mediastinum appeared enlarged on the roentgenogram. An ECG showed sinus rhythm and no evidence of myocardial infarction.

Treatment was begun with oxygen, 1 L of normal saline solution intravenously (IV), dopamine, and bicarbonate, but there was little response. At this time a marked pulsus paradoxus was noted, and a diagnosis of acute cardiac tamponade was made. An emergency pericardiocentesis with a No. 20 needle via a left subxiphoid approach was performed; 65 ml of bloody pericardial fluid was aspirated.

Afterward the blood pressure rose to 110/70 mm Hg, the cyanosis improved, and there was a reduction in the pulsus paradoxus. M-mode echocardiography following pericardiocentesis revealed a small residual effusion. The patient was transferred to the intensive care unit and had an uneventful recovery. There was no evidence of myocardial infarction. Serial echocardiograms showed no reaccumulation of pericardial blood.

At a follow-up visit three months later, she felt perfectly well. Unfortunately, a final diagnosis had not been made.

REFERENCES