decannulation are illustrated in Table 1.

The patient was weaned from ventilator support and the tracheostomy removed two weeks postdecannulation. After a brief period of physical therapy, she returned home, with no medications or supplemental oxygen. Four months after discharge, she has normal arterial blood gas tensions on room air, and vital capacity has increased to 68 percent predicted level. She enjoys normal activity levels with no dyspnea.

**DISCUSSION**

This patient demonstrates the remarkable potential for a patient with severe ARDS to recover when gas exchange can be supported for a prolonged period of time, and when multiple organ failure can be avoided. Despite extreme impairment in lung compliance and gas exchange, and marked pulmonary hypertension and barotrauma, her physiologic derangements were gradually successfully reversed, allowing her to resume a normal quality of life.

Previous reports have emphasized that significant improvement in lung mechanics and gas exchange usually occurs rapidly (within 48 h) in "responders" to extracorporeal CO$_2$ removal. Nonetheless, when the degree of lung dysfunction is more profound, and when mechanical ventilation has been prolonged prior to extracorporeal support, lung function may not improve for several weeks, as in the present case. At this time, no satisfactory criteria indicating lack of reversibility of ARDS are available, other than terminal multiorgan system failure. Open lung biopsy indicating severe fibrosis and obliteration of alveoli has been utilized to justify withdrawal of support; however, biopsy findings in ARDS may not predict subsequent lung function.

Bleeding has been the major complication of extracorporeal membrane oxygenation, largely related to the requirement for anticoagulation to prevent circuit thrombosis and hematologic derangement. We utilized a system in which all blood-contacting surfaces had been coated with heparin. In this process, partially degraded heparin is covalently endpoint-attached to the artificial surface. Thrombin circulates through the heparin coating to sites of antithrombin-heparin binding. The inactivated thrombin-antithrombin complex then leaves the surface, allowing continuing interaction with circulating thrombin. The surface-bound heparin fragments may also inhibit factor Xa. This simulates normal endothelial antithrombotic function, and permits perfusion with small amounts of systemic heparin.

The limited world experience with extracorporeal CO$_2$ removal/low frequency positive pressure ventilation suggests improved survival in patients expected to have a high mortality from severe ARDS. For those patients with near-terminal ARDS, extracorporeal support utilizing heparin-coated surfaces may offer some hope.

ACKNOWLEDGMENTS: The authors thank the nurses, respiratory therapists, and perfusionists who constitute the Sharp Extracorporeal Support Team.

**REFERENCES**


JAMA 1986; 256:881-86

**Orthostatic and Exercise-Induced Advanced Nodal Atrioventricular Block**

Josep Reig, M.D.,†§ Enric Domingo, M.D., Ph.D., F.C.C.P.,‡§ Josep Reguant, M.D.,† and Josep Corrons, M.D.,†

A 69-year-old woman was referred for asthenia and dizziness when walking in the last two months. No clinical abnormalities were found, and sinus rhythm was present when lying down. On orthostatism and walking, advanced AV block developed. Atropine and isoproterenol ameliorated the AV conduction abnormality, suggesting a nodal block. The patient remained asymptomatic after pacemaker implantation.

(Chest 1992; 102:970-72)

Exercise-induced atrioventricular block in patients with normal AV conduction at rest is infrequent, although it has been described previously. However, we are not aware of any published report of an orthostatism-induced advanced AV block with supine 1:1 AV conduction. We report the clinical features of a patient with orthostatic and exercise-induced AV block successfully treated with pacemaker implantation.

**CASE REPORT**

A 69-year-old woman without any previous cardiovascular or neurologic symptoms, presented with walking-related asthenia and dizziness (no syncopes) during the previous two months. Physical examination, chest x-ray film, routine blood and urine tests, bidimensional echocardiography and Doppler, and thoracic CT-scan results were within normal limits. Resting ECG revealed sinus rhythm (60-70 bpm) with first degree AV block (PR = 0.24 s) and incipient right bundle branch block (Fig 1). Immediately after standing up, the patient developed a 2:1 AV block with sinus tachycardia, no change in QRS morphologic condition, and a ventricular rate of 40 to 45 bpm (Fig 15), without any clinical symptoms. Twenty-four hour Holter reading showed 2:1 AV block

*From the †Servei de Cardiologia, Centre Hospitalari-Unitat Coronària, Manresa; §Servei de Cardiologia, Hospital Vall d’Hebron, Barcelona; and ‡Departament de Fisiologia, Facultat de Medicina, Universitat Autònoma de Barcelona, Spain. Reprint requests: Dr. Reig, Unitat Coronària, Capuchina 28, Manresa, Spain 08240
normal with standing up and exercise: (BP at rest and supine, 125/80; at rest and standing, 130/85; and at the end of exercise, 170/70 mm Hg).

Exercise was stopped at 1 min 51 s because of severe dizziness and exhaustion. No chest pain or ECG ischemic changes were detected. Immediately after stopping exercise and when still standing up, advanced AV block regressed to 2:1 AV block. Immediately after lying down, 1:1 AV conduction reappeared (Fig 1L). The same exercise protocol was repeated first under atropine and second, under isoproterenol.

With atropine (1.5 mg IV) (Fig 2, left) heart rate increased (79 bpm) and P-R interval normalized at rest (Fig 2A), and sinus tachycardia (96 bpm) developed in standing up position (Fig 2B). During exercise, 1:1 AV conduction was maintained up to a heart rate of 142 bpm at 3½ min. At this heart frequency, 2:1 AV block progressively appeared (Fig 2C) and remained until exercise was stopped because of exhaustion at 6 min (Fig 2D). Normal AV conduction restarted within the first minute after the end of exercise (Fig 2E). Blood pressure changes were similar to those reported in basal state, and no ischemic ECG changes were detected.

The isoproterenol test (Fig 2, right) revealed similar changes. Sinus tachycardia (142 bpm) with normal PR interval developed at rest (Fig 2A), with $2 \times 10^{-4}$ mg/kg/min saline solution infusion of isoproterenol. In standing position, the infusion rate was $1 \times 10^{-4}$ mg/kg/min, and heart rate reached 130 bpm (Fig 2B) with 1:1 normal conduction. This infusion rate was maintained during the exercise stress test. At 5½ min, sinus tachycardia reached 150 bpm and 2:1 AV block progressively appeared (Fig 2C) and remained until exercise was stopped because of exhaustion at 7.45 min (Fig 2D). Normal AV conduction restarted within the first minute after the end of exercise (Fig 2E). Blood pressure changes were similar to those reported in basal state, and no ischemic ECG changes were detected. The patient was treated with pacemaker implantation and remained asymptomatic thereafter. She did not accept electrophysiologic study.

**DISCUSSION**

Only a few cases of exercise-induced advanced AV block

**ATROPINE**

**ISOPROTERENOL**

---

**FIGURE 1.** Basal resting supine 12-lead ECG. S, standing up and resting, lead 2 ECG (2:1 AV block). E, end of exercise, lead 2 ECG (advanced AV block); L, lying 1 min after exercise, lead 2 ECG (sinus rhythm).

and advanced AV block during daytime when standing up. By night, when lying down, sinus rhythm was maintained without any advanced AV block episode. Wenckebach episodes were not present at any time in Holter recording. Transition between 1:1 AV conduction and advanced AV block was always sudden, without detection of transient Wenckebach episodes between both conditions. Holter recording showed a heart rate between 60 to 70 bpm at night lying down (sinus rhythm with 1:1 conduction), and during most of the day, ventricular rate was under 50 with a sinus rhythm around 85 bpm. During treadmill exercise test (Bruce protocol), at the end of the first minute of stage 1, the patient experienced asthenia and dizziness with simultaneous advanced AV block, sinus tachycardia (135 bpm), a ventricular rate of 50 bpm, and no changes in QRS morphologic findings (Fig 1E). Blood pressure changes were

**FIGURE 2.** Lead 2 ECG. Atropine test in left panel; isoproterenol test in right panel. Supine resting (A), standing resting (B), initiation 2:1 AV block (C), end of exercise (D), 1 min after exercise (E).
have been reported. Electrophysiologic studies in some of these cases revealed block distal to the AV node.\textsuperscript{1,4} Vagolytic effect, and mediated by exercise or atropine, tends to improve AV nodal conduction. However, it has no such effect on infranodal block since the more distally located lesion is beyond vagal influence.\textsuperscript{2,4} \(\beta\)-adrenergic receptor stimulation, induced either by exercise or isoproterenol, improves or speeds impulse conduction through the AV node, without inducing any major changes in the infranodal conduction system.\textsuperscript{4} In fact, several investigators have postulated that the site of AV block occurring during exercise is more likely to be localized to the distal His-Purkinje system rather than the AV node because the His-Purkinje system is relatively insensitive to autonomic modulation, and it has a relatively fixed effective refractory period that fails to decrease sufficiently with decreasing atrial cycle length to permit 1:1 AV conduction.

The case herein described may represent a nodal exercise-induced AV block since it could be corrected with vagolytic or sympathomimetic drugs. We are not aware of any other similar cases reported so far. In addition, in our case, the 2:1 AV block appeared only when standing up, and thus, before starting exercise. We believe this is the first reported case of orthostatic 2:1 AV block.

The fact that sinus rate and blood pressure were normal during postural changes and exercise suggests that the global body autonomic response to standing up and exercise (vagolysis and sympathetic discharge) were maintained. These observations might lead to the conclusion that our patient had a decreased response to a normal autonomic influence on the AV node.

Gilchrist\textsuperscript{*} first advocated the use of exercise to differentiate type 1 from type 2 AV block: type 1 improves whereas type 2 deteriorates with exercise, because the vagolytic effect of exercise does not have any influence on type 2 AV block (distally located beyond vagal influence). In view of our patient's behavior, we can postulate that exercise-induced advanced AV block is of infranodal origin if atropine or sympathomimetic drugs do not improve AV conduction, but if it is of nodal origin, these drugs ameliorate its AV conduction.

REFERENCES
4 Wit AL, Hoffman BF, Rosen MR. Electrophysiology and pharmacology of cardiac arrhythmias: IX. Cardiac electrophysiologic effects of beta adrenergic receptor stimulation and blockade: Part A. Am Heart J 1975; 90:521-33

Polypoid Endobronchial Lesions*  
A Manifestation of Bacillary Angiomatosis

Leonard N. Slater, M.D.;\textsuperscript{†} and Kyung-Whan Min, M.D.\textsuperscript{‡}

Polypoid endobronchial lesions occurred in a patient with acquired immunodeficiency syndrome (AIDS) with recent fever, skin lesions, lymphadenopathy, lung infiltrates, and pleural effusions. His condition improved with antimicrobials and vincristine. After therapy ceased, skin lesions recurred and gastroesophageal mucosal lesions developed.

Bacillary angiomatosis was identified during retrospective analysis of skin and endobronchial biopsy specimens.

(Chest 1992; 102:972-74)

Bacillary angiomatosis (BA) is a vascular proliferative disorder originally described involving skin and local lymph nodes of human immunodeficiency virus (HIV)-infected persons.\textsuperscript{1} Warthin-Starry (WS) silver staining demonstrates bacilli within these lesions.\textsuperscript{2,3} Extracutaneous BA has since been reported in immunocompromised hosts.\textsuperscript{4} Bacillary angiomatosis also has been described in an immunocompetent person.\textsuperscript{8} We report a case of visceral dissemination of BA, manifesting in part as polypoid endobronchial lesions.

CASE REPORT

A 42-year-old man with acquired immunodeficiency syndrome (AIDS) developed violaceous papules of the face and left groin and left inguinal lymphadenopathy in January 1988. He was soon hospitalized with chest pain, fever, chills, nausea, productive cough, and left leg swelling. He had a temperature of 38.9\textdegree C, thrush, polypoid lesions of the oropharynx, face, trunk, and extremities, and enlargement of the left lower extremity. He was hypoxemic, with right-sided alveolar and interstitial infiltrates and bilateral pleural effusions on roentgenograms. Sputum contained moderate numbers of leukocytes and predominantly Gram-negative coccobacilli. Thoracentesis yielded exudative fluid containing few mononuclear leukocytes and no organisms on stains or cultures. Left lower extremity venography was normal. Computed tomography revealed cholelithiasis, hepatosplenomegaly, retroperitoneal lymphadenopathy, and pancreatic heterogeneity.

With empiric trimethoprim/sulfamethoxazole (TMP/SMX), fever remitted in five days. Sputum culture yielded Hemophilus parainfluenzae and oral flora. Hematoxylin-eosin (H&E)-stained sections of a facial lesion biopsy specimen were reported consistent with Kaposi's sarcoma (KS), while a groin lesion biopsy specimen was interpreted as KS vs BA. Bronchoscopy revealed polypoid endobronchial lesions (Fig 1) in the midtrachea, right upper lobe anterior segment bronchus, and right lower lobe medial basal segment bronchus. Biopsy specimens of all endobronchial lesions were obtained; their histologic features were reported consistent with KS.

*From the University of Oklahoma College of Medicine, Oklahoma City.
†Associate Professor of Medicine.
‡Associate Professor of Pathology.

Request reprints: Dr. Slater, 921 NE 13th Street, Oklahoma City 73104