able conduit in the emergency setting. Furthermore, we surmise that the present cost of tissue engineering would be cost-prohibitive, especially for developing countries.

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Treating Heart Failure and Dyspnea in a 78-Year-Old Man With Surgical Correction of Pectus Excavatum

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FEATURE ARTICLES

Reports documenting the cardiopulmonary significance of pectus excavatum are limited, and there remains a lack of consensus as to whether surgical repair can improve function. We present a case of pectus excavatum deformity and heart failure. Surgical repair was performed with significant improvement of performance and heart function.

(Ann Thorac Surg 2009;88:1008–10)

Pectus is a common congenital anomaly in Caucasian males [1]. Frequently recognized during childhood, pectus excavatum (PE) has few symptoms. Despite some reports of cardiopulmonary significance, many patients do not undergo surgical repair. Worsening with age of physiologic impairment is medically undocumented but expressed by many patients [2]. We describe a patient with PE deformity presenting with severe dyspnea and dilated cardiomyopathy. Surgical repair was performed allowing more aggressive medical management and improvement in his symptoms of heart failure.

A 78-year-old man presented to the emergency room with progressive dyspnea and orthopnea. Dyspnea with exertion developed in him 6 months prior. His symptoms progressed until he was unable to perform any activities without dyspnea. Orthopnea prevented normal sleep; however, he denied lower extremity edema, weight gain, or chest discomfort.

Three years prior, he was diagnosed with left bundle branch block. Further evaluation included a normal dobutamine stress echocardiogram. At age 64, he was diagnosed with hereditary peripheral neuropathy Charcot-Marie-Tooth type 2. He was managed with ankle-foot orthoses and had been able to maintain activities of daily living including a daily stationary cycling exercise program.

Pertinent vitals included heart rate of 110 beats per minute, pressure of 140/80 mm Hg, and respiratory rate of 22 with oxygen saturation 97% on ambient air. On examination he was thin and appeared comfortable while speaking. Jugular venous pressure was normal. Lungs were clear and heart tones were laterally displaced without murmurs or gallops. A central bowl-type PE was noted. Liver span was unremarkable and there was no lower extremity edema. Peripheral vascular examination was normal. Intrinsic hand and distal leg muscles showed atrophy and weakness consistent with his neuropathy.

Electrocardiogram showed normal sinus rhythm, left bundle branch block with QRS duration of 142 ms. Chest roentgenogram was unremarkable with exception of PE. Laboratory studies were normal other than elevated B-type natriuretic peptide: 283 pg/mL (normal $<$ 83 pg/mL). A transthoracic echocardiogram revealed a dilated cardiomyopathy with enlargement of both ventricles. Left ventricular ejection fraction was 10% to 15% with moderate-severe decrease in right ventricular function. There were no valvular or pericardial abnormalities seen. The left ventricular end-diastolic dimension was 57 mm. Pulmonary artery pressure was not elevated. He

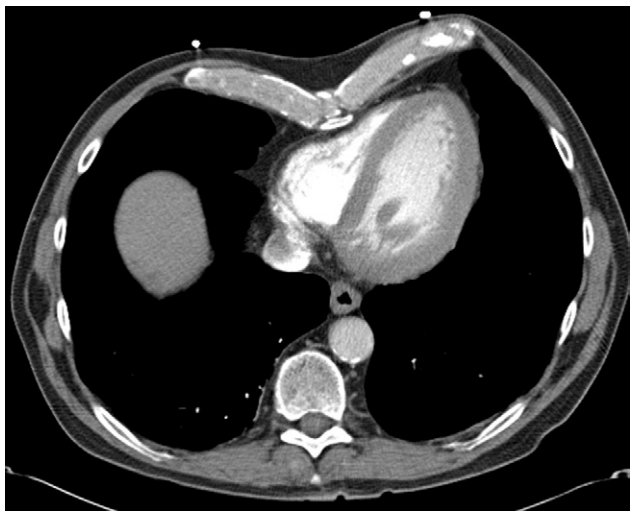

Fig 1. Computed tomographic scan showing pectus excavatum deformity impinging on the right ventricle of the heart.

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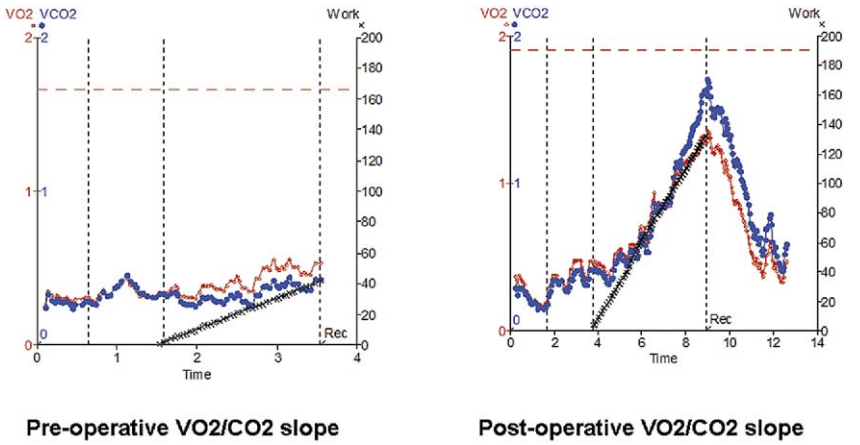

Fig 2. The VO2/CO2 slope of patient before surgical correction (left) and after surgical correction (right).

was admitted to the hospital for possible congestive heart failure.

Treatment was initiated with furosemide (20 mg), enalapril (5 mg), and carvedilol (3.125 mg) orally. Despite successful diuresis, he continued to have orthopnea. He became hypotensive requiring heart failure medications to be held. Cardiac catheterization showed normal coronary arteries. Resting hemodynamics were normal with central venous pressure of 7 mm Hg, pulmonary capillary wedge pressure mean of 10 mm Hg, and cardiac output of 6.1 L/min. Pulmonary function testing including diffusion capacity were within normal limits.

A cardiopulmonary exercise stress test was performed, and his impairment was significant in appearance due to the inability to augment stroke volume at peak exercise. There was no indication of respiratory or ventilatory impairment. After 1 month of medical therapy with follow-up in the heart failure clinic, the patient failed to tolerate increases in his cardiac medications. He continued to have a New York Heart Association functional class of 3 to 4, with frequent complaints of orthopnea. On computed tomographic scan, the PE could be seen impinging on the right ventricle (Fig 1).

We hypothesized the PE was compressing the dilated right ventricle exacerbating symptoms of heart failure. The patient subsequently underwent open surgical repair of the PE as previously described [3]. Postoperatively he did well and was discharged on day 4.

At his 2-year follow-up, he is on carvediol (18.75 mg twice daily), valsartan (80 mg daily), and no diuretics. An echocardiogram showed that his ejection fraction increased to 34% with normal right ventricular size and function. He has improved to New York Heart Association functional class 2. Postoperative cardiopulmonary exercise stress test curves exemplified his improvements (Figs 2 and 3).

Comment

The understanding of PE is evolving and must take into account both the size of the physical defect and its impact on internal structures. In some patients the cardiopulmonary effects of deformities may not manifest until the patient ages. Reported effects include sternal compression decreasing thoracic volume and reducing the SVO_2, exercise tolerance, tidal volume, and vital capacity, causing dyspnea and a decrease in

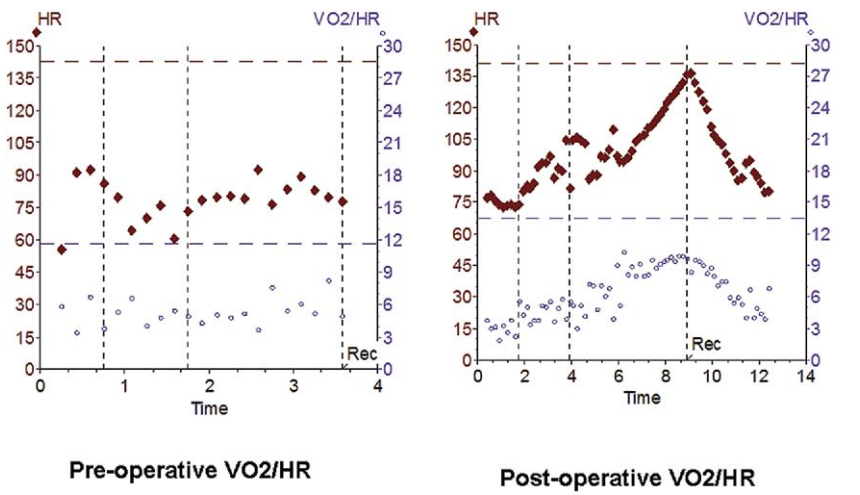

Fig 3. The VO2/heart rate (HR) slope before surgical correction (left) and after surgical correction (right).

endurance during exercise. Cardiac compression can reduce stroke volume and cardiac output in severe deformities, causing accelerated fatigue and compensatory tachycardia [4–7].

Documentation of objective physiologic improvements after surgical correction has been limited, and available studies are controversial for objective improvement in exercise tolerance [4–8]. Many studies suffer from methodological and technical concerns that limit their conclusions. Most are small case series of children and teens without statistical power for detecting meaningful results. A meta-analysis of studies representing 169 published reports encompassing 313 PE patients indicated that surgical repair improved cardiac abnormalities, but did not consistently improve pulmonary abnormalities [8].

In this patient, rapid deterioration of cardiac function was seen in a 3-year period. No cause of his dilated cardiomyopathy was identified; however a PE deformity was suspected as a contributing factor to his symptoms being out of proportion to his resting hemodynamics and inability to tolerate standard heart failure therapy. The patient exhibited significant improvement of symptoms and cardiac function with a combined approach of both surgical repair of the PE and continued medical treatment.

In conclusion, this case report, along with current ongoing investigations, suggest that surgical repair of PE is indicated in symptomatic patients and is more than primarily cosmetic. When patients present with more then one possible cause, a comprehensive evaluation including exercise testing can assist diagnosis. Surgery can be performed safely with few complications and short hospitalization, even in patients with cardiac comorbidities.

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Video-Assisted Mediastinoscopic Drainage of a Bronchogenic Cyst Presenting With Cardiac Dysfunction

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Bronchogenic cysts originate from the anomalous development of the ventral foregut. The usual presentation of bronchogenic cyst in the mediastinum is related to cyst infection or tracheobronchial compression. We describe a case of bronchogenic cyst presenting with cardiac dysfunction and hemodynamic compromise in a 23-year-old man with chest pain and progressive dyspnea. A high body mass index and respiratory dysfunction increased the risk of open surgery. Therefore, video-assisted cervical mediastinoscopy was performed and de-roofing of the cyst achieved resolution of his cardiovascular dysfunction and symptoms.

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Bronchogenic cysts originate from the anomalous development of the ventral foregut. The usual presentation of bronchogenic cyst in the mediastinum is related to cyst infection or tracheobronchial compression. Subcarinal cysts may cause compression of the airway and very rarely present with compression of the posterior wall of the left atrium, reducing left ventricular pre-load and hemodynamic compromise. We describe a case of bronchogenic cyst presenting with cardiac dysfunction and hemodynamic compromise.

A 23-year-old man presented as an emergency with gradual onset of central chest discomfort and dyspnea, which was worst when prone. His body mass index was 32, and his arterial blood gas analysis showed a degree of type 2 respiratory failure (Pao_2 = 7.8 kPa, $PaCo_2$ = 6.4 kPa). A computed tomographic pulmonary angiogram was performed to exclude a pulmonary embolus and reconstructed three-dimensional computed tomographic images revealed a 78 × 63 mm, well-defined subcarinal cystic mass with peripheral calcification, causing significant compression of the posterior wall of the left atrium and the carina (Fig 1). A transthoracic echocardiogram revealed large global pericardial effusion, with 3 cm maximum rim of fluid around the right ventricle, collapsed right atrium, and significantly reduced stroke volume. The patient underwent echocardiographic-

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