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# Letters to the Editor

## Invited letter concerning: Clinical and left ventricular function outcomes up to five years after dynamic cardiomyoplasty

*To the Editor:*

The evolution of the clinical use of skeletal muscle assistance to support the failing heart has not conformed to an idealized scientific progression from observation to experimentation to patient application. Interestingly, the vast majority of experimental work has been performed to devise auxiliary ventricles that function as diastolic counterpulsators. Yet, this mode of skeletal muscle assistance has not been brought to the clinical arena. The plethora of experimental studies on the efficacy of synchronizing skeletal muscle wrapped directly around the heart to contract during systole has mostly followed the initial clinical experience and seemed predicated more on technical feasibility rather than solid animal data demonstrating improved hemodynamics. Despite no definitive experimental study showing consistent and long-term improvement in cardiac function and few clinical reports of objective improvements in hemodynamics and patient longevity, dynamic cardiomyoplasty is applauded by many surgeons as an alternative therapy to cardiac transplantation for patients who either do not meet the stringent screening criteria or may not receive timely organ donation because of donor shortages.

In this context, the article by Dr. Moreira and associates[1] as reported in this issue is particularly enticing. Unfortunately, an objective evaluation of the presented data provides a much less sanguine assessment of the efficacy of this operation. First, the 5-year follow-up is anecdotal because only four of the patients were observed this long and statistical significance for improvement in left ventricular stroke work is lost after 36 months. In fact, improvement in global ejection phase indices of right and left ventricular function are lost at 24 and 12 months, respectively, after the operation. One may also question the physiologic significance of an increase in left ventricular ejection fraction from 19% to 23% or 24% as measured by radioisotope scintigraphy. Alternatively, dynamic cardiomyoplasty may be beneficial by preventing progression of ventricular dilatation as opposed to improving ejection fraction. However, the authors do not provide the end-diastolic volumes at which maintenance of stroke volume occurs to adjudicate this issue. This would have important implications in regard to ventricular wall stress and arrhythmogenesis.

The survival data are even less impressive. For the entire group, actuarial survival at 3 to 5 years is 42.5% ± 11.8%. No patient with New York Heart Association class IV symptoms survived for 3 years and half of these patients had died by one year. Patients in class III had only a 60% 3-year survival. Moreover, these patients had idiopathic dilated cardiomyopathy, and perhaps their prognosis is, in fact, better than that of patients who have ischemic cardiomyopathy. Is this truly an alternative for cardiac transplantation? Is this a viable alternative for medical therapy?

Although the authors mention that they had a nonmatched medically treated control group, I think it is imperative that an objective description of these patients with "medically refractory" heart failure be used to truly identify similar patient cohorts. The prognosis of patients eligible for cardiac transplantation is clearly affected by their degree of pulmonary hypertension, response to exercise, maximal oxygen consumption, and response to aggressive "tailored" medical therapy.[2] The decision to list a patient for transplantation (or perhaps to perform a cardiomyoplasty) is made during a period of relative clinical instability. Yet, the majority of patients improve and are discharged home to await organ donation. Interestingly, Stevenson and associates[3] have found that if a transplant candidate survives 6 months without an allograft, the subsequent 24 month survival is 80%. Granted, the attrition rate during the first 6 months is significant. Yet, most of these patients have class IV disease and would have a similarly poor outcome when subjected to cardiomyoplasty. The cohort with the best results in this series, that is, patients with class III symptoms, should also be stringently evaluated. Mancini and associates[4] measured peak exercise oxygen consumption in a large series of patients referred for cardiac transplantation. In a group who had class III symptoms but had a peak oxygen consumption of 19 ml/kg per minute, survival at 1 and 2 years with continued medical therapy was 94% and 84%, respectively. For the efficacy of cardiomyoplasty versus medical therapy to be adjudicated, the patients must be evaluated by more than functional class and ejection fraction.

I laud the intrepid surgical efforts of Dr. Moreira and colleagues. However, the fact that an operation is new and can be performed does not automatically confer benefit. I wish that one of the groups performing this procedure would find the courage or honesty to insert in the discussion that cardiomyoplasty may really have only limited or no value. If the patient is too sick, it does not work. If the patient is not too sick, perhaps "tailored" medical therapy is the best option. Surgeons should no longer hide behind

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the "better patient selection" excuse for defining the future role of skeletal muscle assistance.

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## Aortopulmonary paraganglioma, a rare tumor

*To the Editor:*

I read with interest the letter on aortopulmonary paraganglioma by Castañon and associates,[1] who referred also to our case of intrathoracic chemodectoma (or paraganglioma) with multiple localizations.[2] I would like to recall the Carney triad of extraadrenal paraganglioma, pulmonary chondroma, and gastrointestinal leiomyo(sarco)ma. After reviewing the clinical data and the available histologic data on our case, Carney included our case in a series of patients with the triad from the Mayo Clinic and from the literature.[3]

Our case concerned a female patient who underwent at the age of 31 years a resection of an anterosuperior mediastinal chemodectoma. An esophageal anomaly had been discovered at the age of 16 years, during an episode of vomiting, but because the patient was afterward asymptomatic, she refused any further investigation of the esophagus. Seventeen years later the patient had a second operation. An aortopulmonary chemodectoma was resected and a pulmonary osteochondroma of the lingula was enucleated. The esophagus was not explored according to the wish of the patient. Two years later a small tumor in the left upper lung lobe was visible and was considered to be a new or recurrent benign pulmonary hamartochondroma, but again 2 years later an inoperable squamous cell carcinoma of the left main bronchus was discovered and the patient died 3 months later at the age of 52 years. Postmortem examination was refused by the relatives, so that the histologic type of the esophageal tumor is still unknown. We believed that the long-lasting esophageal anomaly was a benign leiomyoma and considered that our patient had the Carney triad.

With regard to the case of aortopulmonary paraganglioma reported by Castañon's group,[1] I have some recommendations and some comments.

I recommend regular follow-up of the patient, exclusion of other localizations of the paraganglioma, and exclusion of the triad of Carney. There is no chronologic sequence in the appearance of these three tumors, and their occurrence is more than coincidence.[3] Moreover, malignancy of each part of the triad is possible.[2, 3]

The risk of lung cancer is increased in patients with chondromatous hamartoma, whether the hamartoma itself or the fibrous scar or whether both tumors have common risk factors.[4] The spatial association was present in our case, and the question arises whether the enucleation of a hamartoma should not be altered in a segmental resection. In our case a new small tumor appeared 2 years after the enucleation and was, without proof, considered to be benign, but another 2 years later lung cancer was discovered in the same area.

Finally, patients with pulmonary hamartoma have a high incidence of associated anomalies and benign tumors but also an increased susceptibility to development of malignant tumors. Therefore, the clinical significance of pulmonary hamartoma is in the workup and lifelong follow-up of those patients.[5]

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**12/8/55995**

## Anomalous origin of the left coronary artery from the pulmonary artery: New technique or modification of "old" technique

*To the Editor:*

We read with interest the article by Tashiro and associates,[1] "Anomalous Origin of the Left Coronary Artery