

## CASE REPORT

ADVANCED

## CLINICAL CASE

# Troubleshooting Total Artificial Heart

## Novel Use of Implantable Hemodynamic Monitor



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## ABSTRACT

Accurate device optimization of the Syncardia temporary total artificial heart is difficult while waiting for heart transplantation. In this challenging clinical cohort, using an implantable hemodynamic monitor (CardioMEMS HF system) can assist in volume and hemodynamic assessments. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:1024-8) © 2021 Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## HISTORY OF PRESENTATION

The patient was a 62-year-old man with ischemic cardiomyopathy and biventricular heart failure who underwent SynCardia 70cc temporary total artificial heart (TAH-t) (Tucson, Arizona) implantation as a bridge to transplant. He had a small chest cavity diameter (10.7 cm). Therapeutic anticoagulation was tolerated with no evidence of internal bleeding. While on the floor with a higher beat rate (130 beats/min), he had alarms for low fill volumes, prompting a reduction in beat rate to 115 beats/min. His diuretic was reduced. One week later, he developed hypoxic

respiratory failure from pulmonary edema and pleural effusions (**Figure 1A**). His vitals included blood pressure of 121/67 mm Hg, beat rate of 115 beats/min, respiratory rate of 18, and peripheral oxygen saturation of 95% on high-flow cannula (settings: 40 l and fraction of inspired oxygen 80%). His physical examination was notable for bilateral, coarse crackles, and normal TAH-t sounds. He did not require intubation. Although he had normal renal function, his respiratory status did not improve with diuresis.

## PAST MEDICAL HISTORY

His heart failure was complicated by World Health Organization group II pulmonary hypertension. The Abbott CardioMEM HF System (Atlanta, Georgia), an implantable hemodynamic monitor (IHM), was implanted 9 months before the total artificial heart (TAH). Remote tracking of the IHM did not reveal significant changes in hemodynamics before TAH implantation.

## LEARNING OBJECTIVES

- To evaluate the differential diagnosis of pulmonary edema in a patient with a TAH-t.
- To perform a hemodynamic ramp in a patient with a TAH-t.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

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## DIFFERENTIAL DIAGNOSIS

The differential diagnosis for pulmonary edema and effusions in a patient with a TAH include the following:

1. Inadequate unloading due to a low beat rate, which results in pulmonary congestion.
2. Anatomical or positional compression of the left atrium and pulmonary veins by the TAH, which leads to a physiology similar to pulmonary venous occlusive disease.
3. Intrinsic pulmonary parenchymal conditions, such as diffuse alveolar hemorrhage or atypical pneumonia.

## INVESTIGATIONS

Cardiac computed tomography (CT) angiography revealed patent pulmonary veins, normal left atrial size, and normal TAH components. In addition, he had no hemoptysis and no evidence of pneumonia on sputum cultures. We did not perform a bronchoscopy due to his tenuous respiratory status. To evaluate TAH parameters and mechanical abnormalities, we examined his hemodynamics via the IHM (Figure 2). By increasing the beat rate from 110 to 135 beats/min, there was a significant drop in pulmonary artery pressures (Figures 1B and 1C, Table 1). This improvement in pulmonary pressures correlated with a lower fill volume ranging from 35 to 45 ml. Supine and sitting positions did not alter hemodynamics.

## MANAGEMENT

Consequently, we increased the beat rate to 125 beats/min and continued intravenous diuresis.

## DISCUSSION

To our knowledge, we present the first case of using the IHM to assess hemodynamics and to optimize TAH parameters.

**TAH.** In patients with contraindications to a left ventricular assist device, the TAH offers an alternative to providing a mechanical circulatory support bridge to heart transplantation (1). The device consists of 2 mechanical ventricles sutured to the native atria, aorta, and pulmonary arteries. Because of TAH-t size, the chest diameter should be at least 10 cm (1). At the end of diastole, the device ejects the entire fill volume within the ventricles. That optimal volume is 50 to 60 ml but may be lower in patients with smaller stature because it is calculated using body surface area (2). Because the TAH-t ventricle is inelastic, left atrial

pressure would increase with high fill volumes akin to a restrictive cardiomyopathy physiology (2). In this setting, pulmonary pressures would rise, leading to vascular congestion. The fill volume can be controlled by optimizing intravascular volume and increasing the beat rate (i.e., heart rate equivalence) to lower the diastolic filling time.

Because of the polyurethane and metallic prostheses, it is impossible to optimize TAH parameters using invasive hemodynamics. The IHM has been shown to decrease rehospitalization in patients in New York Heart Association functional class III heart failure by remotely monitoring pulmonary hemodynamics (3). An IHM could provide wirelessly hemodynamic measurements in patients on TAH-t support.

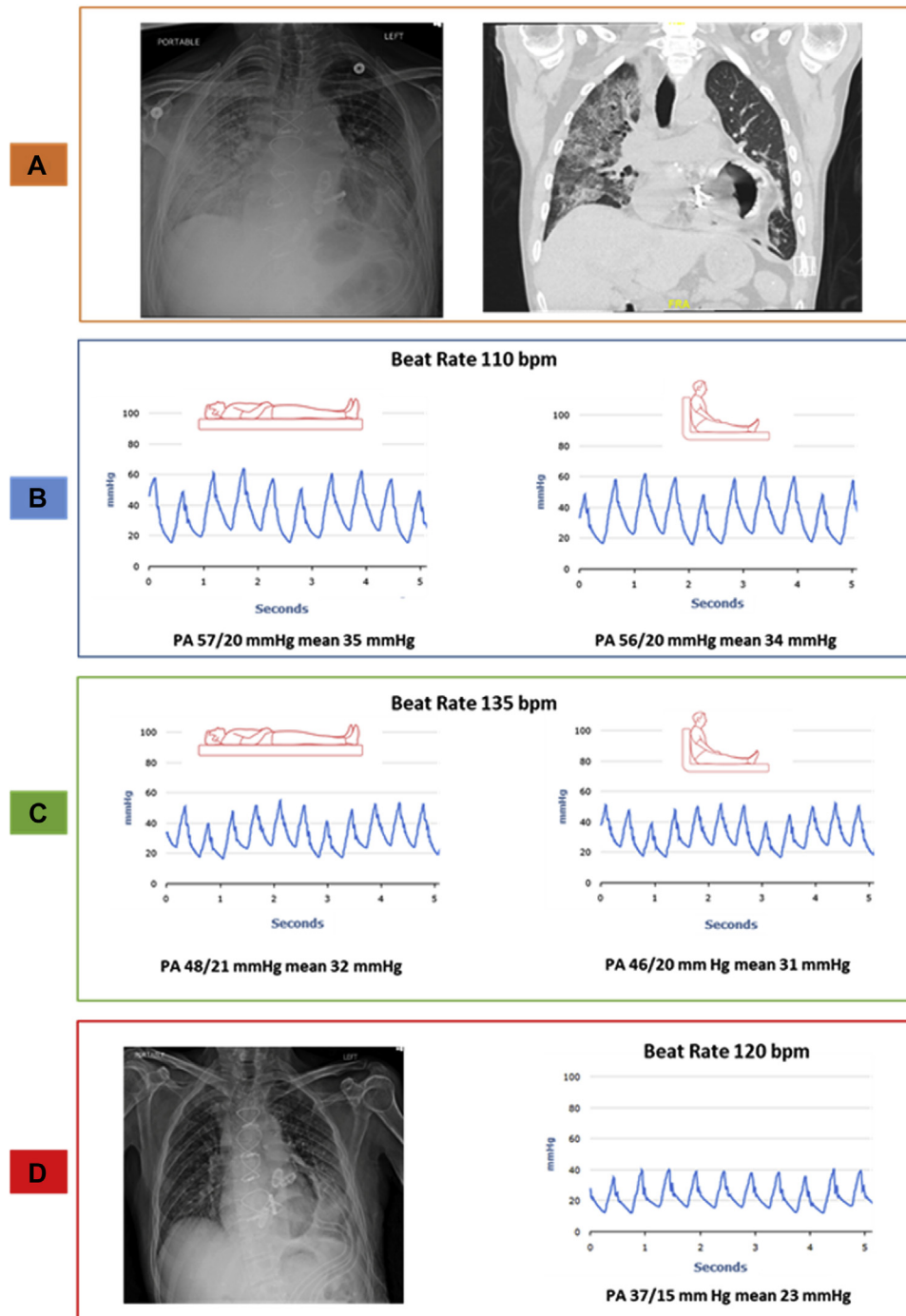
**POTENTIAL COMPLICATIONS OF TAH.** During TAH-t support, several complications may occur (1). In patients with a small chest cavity size, the device components could compress vascular vessels, including the inferior vena cava and pulmonary veins; in that case, there may be pulmonary congestion from a pulmonary venous occlusive disease pathophysiology (4). In this scenario, the device would be oriented in a nonstandard approach or potentially be anchored to the left ribs to displace the TAH laterally from the vasculature (4).

In our clinical case, the patient had unresolved pulmonary opacities. We conducted a cardiac CT angiogram, which ruled out abnormal TAH-t components, pulmonary veins, and left atrial compression. Thus, it was unlikely that the TAH-t was causing a pulmonary venous occlusive physiology. The clinical condition and imaging also did not suggest an infectious process or diffuse alveolar hemorrhage based on his examination and negative cultures. For these reasons, intrinsic pulmonary disease and anatomical abnormalities from the TAH device were unlikely.

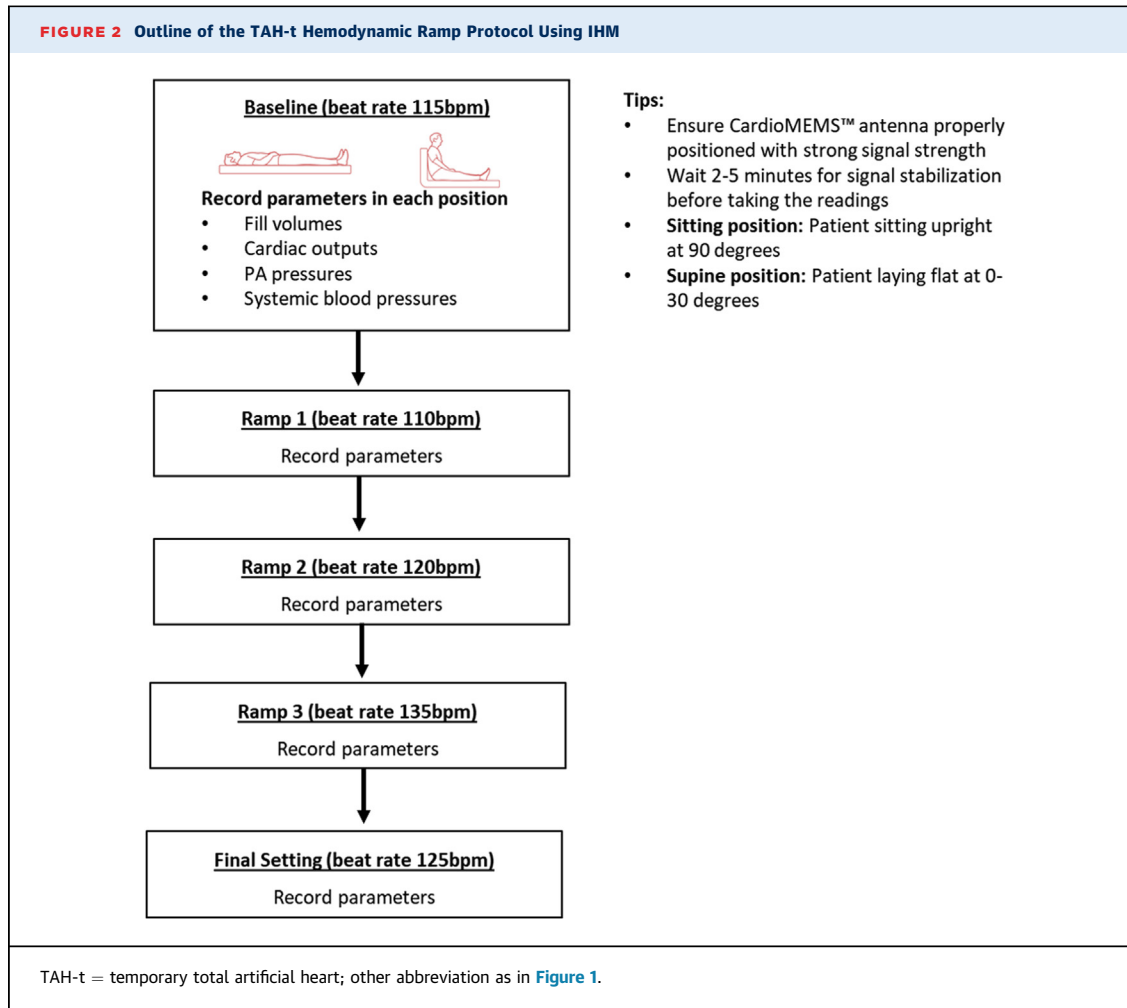
**IHM AND TAH HEMODYNAMIC ASSESSMENT.** We consequently believed that at a beat rate of 115 beats/min and fill volumes of 50 to 60 ml, the TAH-t might not have adequately unloaded his volume, resulting in pulmonary vascular congestion. To test this hypothesis, we took advantage of the IHM to interrogate the hemodynamics in this patient (Figure 2). The beat rate was ramped incrementally from 110 to 140 beats/min, and we observed appropriate decreases in his fill volumes. When the volumes were low (30 to 40 ml), his pulmonary pressures improved, suggesting the patient would need a higher beat rate to properly offload his volume in pulmonary vasculature. In addition, because of his small chest cavity size, we ramped his beat rate in both supine and sitting positions to

## ABBREVIATIONS AND ACRONYMS

- IHM** = implantable hemodynamic monitor  
**TAH** = temporary artificial heart  
**TAH-t** = Syncardia temporary total artificial heart

**FIGURE 1** Significant Pulmonary Edema Involving Both Lungs With Effusions on Chest X-Ray and CT at Baseline BR of 115 beats/min

(A) Hemodynamics via implantable hemodynamic monitor (IHM) interrogation at low (110 beat/min) and high (135 beats/min) beat rate (BR) (B and C) in supine and sitting positions showing that at a higher BR, there is a decrease in pulmonary pressures, suggesting adequate volume unloading. (D) There was marked improvement in airspace opacities on follow-up chest x-ray and implantable hemodynamic monitor readings after increasing BR and further diuresis. CT = computed tomography; PA = pulmonary artery.



examine whether the TAH components were positionally compressing his vessels. No changes in pulmonary pressures or fill volumes were observed between the supine and sitting positions at any given beat rate. The observations provided reassurance that the TAH-t components were working properly and did not exert a positional effect on the pulmonary vasculature. We left the device at 125 beats/min, corresponding to a fill volume of 40 to 45 ml.

Although many patients on TAH support may have concomitant pulmonary hypertension (1), current TAH technology does not allow for direct measurements of pulmonary artery pressures. Several case reports describe the IHM as a tool to monitor pulmonary pressures during TAH support to assess transplant candidacy (5,6). In these patients, the IHM was implanted safely before the TAH operation and could provide reliable hemodynamic data.

To our knowledge, this is the first case of using an IHM to interrogate hemodynamics in a patient on

**TABLE 1 Hemodynamic Data During TAH-t Ramp Study Using IHM**

BR (beats/min)/ Patient Position	Left FV (ml)	Left CO (l/min)	Right FV (ml)	Right CO (l/min)	PAP (mm Hg)	Mean PAP (mm Hg)	BP (mm Hg)	MAP (mm Hg)
115*								
Supine	53	6.1	45	5.2	59/23	35	112/51	71
110								
Supine	56	6.2	48	5.3	56/20	32	117/50	72
Sitting	57	6.3	50	5.5	57/20	32	115/51	72
135								
Supine	46	6.2	38	5.1	50/21	31	124/55	78
Sitting	44	5.9	35	4.7	48/21	30	122/56	78
140								
Supine	42	5.9	34	4.8	46/22	30	116/56	76
125†								
Supine	49	6.1	42	5.3	49/21	30	123/58	78
120‡								
Supine	48	5.4	40	4.6	37/15	22	110/68	82

\*Baseline beat rate (BR) setting of temporary total artificial heart (TAH-t). †Final beat rate setting of TAH-t. ‡Follow up interrogation 2 weeks after the hemodynamic ramp.

BP = blood pressures; CO = cardiac output; FV = fill volume; IHM = implantable hemodynamic monitor; MAP = mean systemic artery pressure; PAP = pulmonary artery pressures.

TAH support to optimize the parameters. There seems to be little interference between the 2 technologies, and they may be synergistic in providing optimal management. An IHM could also potentially be used to optimize patients on durable biventricular ventricular assist device. Future studies are needed to examine the usefulness of implanting an IHM before TAH surgery and using the data to guide device management.

### FOLLOW-UP

Follow-up chest x-ray showed noticeable improvement in airspace opacities (Figure 1D). His TAH-t setting was reduced to 120 beats/min due to low fill volumes (30 to 35 ml) and adequate pulmonary artery pressures (Figure 1D). His repeat IHM interrogation revealed that his pulmonary pressures continued to improve, and an oral diuretic regimen was maintained. The patient underwent transplantation 8 weeks after TAH-t implantation.

### CONCLUSIONS

This case highlights that hemodynamic interrogation using an IHM device was feasible and useful in optimizing TAH parameters and patient clinical status while awaiting heart transplantation.

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**KEY WORDS** CardioMEMS, heart failure, mechanical circulatory support, pulmonary artery hypertension, total artificial heart