Policy Number	**SUR707.031**
Policy Effective Date	**06/15/2025**
Policy End Date	**12/31/2025**

Pulmonary Artery Denervation

Table of Contents

Related Policies (if applicable)
RX501.056: Advanced Therapies for Pharmacologic Treatment of Pulmonary Hypertension

Disclaimer

Carefully check state regulations and/or the member contract.
Each benefit plan, summary plan description or contract defines which services are covered, which services are excluded, and which services are subject to dollar caps or other limitations, conditions or exclusions. Members and their providers have the responsibility for consulting the member's benefit plan, summary plan description or contract to determine if there are any exclusions or other benefit limitations applicable to this service or supply. **If there is a discrepancy between a Medical Policy and a member's benefit plan, summary plan description or contract, the benefit plan, summary plan description or contract will govern.**

Coverage

This medical policy has become inactive as of the end date above. There is no current active version and this policy is not to be used for current claims adjudication or business purposes.

Pulmonary artery denervation by any method including but not limited to radiofrequency ablation, electrical high-frequency stimulation, remote magnetic navigation, or therapeutic intravascular ultrasound (TIVUS™) **is considered experimental, investigational and/or unproven** for all indications, including but not limited to, treatment of pulmonary arterial hypertension.

Policy Guidelines

None.

Description

Pulmonary Hypertension

Pulmonary hypertension (PH) is characterized by elevated pulmonary artery pressure ≥20 mmHg at rest (mean pulmonary artery pressure). The World Health Organization (WHO) classifies individuals with PH into five groups based upon etiology:

- Group 1 – Pulmonary arterial hypertension (PAH);
- Group 2 – PH due to left heart disease;
- Group 3 – PH due to chronic lung disease and/or hypoxemia;
- Group 4 – PH due to pulmonary artery obstructions;
- Group 5 – PH due to unclear multifactorial mechanisms. (1)

The term PAH is used to describe those included in group 1, while the term PH is used when describing all five groups. (1)

Pulmonary Artery Hypertension (PAH)

According to the National Organization for Rare Disorders (NORD), PAH is a rare, progressive disorder that is characterized by high blood pressure (hypertension) in the arteries of the lungs (pulmonary artery) for no apparent reason. (2) The pulmonary arteries carry blood from the right side of the heart through the lungs. Symptoms include shortness of breath, especially during exercise, chest pain, and fainting episodes. PAH is treatable; and while the exact cause is unknown, there is no known cure.

PAH impacts women between the ages of 30 and 60 more than men; and approximately 15-20% of individuals with PAH have heritable forms of the disease. Heritable PAH results from either an autosomal dominant genetic condition; or members of a family in which PAH is known to occur as primary disease. (1, 2)

Diagnosis of PAH can be difficult as the symptoms are not unique and can be confused with other diseases that cause a lack of oxygen in the blood. Common tests used for diagnosis can include echocardiography, blood tests, pulmonary function tests (PFTs), X-rays of the chest, lung blood flow scans, electrocardiography (ECG) and the 6-minute walk test to determine how far an individual can walk in that time period. Heritable PAH is confirmed if two or more family members have PAH or if a bone morphogenetic protein receptor 2 (*BMPR2)* gene mutation or mutation in another gene known to cause PAH is identified in the affected individual. (1, 2)

Although there is no cure for PAH, there are medications and procedures that can slow the progression of the disease and improve quality of life. Treatment aims to restore balance among one or more of three substances that are produced by the lungs: nitric oxide, endothelin, and prostacyclin. Although a test does not currently exist to determine which of these substances is not balanced, PAH medications act on these three pathways to help slow disease progression. (3) In severe cases of PAH, a heart-lung, single lung, or double-lung transplant may be recommended. These treatments are not discussed in this medical policy document.

Pulmonary Artery Denervation

Pulmonary artery denervation (PAD or PADN) is being investigated as a potential treatment for individuals with PAH who are unresponsive to medication. This procedure involves denervation of the sympathetic nerves of the pulmonary vasculature using radiofrequency ablation. During right heart catheterization, pulmonary angiography is used to define the main, right and left pulmonary arteries for ablation catheter placement. An ablation catheter is inserted and pushed along the transseptal guiding introducer sheath into the main pulmonary artery and positioned at the ostium of the left pulmonary artery (Level 1 of ablation). The ablation targets are selected, and after ablation at this level, the circular tip is moved down to the distal bifurcation area of the main pulmonary artery (Level 2 of ablation). Next the circular tip is moved distal to the ostial right pulmonary artery (Level 3 of ablation). Hemodynamic measurements and blood oxygen pressure/saturation determinations from the right atrium, right ventricle, and pulmonary artery are performed after the ablations. (4)

Therapeutic Intravascular Ultrasound (TIVUS)
Therapeutic intravascular ultrasound (TIVUS) is an investigational intravascular treatment that can be performed as part of a right heart catheterization for patients suffering from pulmonary arterial hypertension (PAH) according to the manufacturer website. (5) A catheter-based technology, it causes denervation of nerves surrounding blood vessels and other structures like the bronchus. Per information on the manufacturer website, "the catheter generates non-focused ultrasound waves that pass through the flowing blood and through the wall of the artery where the energy is taken up by the tissue outside of the blood vessel, specifically the nerve bundles. It results in the nerves being heated to the point of causing necrosis and the nerves effectively become ablated, losing their ability to pass signals. This results in a decrease in sympathetic hormones that are released from the nerves. This reduction in hormones results in the blood vessels relaxing, reducing the resistance in the vessels, reducing the pressure in the vessels and in pulmonary hypertension, reduces the resistance in the right side of the heart, potentially leading to improvements in exercise tolerance."

Rationale

In 2013 Chen et al. reported on a single-center, prospective, first-in-man study designed to test the safety and efficacy of pulmonary artery denervation (PADN) for patients with idiopathic pulmonary arterial hypertension (IPAH) not responding to medical therapy. (4) Of 21 patients with IPAH, 13 patients underwent the procedure, and the other 8 who refused the procedure, were assigned to the control group. PADN was performed at the bifurcation of the main pulmonary artery (PA), and at the ostial right and left PA. Serial echocardiography, right heart catheterization and a 6-minute walk test (6MWT) were performed. The primary endpoints were the change of PA pressure (PAP), tricuspid excursion index (Tei), and 6MWT at 3 months follow-up. Compared with the control group, at 3 months follow-up, the patients who underwent the PADN procedure showed significant reduction of mean PAP (from 55± 5 mm Hg [millimeters mercury] to 36 ± 5 mm Hg, $p<0.01$), and significant improvement of the 6MWT (from 324 ± 21 m [meters] to 491 ± 38 m, $p<0.006$) and of the Tei index (from 0.7 ± 0.04 to 0.50 ± 0.04, $p<0.001$). The authors noted that given the small numbers in this study, potential bias in patient

selection, and the fact this was not a placebo-controlled double-blind study, the results are promising. Only carefully designed trials with controls and appropriate blinding must be done in a much larger cohort and at multiple centers before definitive conclusions about efficacy can be determined.

A 2015 study to analyze the hemodynamic, functional, and clinical responses to PADN in patients with PAH of different causes was published by Chen et al. (6) Between April 2012 and April 2014, 66 consecutive patients with a resting mean pulmonary arterial pressure ≥25 mm Hg treated with PADN were prospectively followed up. Target drugs (endothelial-receptor antagonists, phosphodiesterase type-5 inhibitors, and prostacyclin analogs) were discontinued after the PADN procedure. Hemodynamic response and 6-minute walk distance were repeatedly measured within the 1-year post PADN follow-up. The clinical end point was the occurrence of PAH-related events at the 1-year follow-up. There were no PADN-related complications. Hemodynamic success (defined as the reduction in mean pulmonary arterial pressure by a minimal 10% post PADN) was achieved in 94% of all patients, with a mean absolute reduction in systolic pulmonary arterial pressure and mean pulmonary arterial pressure within 24 hours of −10 mm Hg and −7 mm Hg, respectively. The average increment in 6-minute walk distance after PADN was 94 m. Worse PAH-related events occurred in 10 patients (15%), mostly driven by the worsening of PAH (12%). There were 8 (12%) all-cause deaths, with 6 (9%) PAH-related deaths. This study was a nonrandomized and open-label study that may have a possible bias; the study also lacked a control group. The small number of patients precluded detailed analysis of whether there were differential responses to PADN based on the PAH mechanism. All PADN procedures were performed by one surgeon. Multicenter randomized trials are warranted to determine the use of PADN in patients with PAH.

In 2019 Zhang et al. assessed the benefits of PADN among combined pre- and post-capillary pulmonary hypertension (CpcPH) patients in a prospective, randomized, sham-controlled trial. (7) Ninety-eight CpcPH patients, defined as mean pulmonary arterial pressure ≥25 mm Hg, pulmonary capillary wedge pressure >15 mm Hg, and pulmonary vascular resistance (PVR) >3.0 Wood units (WU), were randomly assigned to PADN or sildenafil plus sham PADN. Standard medical therapy for heart failure was administered to all patients in both groups. The primary endpoint was the increase in the 6-minute walk distance at the 6-month follow-up. The secondary endpoint was change in PVR. The main safety endpoint was occurrence of pulmonary embolism. At 6 months, the mean increases in the 6-min walk distance were 83 m in the PADN group and 15 m in the sildenafil group (least square mean difference 66 m, 95% confidence interval: 38.2 to 98.8 m; p<0.001). PADN treatment was associated with a significantly lower PVR than in the sildenafil group (4.2 ± 1.5 WU vs. 6.1 ± 2.9 WU; p=0.001). Clinical worsening was less frequent in the PADN group compared to the sildenafil group (16.7% vs 40%; p=0.014). At the end of the study, there were 7 all-cause deaths and 2 cases of pulmonary embolism. At the 6-month follow-up clinical worsening was reported in 28 patients, 20 (40%) in the sildenafil group and 8 (16.7%) in the PADN group (hazard ratio [HR]: 2.690, 95% confidence interval [CI]: 1.184 to 6.113; p=0.018), primarily driven by the protocol defined worsening of heart failure (HF) (36.0% vs. 14.6%, HR: 2.743, 95% CI: 1.145 to 6.571; p=0.024).

Additional randomized clinical trials are needed to compare long-term improvements in clinical outcomes in this patient population. The authors noted several limitations in their study. 1) They selected to use sildenafil plus standard medical therapy and sham PADN in the control group, even though neither sildenafil nor other PAH therapies are recommended in patients with CpcPH according to international guidelines. 2) The use of sildenafil without a comparable placebo in the PADN group may have affected the blinding of patients and treating physicians to the treatment allocation. 3) The 6MWD but not the clinical endpoint, was the primary endpoint in this study; 6MWD measurements may be different from day to day. 4) Patients were screened only after being treated medically for 3 days and adequate diuresis was not objectively measured, which may not have been adequate to stabilize and optimally treat their heart failure. 5) International normalized ratio values below 2 in anticoagulated patients would be considered subtherapeutic. 6) They did not investigate the underlying mechanisms that contributed to the improvement of cardiac function by PADN; but feel their results imply there is a beneficial effect of PADN on pulmonary arterial compliance. 7) Both patients with heart failure with reduced ejection fraction (HFrEF) and those with heart failure with preserved ejection fraction (HFpEF) were included. They felt it is important to further study the mechanisms of PADN treatment focusing on HFpEF.

A pilot feasibility study by Goncharova et al. in 2020 wanted to assess the feasibility of selective radiofrequency pulmonary artery (PA) ablation based on response to high-frequency stimulation mapping. (8) This study included three female patients with idiopathic PAH (IPAH). The following reactions to PA stimulation were noted and marked by color points on the three-dimensional map: sinus bradycardia (heart rate decrease ≥15%), tachycardia (heart rate increase ≥15%), phrenic nerve capture, and cough. Since the most appropriate ablation strategy was unknown, two approaches were suggested, according to stimulation results: ablation at points with any heart rate response (either bradycardia or tachycardia)—this approach was applied in patient #1 (IPAH long-term responder to calcium channel blockers); segmental ablation at points with no response and with tachycardia response (one IPAH long-term responder to calcium channel blockers patient and one–IPAH with negative vasoreactive testing). Hemodynamic measurements were performed before and after denervation. Follow-up visits were scheduled at 6 and 12 months. Six-months follow-up was uneventful for patients #1 and 3; patient #2 had one syncope and reduced 6-minute walk test distance and peak oxygen uptake (VO_2) consumption. At 12 months, there was a normalization of mean PA pressure and pulmonary vascular resistance (PVR) in patient #1. Patient #2 had no change in PA pressure and PVR at 12 months. Patient #3 remained in II functional class; however, there was an increase in mean PA pressure and loss of vasoreactivity. Electrical high-frequency stimulation of the PA identifies several types of evoked reactions: heart rate slowing, acceleration, phrenic nerve capture, and cough. The improvement in clinical and hemodynamic parameters following targeted PA ablation in the IPAH patient with positive vasoreactive testing should be confirmed in larger studies.

Results of a prospective, pilot, randomized, sham-controlled trial to test the efficacy and assess the safety of PADN using remote magnetic navigation (RMN) system in patients with residual chronic thromboembolic pulmonary hypertension (CTEPH) after surgical pulmonary

endarterectomy (PEA) was published by Romanov et al. in 2020. (9) Fifty patients with residual CTEPH despite medical therapy at least 6 months after PEA, who had mean pulmonary artery pressure ≥25 mm Hg or pulmonary vascular resistance (PVR) >400 dyn·s·cm^{-5} based on right heart catheterization were randomized to treatment with PADN (PADN group, n=25) using remote magnetic navigation for ablation or medical therapy with riociguat (MED group, n=25). In the MED group, a sham procedure with mapping but no ablation was performed. The primary endpoint was PVR at 12 months after randomization. Key secondary endpoint included the 6-min walk test (6MWT). After the PADN procedure, 2 patients (1 in each group) developed groin hematoma that resolved without any consequence. At 12 months, mean PVR reduction was 258 ± 135 dyn·s·cm^{-5} in the PADN group versus 149 ± 73 dyn·s·cm^{-5} in the MED group, mean between group difference was 109 dyn·s·cm^{-5} (95% confidence interval; 45 to 171; p=0.001). The 6MWT distance was significantly increased in the PADN group as compared to the distance in the MED group (470 ± 84 m vs. 399 ± 116 m respectively; p=0.03).

Rothman et al. published the results of an early feasibility study on TIVUS in 2020. (10) The aim of this study was to investigate whether therapeutic intravascular ultrasound pulmonary artery denervation (PDN) is safe and reduces pulmonary vascular resistance (PVR) in patients with PAH on a minimum of dual oral therapy. TROPHY1 (Treatment of Pulmonary Hypertension 1) was a multicenter, international, open-label trial undertaken at 8 specialist centers. Patients 18 to 75 years of age with PAH were eligible if taking dual oral or triple non-parenteral therapy and not responsive to acute vasodilator testing. Eligible patients underwent PDN (TIVUS System). The primary safety endpoint was procedure-related adverse events at 30 days. Secondary endpoints included procedure-related adverse events, disease worsening and death to 12 months, and efficacy endpoints that included change in pulmonary hemodynamic status, 6-min walk distance, and quality of life from baseline to 4 or 6 months. Patients were to remain on disease-specific medication for the duration of the study. Twenty-three patients underwent PDN, with no procedure-related serious adverse events reported. Pulmonary vascular resistance (PVR) was reduced 17.8% at 4- or 6-month follow-up which was associated with a 42-minute increase in 6-min walk distance as well as an increase in daily activity. In this multicenter early feasibility study, PDN with an intravascular ultrasound catheter was performed without procedure-related adverse events and was associated with a reduction in PVR and increases in 6-min walk distance and daily activity in patients with PAH on background dual or triple therapy. The authors concluded further studies are required to evaluate the efficacy, durability, safety, and long-term clinical impact of PDN in patients with pulmonary hypertension of various forms.

Zuo et al. (2022) conducted a systematic meta-analysis of the effectiveness of PADN in the treatment of PH patients. (11) Outcomes were mean pulmonary artery pressure (mPAP), PVR, cardiac output (CO), right ventricular (RV) Tei index, 6-minute walk distance (6MWD), and New York Heart Association (NYHA) cardiac function grading. A total of eight clinical studies with 213 PH patients who underwent PADN were included. Meta-analysis showed that after PADN, mPAP (mean difference [MD] -12.51, 95% CI -17.74 to -7.27, P<0.00001) (mmHg) and PVR (-5.17, 95% CI -7.70 to -2.65, P<0.0001) (Wood unit) decreased significantly, CO (0.59, 95% CI 0.32 to 0.86, P<0.0001) (L/min) and 6MWD (107.75, 95% CI 65.64 to 149.86, P<0.00001) (meter) increased significantly, and RV Tei index (-0.05, 95% CI -0.28 to 0.17, P=0.63) did not

change significantly. Also after PADN, the proportion of NYHA cardiac function grading (risk ratio 0.23, 95% CI 0.14 to 0.37, P<0.00001) III and IV decreased significantly. Reviewers concluded that while this meta-analysis supports PADN as a potential new treatment for PH, further high-quality randomized controlled studies are needed.

In a multicenter, sham-controlled, single-blind, randomized PADN-CFDA trial, Zhang et al. (2022) investigated the treatment effects of PADN in patients with group 1 PAH. (12) Patients were assigned to receive PADN plus a phosphodiesterase-5 inhibitor or a sham procedure plus a phosphodiesterase-5 inhibitor. The primary endpoint was the between-group difference in the change in 6MWD from baseline to 6 months. Among 128 randomized patients, those treated with PADN compared with sham had a greater improvement in 6MWD from baseline to 6 months (mean adjusted between-group difference 33.8 m; 95% CI: 16.7-50.9 m; *P* < 0.001). From baseline to 6 months, pulmonary vascular resistance was reduced by −3.0 ± 0.3 WU after PADN and −1.9 ± 0.3 WU after sham (adjusted difference −1.4; 95% CI: −2.6 to −0.2). PADN also improved right ventricular function, reduced tricuspid regurgitation, and decreased N-terminal pro–brain natriuretic peptide. Clinical worsening was less (1.6% vs 13.8%; OR: 0.11; 95% CI: 0.01-0.87), and a satisfactory clinical response was greater (57.1% vs 32.3%; OR: 2.79; 95% CI: 1.37-5.82) with PADN treatment during 6-month follow-up. Researchers concluded that treatment with PADN plus a PDE-5 inhibitor was safe and resulted in improved exercise capacity at 6 months compared with background therapy of PDE-5 inhibitor treatment alone. In addition, treatment with PADN reduced PVR and PAP, improved right ventricular function, reduced tricuspid regurgitation and NT-proBNP level, and improved clinical outcomes during 6-month follow-up. However, these results should be replicated in larger studies (especially in patients with a suboptimal response to combination pharmacotherapy) before widespread application of this novel approach.

The differential treatment effect of pulmonary artery denervation (PADN) in pulmonary arterial hypertension (PAH) patients with different risk burdens remains unclear. A 2023 study from Zhang et al., aimed to determine the effectiveness of PADN in low versus intermediate-high-risk PAH patients. (13) In total, 128 patients with treatment naive PAH included in the 2022 PADN-CFDA trial were categorized into low-risk and intermediate-high-risk patients. The primary endpoint was the between-group difference in the change in 6MWD from baseline to 6 months. In the intermediate-high-risk group, those treated with PADN and PDE-5i had a greater improvement in 6MWD from baseline to 6 months as compared to those treated with sham plus PDE-5i. From baseline to 6 months, pulmonary vascular resistance (PVR) was reduced by -6.1 ± 0.6 and -2.0 ± 0.7 Wood units following PADN plus PDE-5i and sham plus PDE-5i, respectively, along with the significant reduction of NT-proBNP in the intermediate-high-risk group. However, there were no significant differences in 6 MWD, PVR, and NT-proBNP between the PADN plus PDE-5i and sham plus PDE-5i groups among low-risk patients. Moreover, the right ventricular function was equally improved by PADN treatment across the low-, intermediate-, and high-risk groups. Clinical worsening was less with PADN plus PDE-5i treatment during the 6-month follow-up.

In 2023, Kan et al. reported on one-year clinical follow-up in all 128 patients from the 2022 PADN-CFDA trial. At 1 year, clinical worsening had occurred in 3 (4.8%) patients in the PADN plus PDE-5i group and in 15 patients (23.1%) in the sham plus PDE-5i group (adjusted hazard ratio: 0.17; 95% confidence interval [CI]: 0.05-0.60; p=0.006), driven by significantly increased rates of PAH-related hospitalizations, worsening functional class and the requirement for additional PAH treatments in the sham group. Results were consistent in high-risk, intermediate-risk and low-risk patients ($p_{interaction}$=0.186). Patients treated with PADN plus PDE-5i had an improvement in the between-group change in the 6MWD from baseline to 1 year of 81.2 m (95% CI: 50.3-112.2; p<0.001) compared with PDE-5i treatment alone. (14)

In 2023, Salazar et al. conducted a systematic review and meta-analysis looking at PADN as a new therapeutic option for PH. (15) Information was analyzed for the following outcomes: 6MWD, mPAP, PVR, and CO. Subgroup analysis comparing pre and post PADN in different PH groups was done. This meta-analysis included 6 controlled trials and 6 single-arm prospective studies with a total of 616 patients. The pooled analysis showed a significant reduction in mPAP [WMD -6.51, 95% CI (-9.87, -3.15), p = 0.0001], PVR [WMD -3.69, 95% CI (-6.74, -0.64), p = 0.02] and increased CO [WMD -0.37, 95% CI (0.08, 0.65), p = 0.01]. Subgroup analysis pre and post PADN demonstrated a significant improvement in 6MWD in the WHO group 1 [WMD 99.53, 95% CI (19.60, 179.47), p = 0.01], group 2 [WMD: 69.96, 95% CI (36.40, 103.51), p = < 0.0001] and group 4 [WMD: 99.54, 95% CI (21.80, 177.28), p = 0.01]. Reviewers concluded that this meta-analysis supports PADN as a therapeutic option for patients with PH, regardless of group class. However, further randomized trials are still needed to evaluate safety and efficacy.

Up To Date

In an article from UpToDate on the treatment of pulmonary hypertension, pulmonary artery denervation was listed as an investigational therapy. (16)

Summary of Evidence

For individuals with pulmonary hypertension refractory to medical therapies, the evidence includes single-center pilot and feasibility studies, a non-randomized open label study, prospective randomized sham-controlled trials, and systematic reviews and meta-analyses. All studies had small number of patients; some had no control groups. Primary and secondary endpoints included changes in pulmonary artery pressure, pulmonary vascular resistance and the 6-minute walk test. Controlled studies with larger number of patients and longer follow-up are needed to determine safety and efficacy of this procedure. Therefore, pulmonary artery denervation is considered experimental, investigational and/or unproven for all indications, including, but not limited to, the treatment of pulmonary artery hypertension.

Clinical Practice Guidelines and Position Statements

The European Society of Cardiology (ESC) and the European Respiratory Society (ERS)

The 2022 ESC/ERS Guidelines for the Diagnosis and Treatment of Pulmonary Hypertension (17) state in part: "..there is little evidence yet from multicentre RCTs [randomized controlled trials] demonstrating a benefit of PADN [pulmonary artery denervation] in patients already receiving

recommended medical therapy…Although potentially promising, PADN should be considered experimental."

Coding

Procedure codes on Medical Policy documents are included **only** as a general reference tool for each policy. **They may not be all-inclusive.**

The presence or absence of procedure, service, supply, or device codes in a Medical Policy document has no relevance for determination of benefit coverage for members or reimbursement for providers. **Only the written coverage position in a Medical Policy should be used for such determinations.**

Benefit coverage determinations based on written Medical Policy coverage positions must include review of the member's benefit contract or Summary Plan Description (SPD) for defined coverage vs. non-coverage, benefit exclusions, and benefit limitations such as dollar or duration caps.

CPT Codes	0632T, 0793T
HCPCS Codes	None

*Current Procedural Terminology (CPT®) ©2024 American Medical Association: Chicago, IL.

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Centers for Medicare and Medicaid Services (CMS)

The information contained in this section is for informational purposes only. HCSC makes no representation as to the accuracy of this information. It is not to be used for claims adjudication for HCSC Plans.

The Centers for Medicare and Medicaid Services (CMS) does not have a national Medicare coverage position. Coverage may be subject to local carrier discretion.

A national coverage position for Medicare may have been developed since this medical policy document was written. See Medicare's National Coverage at <https://www.cms.hhs.gov>.

Policy History/Revision	
Date	**Description of Change**
12/31/2025	Document became inactive.
06/15/2025	Reviewed. No changes.
04/01/2024	Document updated with literature review. Coverage unchanged. References 3 and 11-16 added; others updated.
01/15/2023	New medical document. Pulmonary artery denervation by any method including but not limited to radiofrequency ablation or therapeutic intravascular ultrasound (TIVUS™) is considered experimental, investigational and/or unproven for all indications, including but not limited to, treatment of pulmonary arterial hypertension. Coverage position for therapeutic intravascular ultrasound (TIVUS) was previously addressed on ADM1001.032 Experimental, Investigational and/or Unproven Procedures/Services.