Subject: Alcohol Ablation for Hypertrophic Obstructive Cardiomyopathy (HOCM)

Background: Alcohol ablation for hypertrophic obstructive cardiomyopathy (HOCM) induces an artificial myocardial infarction by insertion of pure alcohol into the most proximally situated septal branch of the left anterior descending coronary artery. The induced infarction leads to a decrease in the intraventricular pressure gradient, shrinkage of the hypertrophied septal bulge, and widening of the outflow tract.

The New York Heart Association (NYHA) determines Heart failure by the following classes:

- **Class I (Mild)**: No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).
- **Class II (Mild)**: Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
- **Class III (Moderate)**: Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
- **Class IV (Severe)**: Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.

Policy and Coverage Criteria:
Harvard Pilgrim Health Care (HPHC) considers alcohol ablation for hypertrophic obstructive cardiomyopathy (HOCM) as reasonable and medically necessary when ALL of the following criteria are met:

- Severe heart failure symptoms as reflected by the New York Heart Association (NYHA) class III or IV or other symptoms upon exertion (such as syncope or near syncope) non-responsive to drug therapy; AND
- Left ventricular outflow tract (LVOT) gradient greater than or equal to 50 mm Hg at rest or with physiological provocation, including but not limited to: exercise or medication administration; AND
- Absence of coronary artery disease that would impede performance of the procedure.

Exclusions:
Harvard Pilgrim Health Care (HPHC) considers alcohol ablation for hypertrophic obstructive cardiomyopathy as experimental/investigational for all other indications.

Supporting Information:
Percutaneous alcohol ablation of the septum does not require open chest surgery or cardiopulmonary bypass. Through a catheter-based approach, the ablation is performed using conventional interventional methodology currently available for treating atherosclerotic coronary artery disease. The procedure is also known as percutaneous transluminal septal myocardial ablation (PTSMA), transcoronary ablation of septal hypertrophy (TASH), and alcohol septal ablation (ASA). Necrosis of the anterior basal septum is caused by introducing absolute alcohol directly into a proximal septal perforator artery, producing a myocardial infarction within the proximal ventricular septum. This ultimately reduces LV wall thickness, enlarging the outflow tract and reducing mechanical impedance to left ventricular (LV) ejection. Although immediate rapid reduction of the resting outflow...
gradient may occur, more frequently a progressive decrease occurs after 6–12 months. Non-surgical ablation of the septum is potentially a less risky procedure with a shorter recovery time. However, it may increase the risk of dangerous abnormal heart rhythms. Some patients may require a pacemaker after the procedure (NICE, 2003).

A review of the literature shows alcohol ablation of the septum for HOCM to be safe and effective for select patients. Maron and Nishimura (2014) assessed the controversy of surgical septal myectomy versus alcohol septal ablation. The authors concluded that: "ASA is more advantageous for older patients, including those with comorbidities, although overall it is associated with a risk for sudden death or ventricular tachyarrhythmias that exceeds that of surgery. Therefore, the preponderance of evidence, expert consensus, and guideline opinion continues to favor surgical septal myectomy as the preferred treatment for most drug-refractory, severely symptomatic patients with obstructive HCM. ASA is a useful alternative option for selected patients.” This conclusion agrees with the ACC/European Society of Cardiology consensus document and ACC/AHA guidelines that “[s]urgical myectomy is the preferred (gold standard) and safest septal reduction procedure for most severely symptomatic obstructive HCM patients refractory to maximum medical management, and ASA is not regarded as the primary treatment option for such patients with a similar severity of functional limitation but rather as a useful, selective alternative to septal myectomy for patients of advanced age, patients at high operative risk as a result of important comorbidities, or those with a strong personal preference to avoid surgery. ASA can be performed only when the coronary artery anatomy is suitable and cannot be undertaken when there is a need for concomitant cardiac surgery (eg, coronary artery disease requiring bypass grafting; resection of a subaortic membrane; intrinsic mitral valve disease dictating repair or replacement).”

Sedehi et al. (2014) studied 52 patients who underwent ASA for symptomatic LVOT obstruction. ASA resulted in a significant improvement in functional class, resting gradient, and provoked gradient. Survival after ASA at 2 and 5 years was 97.8% and 94.7% and was comparable to that of the general population. The authors concluded that ASA is effective for symptom relief and LVOT gradient reduction and is associated with favorable survival.

Veselka et al. (2014) retrospectively evaluated 290 highly symptomatic patients aged ≤ 50 years with HOCM who underwent ASA. Survival free of all-cause mortality at 1, 5, and 10 years was 97%, 94%, and 94%, suggesting a low risk of all-cause death in long-term follow up. The annual mortality rate combined with the first appropriate implantable cardioverter-defibrillator discharge was 1.43%, suggesting a low risk at long-term follow up. Jensen et al. (2013) conducted an observational cohort study to assess survival, incidence of sudden cardiac death after ASA and effects of ASA on the traditional risk factors for sudden cardiac death. The ten year survival was 88% after ASA compared with 84% in a matched population. The ten year survival free of sudden cardiac death was 95%. A reduction in the prevalence of abnormal blood pressure response, syncope, non-sustained ventricular tachycardia, and maximal wall thickness ≥ 30 mm was found after ASA. There was a significant reduction in the proportion of patients at high risk. The authors concluded that clinically applied ASA is safe.

Alcohol ablation is an accepted treatment according to the most recent American College of Cardiology Foundation/American Heart Association (ACCF/AHA) guideline for the diagnosis and treatment of hypertrophic cardiomyopathy (Gersh et al. 2011).

**Coding:**

Codes are listed below for informational purposes only, and do not guarantee member coverage or provider reimbursement. The list may not be all-inclusive. Deleted codes and codes which are not effective at the time the service is rendered may not be eligible.

<table>
<thead>
<tr>
<th>CPT® Codes</th>
<th>Description</th>
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<tr>
<td>93583</td>
<td>Percutaneous transcatheter septal reduction therapy (eg, alcohol septal ablation) including temporary pacemaker insertion when performed</td>
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**References:**

HPHC Clinical Medical Policy

Alcohol Ablation for Hypertrophic Obstructive Cardiomyopathy (HOCM)

Summary of Changes:

<table>
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<th>Date</th>
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<tr>
<td>6/17</td>
<td>Added coding and references clarified criteria</td>
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Coverage described in this policy is standard under most HPHC plans. Specific benefits may vary by product and/or employer group. Please reference appropriate member materials (e.g., Benefit Handbook, Certificate of Coverage) for member-specific benefit information.
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