Select Health

Select Health Medical Policies

Cardiovascular Policies

Table of Contents

Select Health

MEDICAL POLICY

AUTOLOGOUS STEM CELL INFUSION FOR MYOCARDIAL INFARCTION

Policy # 394

Implementation Date:3/5/08

Review Dates: 2/19/09, 2/17/11, 2/16/12, 4/25/13, 2/20/14, 3/19/15, 2/11/16, 2/16/17, 2/15/18, 2/5/19, 2/11/20, 2/18/21, 5/5/22, 2/16/23, 2/15/24, 2/20/25

Revision Dates: 2/18/10

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

Acute myocardial infarction (AMI) is the death of heart muscle secondary to prolonged lack of oxygen due to blockage of the artery supplying the muscle. Approximately 1.5 million cases of AMI occur each year in the United States and cardiovascular disease is the leading cause of death in the U.S; approximately 500,000−700,000 deaths related to coronary artery disease (CAD) occur each year.

Dead heart muscle reduces the heart's ability to function properly which can result in result in congestive heart failure or other complications which impair an individual's ability to function and may ultimately lead to an early death.

Various therapies have been attempted to improve heart function in cases of ischemic heart disease. These include medical therapy, surgical excision of dead heart muscle, placement of left ventricular devices (LVD), and even heart transplantation or placement of a temporary artificial heart. Recently, stem cell transplantation has been tried to replace damaged heart cells with healthy viable cells. A relatively new concept of adult stem cell therapy predicts that stem cells can differentiate into cell types outside of their original lineage. Results from studies have suggested that blood line stem cells can change into heart muscle cells and vascular cells after transplantation into damaged heart tissue.

Many studies have been done investigating stem cell infusions for the replacement of damaged heart cells. Various methods of administrating this therapy have been used with cells being implanted directly into heart muscle externally through the chest wall, through the veins leading to the heart and via heart catheterization into the coronary. Additionally, various cell lines have been used for this treatment including bone marrow cells, undifferentiated muscle cells, and peripheral blood cells, further clouding the question of efficacy. The exact mechanism in which stem cell transfer can improve perfusion and contractile performance of the injured heart remains unknown, and the controversies surrounding the ability of these cells to undergo this change continues to exist. However, irrespective of the mechanism, there appears to be a general agreement that stem cell transplantation has some impact on heart function post-MI. Questions remain, though, as to whether there is a significant clinical impact on patients.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health does NOT cover autologous stem cell infusion for myocardial infarction due to the lack of evidence proving clinical efficacy; and there are many unanswered questions related to the optimal progenitor cells and methods to deliver stem cells. This meets the plan's definition of experimental/investigational.

Autologous Stem Cell Infusion for Mycardial Infarction, continued

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

A Medical Technology Assessment performed in March 2008 identified 37 articles, including many randomized controlled trials, with most controls receiving normal saline injections or standard therapy. Most of these were small sample studies reporting outcomes over 3−6 months, and there was substantial heterogeneity in criteria for sample selection and infusion method. Of these studies, 28 studies focused on stem cells derived from bone marrow, 7 utilized circulating progenitor cells, and 2 used cells from skeletal myoblasts. A variety of cardiac outcomes were measured including change in left ventricular ejection fraction, global ejection fraction, various blood volume measures contractility, infarct size, and oxygen intake.

Many of these studies suggest potential for stem-cell infusion to improve cardiac function. For example, the largest study to date, REPAIR-AMI, is a multi-site randomized controlled trial of 204 patients with acute myocardial infarction successfully re-perfused with stent implantation. Patients were infused with either bone-marrow-derived progenitor cells or placebo. At 4 months, global LVEF was significantly higher in the treatment group, having increased from a mean of 48.3 ± 9.2% to 53.8 ± 10.2%, compared with a change from 46.9 ± 10.4% to 49.9 ± 13.0%, in the placebo group. The treatment group also experienced greater improvement in regional contractility.

In contrast, the ASTAMI randomized controlled trial of 100 patients found no difference between the bone marrow and placebo groups at 6 months in LVEF, end-diastolic volume, or infarct size. Similarly, LVEF was no different between treated and placebo patients at 18 months in the BOOST study of 60 patients.

Far fewer studies measured important clinical outcomes such as mortality, additional cardiac events, and quality of life post-infusion. The REPAIR-AMI study reported fewer deaths, repeat myocardial infarctions, or revascularization procedures, in the treatment group compared with placebo at the one-year follow-up. Stem cell infusion resulted in improved exercise capacity at 6 months in the ASTAMI trial and at 12 months in a nonrandomized cohort of 20 patients. Treatment with stem-cells conferred no improvement in quality of life at 6 months in the ASTAMI trial. The TOPCARE-AMI study of 59 patients reported no additional cardiac events at 12 months in patients treated with circulating progenitor cells.

Overall, while these studies suggest that stem cell infusion confers statistical improvement in cardiac functioning in the short-term, additional longitudinal studies are needed to evaluate whether the treatment reliably improves quality of life and reduces mortality from future cardiac events. Moreover, standardized procedures are needed for extraction and infusion in addition to better data on the appropriate patient population for this procedure. Several clinical trials are ongoing, which should contribute important data towards addressing these issues.

A literature review in February 2010 identified a BCBS TEC Summary, concluding there was insufficient evidence to allow a conclusion on the benefits of progenitor cell therapy.

Autologous Stem Cell Infusion for Mycardial Infarction, continued

Billing/Coding Information

CPT CODES

Not Covered: Investigational/Experimental/Unproven for This Indication

33999 Unlisted procedure, cardiac surgery

HCPCS CODES

No specific codes identified

Key References

1. Adelaide Health Technology Assessment (AHTA) on behalf of National Horizon Scanning Unit (HealthPACT and MSAC). Autologous bone marrow cell transplantation for myocardial infarction. 2007. Available: http://www.horizonscanning.gov.au/internet/horizon/publishing.nsf/Content/19C3AB9EE16F6D96CA25715C00039A50/$File/HORIZON%20SCANNING%20REPORT-%20Autologous%20BMC%20for%20MI%20HF.pdf. Date Accessed: December 17, 2007.
2. Assmus B, Honold J, Schachinger V, et al. "Transcoronary transplantation of progenitor cells after myocardial infarction." N Engl J Med 355.12 (2006): 1222-32.
3. Assmus B, Schachinger V, Teupe C, et al. "Transplantation of Progenitor Cells and Regeneration Enhancement in Acute Myocardial Infarction (TOPCARE-AMI)." Circulation 106.24 (2002): 3009-17.
4. Assmus B, Walter DH, Lehmann R, et al. "Intracoronary infusion of progenitor cells is not associated with aggravated restenosis development or atherosclerotic disease progression in patients with acute myocardial infarction." Eur Heart J 27.24 (2006): 2989-95.
5. Bearzi C, Rota M, Hosoda T, et al. "Human cardiac stem cells." Proc Natl Acad Sci USA 104.35 (2007): 14068-73.
6. BCBS TEC Assessment. (2008). Autologous Progenitor Cell Therapy for the Treatment of Ischemic Heart Disease. BlueCross BlueShield Association. September. Available: http://www.bcbs.com/blueresources/tec/vols/23/23_04.pdf. Date Accessed: February 12, 2010.
7. Brehm M, Strauer BE. "Stem cell therapy in postinfarction chronic coronary heart disease." Nat Clin Pract Cardiovasc Med 3 Suppl 1 (2006): S101-4.
8. Britten MB, Abolmaali ND, Assmus B, et al. "Infarct remodeling after intracoronary progenitor cell treatment in patients with acute myocardial infarction (TOPCARE-AMI): mechanistic insights from serial contrast-enhanced magnetic resonance imaging." Circulation 108.18 (2003): 2212-8.
9. Chen S, Liu Z, Tian N, et al. "Intracoronary transplantation of autologous bone marrow mesenchymal stem cells for ischemic cardiomyopathy due to isolated chronic occluded left anterior descending artery." J Invasive Cardiol 18.11 (2006): 552-6.
10. Chen SL, Fang WW, Qian J, et al. "Improvement of cardiac function after transplantation of autologous bone marrow mesenchymal stem cells in patients with acute myocardial infarction." Chin Med J (Engl) 117.10 (2004): 1443-8.
11. Chen SL, Fang WW, Ye F, et al. "Effect on left ventricular function of intracoronary transplantation of autologous bone marrow mesenchymal stem cell in patients with acute myocardial infarction." Am J Cardiol 94.1 (2004): 92-5.
12. Dimmeler S, Zeiher AM, Schneider MD. "Unchain my heart: the scientific foundations of cardiac repair." J Clin Invest 115.3 (2005): 572-83.
13. Erbs S, Linke A, Adams V, et al. "Transplantation of blood-derived progenitor cells after recanalization of chronic coronary artery occlusion: first randomized and placebo-controlled study." Circ Res 97.8 (2005): 756-62.
14. Garas S, Zafari AM. Myocardial Infarction. 2006. EMedicine. Available: http://www.emedicine.com/med/TOPIC1567.HTM. Date Accessed: December 17, 2007.
15. Ge J, Li Y, Qian J, et al. "Efficacy of emergent transcatheter transplantation of stem cells for treatment of acute myocardial infarction (TCT-STAMI)." Heart 92.12 (2006): 1764-7.
16. Herreros J, Prosper F, Perez A, et al. "Autologous intramyocardial injection of cultured skeletal muscle-derived stem cells in patients with non-acute myocardial infarction." Eur Heart J 24.22 (2003): 2012-20.
17. Janssens S, Dubois C, Bogaert J, et al. "Autologous bone marrow-derived stem-cell transfer in patients with ST-segment elevation myocardial infarction: double-blind, randomised controlled trial." Lancet 367.9505 (2006): 113-21.
18. Jyotsna M, Vemuganti GK, Reddy P, Chandra KS. "Autologous bone marrow-derived progenitor cell myocardial delivery for recent myocardial infarction patients following early angioplasty: results from a pilot study." Cardiovasc Revasc Med 7.4 (2006): 217-21.
19. Katritsis DG, Sotiropoulou P, Giazitzoglou E, Karvouni E, Papamichail M. "Electrophysiological effects of intracoronary transplantation of autologous mesenchymal and endothelial progenitor cells." Europace 9.3 (2007): 167-71.
20. Li ZQ, Zhang M, Jing YZ, et al. "The clinical study of autologous peripheral blood stem cell transplantation by intracoronary infusion in patients with acute myocardial infarction (AMI)." Int J Cardiol 115.1 (2007): 52-6.
21. Lunde K, Solheim S, Aakhus S, Arnesen H, Abdelnoor M, Forfang K. "Autologous stem cell transplantation in acute myocardial infarction: The ASTAMI randomized controlled trial. Intracoronary transplantation of autologous mononuclear bone marrow cells, study design and safety aspects." Scand Cardiovasc J 39.3 (2005): 150-8.
22. Lunde K, Solheim S, Aakhus S, et al. "Exercise capacity and quality of life after intracoronary injection of autologous mononuclear bone marrow cells in acute myocardial infarction: results from the Autologous Stem cell Transplantation in Acute Myocardial Infarction (ASTAMI) randomized controlled trial." Am Heart J 154.4 (2007): 710 e1-8.
23. Lunde K, Solheim S, Aakhus S, et al. "Intracoronary injection of mononuclear bone marrow cells in acute myocardial infarction." N Engl J Med 355.12 (2006): 1199-209.
24. Meluzin J, Janousek S, Mayer J, et al. "Three-, 6-, and 12-month results of autologous transplantation of mononuclear bone marrow cells in patients with acute myocardial infarction." Int J Cardiol (2007).

Autologous Stem Cell Infusion for Mycardial Infarction, continued

25. Meluzin J, Mayer J, Groch L, et al. "Autologous transplantation of mononuclear bone marrow cells in patients with acute myocardial infarction: the effect of the dose of transplanted cells on myocardial function." Am Heart J 152.5 (2006): 975 e9-15.
26. Meyer GP, Wollert KC, Lotz J, et al. "Intracoronary bone marrow cell transfer after myocardial infarction: eighteen months' follow-up data from the randomized, controlled BOOST (BOne marrOw transfer to enhance ST-elevation infarct regeneration) trial." Circulation 113.10 (2006): 1287-94.
27. Mocini D, Staibano M, Mele L, et al. "Autologous bone marrow mononuclear cell transplantation in patients undergoing coronary artery bypass grafting." Am Heart J 151.1 (2006): 192-7.
28. Numaguchi Y, Sone T, Okumura K, et al. "The impact of the capability of circulating progenitor cell to differentiate on myocardial salvage in patients with primary acute myocardial infarction." Circulation 114.1 Suppl (2006): I114-9.
29. Obradovic S, Rusovic S, Balint B, et al. "Autologous bone marrow-derived progenitor cell transplantation for myocardial regeneration after acute infarction." Vojnosanit Pregl 61.5 (2004): 519-29.
30. Patel AN, Geffner L, Vina RF, et al. "Surgical treatment for congestive heart failure with autologous adult stem cell transplantation: a prospective randomized study." J Thorac Cardiovasc Surg 130.6 (2005): 1631-8.
31. Penicka M, Lang O, Widimsky P, et al. "One-day kinetics of myocardial engraftment after intracoronary injection of bone marrow mononuclear cells in patients with acute and chronic myocardial infarction." Heart 93.7 (2007): 837-41.
32. Perin EC, Dohmann HF, Borojevic R, et al. "Improved exercise capacity and ischemia 6 and 12 months after transendocardial injection of autologous bone marrow mononuclear cells for ischemic cardiomyopathy." Circulation 110.11 Suppl 1 (2004): II213-8.
33. Perin EC, Dohmann HF, Borojevic R, et al. "Transendocardial, autologous bone marrow cell transplantation for severe, chronic ischemic heart failure." Circulation 107.18 (2003): 2294-302.
34. Ruan W, Pan CZ, Huang GQ, Li YL, Ge JB, Shu XH. "Assessment of left ventricular segmental function after autologous bone marrow stem cells transplantation in patients with acute myocardial infarction by tissue tracking and strain imaging." Chin Med J (Engl) 118.14 (2005): 1175-81.
35. Schachinger V, Assmus B, Britten MB, et al. "Transplantation of progenitor cells and regeneration enhancement in acute myocardial infarction: final one-year results of the TOPCARE-AMI Trial." J Am Coll Cardiol 44.8 (2004): 1690-9.
36. Schachinger V, Erbs S, Elsasser A, et al. "Improved clinical outcome after intracoronary administration of bone-marrow-derived progenitor cells in acute myocardial infarction: final 1-year results of the REPAIR-AMI trial." Eur Heart J 27.23 (2006): 2775-83.
37. Schachinger V, Erbs S, Elsasser A, et al. "Intracoronary bone marrow-derived progenitor cells in acute myocardial infarction." N Engl J Med 355.12 (2006): 1210-21.
38. Smits PC, van Geuns RJ, Poldermans D, et al. "Catheter-based intramyocardial injection of autologous skeletal myoblasts as a primary treatment of ischemic heart failure: clinical experience with six-month follow-up." J Am Coll Cardiol 42.12 (2003): 2063-9.
39. Strauer BE, Brehm M, Zeus T, et al. "Regeneration of human infarcted heart muscle by intracoronary autologous bone marrow cell transplantation in chronic coronary artery disease: the IACT Study." J Am Coll Cardiol 46.9 (2005): 1651-8.
40. Strauer BE, Brehm M, Zeus T, et al. "Repair of infarcted myocardium by autologous intracoronary mononuclear bone marrow cell transplantation in humans." Circulation 106.15 (2002): 1913-8.
41. Summaries for patients. Can stem cells restore cardiac tissue after a heart attack? Ann Intern Med 140.9 (2004): I82.
42. Wollert KC, Meyer GP, Lotz J, et al. "Intracoronary autologous bone-marrow cell transfer after myocardial infarction: the BOOST randomised controlled clinical trial." Lancet 364.9429 (2004): 141-8.

Select Health

Select Health

MEDICAL POLICY

CAROTID PROCEDURES FOR STROKE PREVENTION

Policy # 461
Implementation Date: 9/28/10
Review Dates: 9/15/11, 11/29/12, 12/19/13, 12/18/14, 12/10/15, 12/15/16, 12/21/17, 12/11/18, 12/15/19, 1/6/21, 3/25/22, 1/13/23, 12/5/23, 12/17/24
Revision Dates: 6/17/22, 2/9/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Carotid endarterectomy is a surgical procedure to remove a build-up of fatty deposits (plaque), which cause narrowing of a carotid artery. The carotid arteries are the main blood vessels that supply blood to the neck, face, and brain. Carotid endarterectomies are performed when one or both carotid arteries become narrowed because of a build-up of fatty deposits (plaque). This is known as carotid artery disease or carotid artery stenosis.

The effectiveness of carotid endarterectomy (CEA) for moderate-to-severe asymptomatic or symptomatic carotid artery stenosis has been established in large, randomized trials. For patients with indications for bilateral CEA, a staged rather than combined procedure is performed.

Carotid artery stenting (CAS) involves placing an angioplasty and a stent in the narrowed artery. The stent maintains artery patency and decreases the chance of it narrowing again. CAS is an option for selected patients with contraindications to CEA due to high-risk anatomical or physiological factors for symptomatic (≥ 50 percent) or asymptomatic high-grade (≥ 80 percent) internal carotid artery stenosis.

Transcarotid artery revascularization (TCAR) is a distinct hybrid approach to carotid stenting that requires surgical exposure of the common carotid artery at the base of the neck for stent deployment. TCAR uses a specialized arterial sheath through which the stent is deployed to treat the carotid stenosis. TCAR uses dynamic flow reversal for cerebral protection. The TCAR neuroprotection system consists of the arterial sheath, venous sheath, and a flow controller with a built-in filter.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers carotid procedures for stroke prevention in *limited* circumstances; carotid endarterectomy, carotid stenting, and transcarotid artery revascularization, as the current literature demonstrates improved outcomes in select populations. Must meet either A or B.

A. Recommended by Intermountain Health Cardiovascular Clinical Provider; **OR**

B. For all other clinicians, the following criteria must be met:

1) Patients with symptomatic carotid artery stenosis ≥ 50%; or

2) Patients with asymptomatic carotid artery stenosis ≥ 70%.

Carotid Procedures for Stroke Prevention, continued

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

Several systematic reviews are available, and all describe the international studies comparing carotid artery stenting (CAS) and carotid endarterectomy (CEA). Most have concluded that CEA is the preferred method for treating patients with symptomatic stenoses greater than 50% and for patients with asymptomatic stenoses greater than 70% narrowing. The Carotid Revascularization Endarterectomy versus Stenting Trial (CREST) trial appears to have demonstrated a non-inferiority status for CAS compared with CEA. Previously, international randomized studies have shown increased rates of intraoperative CVA during CAS limiting the role of CAS in CVD. CAS would be used only when there were strict contraindications for surgery.

CREST demonstrated low rates of periprocedural stroke and death in both treatment groups as compared with the other trials. For example, the rate of periprocedural stroke among symptomatic patients treated by carotid artery stenting was 6% in CREST but 9.6% in the EVA-3S. Compared with previous studies, CREST was the only trial which used MI (myocardial infarction) as an endpoint. CAS resulted in 1.1% risk of MI compared with CEA at 2.3%. Because MI was included as an endpoint along with CVA and death, the CREST trial results demonstrated that CAS showed comparative clinical outcomes compared with CEA. Critics have suggested that the increased rate of MI cannot be considered equivalent to CVA. They illustrate that both the increased economic and health status of the patient is much worse with a CVA. Thus, CEA should still be considered the standard of care for carotid revascularization.

Many factors are important to emphasize with CREST and previous studies. Besides the influence of choosing endpoints as discussed above, the following issues are significant in determining the clinical outcomes of the studies. These include:

- Technical proficiency of the proceduralist - Enhanced training improved results in CREST compared with previous studies where training for CAS was limited
- The age of the patient - Younger patients favored CAS
- Recent symptoms of either a TIA or CVA and the need for intervention favors CEA
- Associated co–morbid condition which could increase perioperative mortality favoring CAS.

Besides these factors, critics also illustrate there were different criteria to be included in the studies. For example, carotid ultrasound (US), magnetic resonance angiogram (MRA), and computed tomography (CT) carotid angiogram were the imaging techniques used for the diagnosis of carotid stenosis. International studies compared with CREST did not use the same thresholds, thus, direct comparisons of the studies, including their overall outcomes, are difficult to perform. Newer methods to avoid peri-operative emboli have decreased the rate of stroke in CREST compared with previous studies. It is anticipated that future refinement will limit the events in the future.

Unfortunately, most randomized trials for carotid intervention have failed to include a group randomized to best medical therapy (BMT). The SPACE-2 trial did include randomization to best medical therapy for patients with asymptomatic carotid stenosis, demonstrating no difference in 30-day stroke rates for CEA,

Carotid Procedures for Stroke Prevention, continued

CAS, or BMT. Unfortunately, the trial was underpowered due to inadequate subject recruitment. Therefore, this data must be treated cautiously, and not as definitive evidence that interventional treatment of asymptomatic carotid stenosis is not warranted. A similar trial, CREST-2, has completed patient recruitment; the results of this trial are not yet published, but will likely influence optimal treatment of carotid stenosis.

Billing/Coding Information
Covered: For the conditions outline above

CPT CODES

37215	Transcatheter placement of intravascular stent(s), cervical carotid artery, percutaneous: with distal embolic protection
37217	Transcatheter placement of an intravascular stent(s), intrathoracic common carotid artery or innominate artery by retrograde treatment, via open ipsilateral cervical carotid artery exposure, including angioplasty, when performed, and radiological supervision and interpretation
37218	Transcatheter placement of intravascular stent(s), intrathoracic common carotid artery or innominate artery, open or percutaneous antegrade approach, including angioplasty, when performed, and radiological supervision and interpretation
76937-26	Ultrasound guidance for vascular access requiring ultrasound evaluation of potential access sites, documentation of selected vessel patency, concurrent real-time ultrasound visualization of vascular needle entry, with permanent recording and reporting (List separately in addition to code for primary procedure)

HCPCS CODES

No specific codes identified

ICD-10 CODES

X2AH336	Cerebral Embolic Filtration, Extracorporeal Flow Reversal Circuit from Right Common Carotid Artery, Percutaneous Approach, New Technology Group 6
X2AJ336	Cerebral Embolic Filtration, Extracorporeal Flow Reversal Circuit from Left Common Carotid Artery, Percutaneous Approach, New Technology Group 6

Key References

1. Bates, ER, Babb, JD, Casey, DE, Jr., et al. (2007). ACCF/SCAI/SVMB/SIR/ASITN 2007 Clinical Expert Consensus Document on carotid stenting. Vasc Med 12.1: 35-83.
2. Baylor College of Medicine. (2010) Carotid Artery Stenting. Baylor College of Medicine. http://www.debakeydepartmentofsurgery.org/home/content.cfm?proc_name=Carotid+Stenting&content_id=272.
3. Brott, TG, Hobson, RW, 2nd, Howard, G, et al. (2010). Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med 363.1: 11-23.
4. Carotid endarterectomy or stenting or best medical treatment alone for moderate-to-severe asymptomatic carotid artery stenosis: 5-year results of a multicentre,randomised controlled trial Tilman Reiff, Hans-Henning Eckstein, Ulrich Mansmann, Olav Jansen, Gustav Fraedrich, Harald Mudra, Dittmar Böckler, Michael Böhm,E Sebastian Debus, Jens Fiehler, Klaus Mathias, Erich B Ringelstein, Jürg Schmidli, Robert Stingele, Ralf Zahn, Thomas Zeller, Wolf-Dirk Niesen, Kristian Barlinn, Andreas Binder, Jörg Glahn, Werner Hacke, Peter Arthur Ringleb, for the SPACE-2 Investigators. www.thelancet.com/neurology. Vol 21, October 2022.
5. Cleveland Clinic. (2010) What happens during the carotid angioplasty and stenting procedure? Cleveland Clinic. http://my.clevelandclinic.org/heart/services/tests/procedures/carotidstent.aspx#3.
6. Ederle, J, Dobson, J, Featherstone, RL, et al. (2010). Carotid artery stenting compared with endarterectomy in patients with symptomatic carotid stenosis (International Carotid Stenting Study): an interim analysis of a randomised controlled trial. Lancet 375.9719: 985-97.
7. Hollander, M, Bots, ML, Del Sol, AI, et al. (2002). Carotid plaques increase the risk of stroke and subtypes of cerebral infarction in asymptomatic elderly: the Rotterdam study. Circulation 105.24: 2872-7.
8. Hopkins, LN, Roubin, GS, Chakhtoura, EY, et al. (2010). The Carotid Revascularization Endarterectomy versus Stenting Trial: credentialing of interventionalists and final results of lead-in phase. J Stroke Cerebrovasc Dis. 19.2: 153-62.
9. https://clinicaltrials.gov/study/NCT02089217
10. Jim, J. Carotid endarterectomy. *UpToDate*. Last updated: Nov. 14, 2022. https://www.uptodate.com/contents/carotid-endarterectomy

Select Health

Carotid Procedures for Stroke Prevention, continued

11. Jim, J. Overview of carotid artery stenting. *UpToDate*. Last updated: Aug. 8, 2023. https://www.uptodate.com/contents/overview-of-carotid-artery-stenting
12. Jim, J. Transcarotid artery revascularization. *UpToDate*. Last updated: Nov. 14, 2023. https://www.uptodate.com/contents/transcarotid-artery-revascularization
13. Kernan, W.N., Ovbiagele, B., Black. H. R., et al. (2014). Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack. AHA/ASA Guideline. http://stroke.ahajournals.org/lookup/suppl/doi:10.1161/STR.0000000000000024/-/DC1.
14. Mas, JL, Chatellier, G, Beyssen, B, et al. (2006). Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Engl J Med 355.16: 1660-71.
15. Mayo Clinic. (2010) Carotid Artery Disease. Mayo Clinic. Available: http://www.mayoclinic.com/health/carotid-artery-disease/DS01030.
16. Medical Technology Directory. (2009) Carotid Angioplasty and Stenting for Carotid Artery Stenosis. September 29. Winifred S. Hayes.
17. Nadalo, LAW, M.C. (2009) Carotid Artery, Stenosis. emedicine. Available: http://emedicine.medscape.com/article/417524-overview. Date accessed: August 3, 2010.
18. National Coverage Analysis (NCA): Decision Memo - CAG-00085R8. Percutaneous Transluminal Angioplasty (PTA) of the Carotid Artery Concurrent with Stenting. October 11, 2023. https://www.cms.gov/medicare-coverage-database/view/ncacal-decision-memo.aspx?proposed=N&ncaid=311.
19. Polisena, J, Banks, R. (2009) Carotid Artery Stenting vs. Carotid Endarterectomy: A Review of the Clinical and Cost-Effectiveness. September 22. Canadian Agency for Drugs and Technologies in Health (CADTH).
20. Radiology Associates of Northern Kentucky. (2010) Carotid Artery Stenting. Radiology Associates of Northern Kentucky. Available: http://www.radassociatesnky.com/int_CarotidArteryStenting.html.
21. Safian, RD, Jaff, MR, Bresnahan, JF, et al. (2010). Protected Carotid Stenting in High-Risk Patients: Results of the SpideRX Arm of the Carotid Revascularization with ev3 Arterial Technology Evolution Trial. J Interv Cardiol.
22. Stork, S, van den Beld, AW, von Schacky, C, et al. (2004). Carotid artery plaque burden, stiffness, and mortality risk in elderly men: a prospective, population-based cohort study. Circulation 110.3: 344-8.
23. Technology Evaluation Center. (2007) Angioplasty and Stenting of the Cervical Carotid Artery with Embolic Protections of the Cerebral Circulation. June. BCBS TEC Assessment Program.
24. Tice, JA. (2009) Carotid Artery Stenting. June 17. California Technology Assessment Forum (CTAF).
25. Wilterdink, JLF, K.L.; Kistler, J.P. (2010) Pathophysiology of symptoms from carotid atherosclerosis. April 12, 2010. Up to Date. http://www.uptodate.com/online/content/topic.do?topicKey=cva_dise/5449&selectedTitle=4%7E150&source=search_result.

Revision History

Revision Date	Summary of Changes
2/9/24	Modified title of policy (previously titled, Carotid Artery Stenting (CAS)), and for Commercial Plan Policy, updated coverage criteria to align with current clinical standards: "Select Health covers carotid procedures for stroke prevention in limited circumstances; carotid endarterectomy, carotid stenting, and transcarotid artery revascularization, as the current literature demonstrates improved outcomes in select populations. Must meet either A or B.; A. Recommended by Intermountain Health Cardiovascular Clinical Provider; OR B. For all other clinicians, the following criteria must be met: 1) Patients with symptomatic carotid artery stenosis ≥ 50%; or 2) Patients with asymptomatic carotid artery stenosis ≥ 70%."

Select Health

Carotid Procedures for Stroke Prevention, continued

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

HEART TRANSPLANT: ADULT

Policy # 125
Implementation Date: 2/10/99
Review Dates: 1/4/00, 2/27/01, 5/21/01, 5/13/02, 5/25/03, 4/22/04, 4/17/06, 5/17/07, 4/24/08 4/23/09, 8/19/10, 9/15/11, 4/25/13, 2/20/14, 3/19/15, 2/16/17, 10/4/18, 10/15/19, 6/17/21, 8/2/22, 8/17/23, 6/18/24
Revision Dates: 5/30/04, 1/28/10, 2/14/12, 2/16/16, 10/12/17, 10/8/18, 12/10/21, 7/8/22

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Cardiac transplantation remains the treatment of choice for many patients with end-stage heart failure (HF) with severely impaired functional capacity, despite optimal medical therapy. Although barriers to long-term survival remain, the outcome among transplant recipients has improved over several decades as a result of careful recipient and donor selection, advances in immunosuppression, and the prevention and treatment of opportunistic infections.

In the most recent registry report, the median survival for heart transplants performed between 1982 and June 2015 was 11 years for adult recipients and 16 years for pediatric recipients. Patient survival has steadily improved since the 1980s, with one-year survival rates now exceeding 85 percent for adult patients and 90 percent for pediatric patients transplanted in the most current era (2009 through June 2015). The major survival gains are limited to the first 6 to 12 months, with a long-term attrition rate of 3.4 percent per year thereafter, remaining largely unchanged. The improvement is probably larger than it appears since the risk profile of recipients and the age of donors continue to increase.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

To qualify for coverage, adult candidates for transplant must meet **EITHER** of the following criteria **(A or B)** (except for special cardiac conditions*):

A. Approved if recommended by Intermountain Transplant Services;

OR

B. Must meet **ALL** the following criteria:

1. Provided by In-Network Providers in an In-Network Facility** unless otherwise approved in writing in advance by SelectHealth. **This criterion does not apply to Idaho commercial plans. Members on Idaho commercial plans may use their out-of-network benefits with an out-of-network provider if all other criteria are met.
2. The patient has end-stage cardiac disease that is irreversible and progressive, with limited life expectancy, and with no available reasonable alternative medical or surgical therapy; and
3. NYHA Class III or Class IV cardiac dysfunction (see below for definition of NYHA functional classifications); and
4. Must have at least **ONE** of the following diagnoses:

Heart Transplant: Adult, continued

   a. Coronary artery disease/ischemic cardiomyopathy
   b. Idiopathic dilated cardiomyopathy
   c. Valvular heart disease
   d. Myocarditis
   e. Restrictive cardiomyopathy
   f. Congenital heart disease
   g. Adriamycin cardiomyopathy
   h. Peripartum cardiomyopathy
   i. Hypertrophic cardiomyopathy
   j. Infiltrative cardiomyopathy—only if confined to heart (if not, it is an absolute contraindication)
   k. Chagas Disease
   l. *Unresectable primary cardiac tumor
   m. *Intractable life-threatening arrhythmias
   n. *Severe cardiac allograft vasculopathy
   o. When pulmonary hypertension exists, pulmonary vascular resistance less than or equal to 5 wood units, with or without afterload reduction (e.g., Nitroprusside, etc.)
5. A reasonable expectation that the patient's quality of life, i.e., physical and social function suited to activities of daily living, will be improved
6. Strong motivation by the patient to undergo the procedure and a thorough understanding by the patient and family of the magnitude of the operation, its risks, and sequelae, including lifetime follow-up
7. Medical assessment that the patient will have a tolerance for immunosuppressive thrapy and that no other major system disease or anomaly is present which would preclude surgery or a reasonable chance of survival
8. Medical and social assessment that the patient has sufficient social stability to provide assurance that they will cooperate with the long-term follow-up and the immunosuppressive program, which is required
9. No uncontrolled and/or untreated psychiatric disorder or substance use disorder that would interfere with compliance to a treatment regimen
10. Age at time of transplant listing: > 18 and ≤ 70 years

<u>Absolute Contraindications:</u>

1. Severe, irreversible pulmonary hypertension
2. Pulmonary disease as listed below:
   a. Cystic fibrosis
   b. Obstructive pulmonary disease (FEV < 65% of predicted)
   c. Restrictive pulmonary disease (FVC < 50% of predicted)
   d. Unresolved pulmonary roentgenographic abnormalities of unclear etiology
   e. Unresolved pulmonary infarction
3. Vascular disease
4. Unresolved GI bleeding
5. Unresolved diverticulitis
6. Irreversible liver disease
7. Hepatitis B antigen positivity
8. Untreated positive Hepatitis C serology with severe pathology on liver biopsy and/or elevated transaminases
9. Active malignant disease

Heart Transplant: Adult, continued

10. Active infection
11. Neuromuscular disorders with physical limitations impacting survival/QOL post-transplant
12. History of medical non-compliance

Relative Contraindications:

13. History of psychiatric illness
14. Morbid obesity (Body Mass Index > 35 kg/m²)
15. Renal insufficiency as manifested a creatinine clearance below 30 mL/min. The concern in this setting is the superimposed nephrotoxicity of long-term cyclosporine therapy.
16. HIV positivity (can be considered if **ALL** the following):
    a. No active or prior opportunistic infections or CNS lymphoma, or visceral Kaposi sarcoma,
    b. Clinically stable and compliant on combination antiretroviral therapy (cART) for 3 months,
    c. Have undetectable HIV RNA and have CD4 counts > 200 cells/µl for > 3 months).
17. Advanced hepatic disease. Cirrhosis, for example, can limit survival and increase perioperative morbidity, particularly if a coagulopathy is present.
18. Diabetes mellitus with end-organ dysfunction (other than non-proliferative retinopathy), chronic infections, leg ulcers, or persistent poor glycemic control (HbA1C > 7.5% despite optimal effort.
19. Peripheral vascular disease is a relative contraindication for transplantation when its presence limits rehabilitation and revascularization is not a viable option.

Several other conditions increase the rate of perioperative complications or interact poorly with immunosuppressive agents. Included in this group are advanced peripheral vascular disease, morbid obesity, active peptic ulcer disease, cholelithiasis, and diverticulosis.

All cardiac transplantation candidates should undergo a complete psychosocial evaluation during the initial screening process. This may identify social and behavioral factors that cause difficulty during the waiting period, convalescence, and long-term post-operative management. The patient must understand that full cooperation and compliance are critical to the safe and effective use of immunosuppressive agents.

New York Heart Association (NYHA) Functional Cardiac Classifications:

20. Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea or anginal pain.
21. Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea, or anginal pain.
22. Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitation, dyspnea, or anginal pain.
23. Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

Heart Transplant: Adult, continued

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

In 2001, the Clinical Practice Committee of the American Society of Transplantation published recommendations for considering transplantation in patients with cardiac conditions that have not responded to maximal medical management.

Improved survival should be the primary indication for cardiac transplantation and the primary selection task is to predict the prognosis in severe heart failure. Many predictors of poor prognosis have been identified, including NYHA functional class III or IV, reduced left ventricular ejection fraction (LVEF), and hyponatremia. However, there is sufficient overlap that these factors are of limited use in a particular individual. This is not surprising in view of the complexity of HF and the multiple neurohumoral, hemodynamic, and electrophysiological factors that may contribute to morbidity and mortality.

In general, the peak VO2 appears to provide the most objective assessment of functional capacity in patients with HF, and may be the best predictor of when to list an individual patient for cardiac transplantation. The 2002 task force of the ACC/AHA has given a class I recommendation to the use of exercise testing with ventilator gas analysis for this purpose.

The value of using the peak VO2 for this purpose is illustrated by a study of 114 consecutive ambulatory patients with advanced disease referred for possible cardiac transplantation. Three groups of patients were prospectively divided and had their outcomes compared based on their VO2 outcomes. They were comparable with regards to other clinical parameters of disease severity, including NYHA functional class, LVEF, and cardiac index. The 1-year survival of the healthier patients in group 2 was 94%, an outcome that was similar to that in transplanted patients in group 1. The prognosis was poorest in group 3, although survival varied with the peak VO2 in this group. Patients with a value less than or equal to 10 mL/kg per min had the lowest survival, while those between 10−14 mL/kg per min had an outcome that was only slightly worse than patients in group 1. Thus, patients with a profoundly reduced exercise capacity of 10 mL/kg per min are likely to experience the most pronounced improvement in survival with transplantation.

The small survival benefit seen for group 3 patients with a peak VO2 of 10−14 mL/kg per min illustrates the difficulty in selection of patients for transplantation. Many of these patients will benefit from transplantation, particularly those with a peak VO2 of 12 mL/kg per min. Patients in this intermediate range of peak VO2 who are initially considered too well for transplantation should have several measurements of exercise capacity over a period. Some will improve on repeated testing, but those with persistent values of 10−12 mL/kg per min and poor exercise tolerance should generally be considered for transplantation. Repeated hospitalization and/or the requirement for increasing medical therapy are additional indicators of likely benefit from transplantation.

It must be emphasized that these observations and recommendations for transplantation are applicable only if a severely reduced peak VO2 is caused by HF. Proper interpretation of the test requires that the patient achieve the anaerobic threshold, indicating that the level of exercise performed exceeded that which can be supported by the cardiovascular system on an aerobic basis. Factors that can prematurely terminate the test must be excluded, including significant peripheral vascular disease, arthritis, or angina pectoris. The peak VO2 should also be interpreted in light of the patient's age, lifestyle, and expectations. A peak VO2 of 14 mL/kg per min may represent mild impairment to a 60-year-old patient, but marked impairment to a 20-year-old patient.

Although peak VO2 is generally used to guide the selection of heart transplant candidates, a single variable cannot provide an optimal risk profile. As a result, several risk models have been developed that use factors identified in multivariable survival analysis to establish a risk score for prognosis in these patients.

Heart Transplant: Adult, continued

One model that has been validated prospectively is the Heart Failure Survival Score (HFSS). This score was derived from a multivariable analysis of 268 ambulatory patients referred for consideration of cardiac transplantation and validated in 199 similar patients. Seven variables were used as predictors of survival in the HFSS. These include the presence or absence of coronary artery disease, resting heart rate, left ventricular ejection fraction, mean arterial blood pressure, presence or absence of an interventricular conduction delay on ECG, serum sodium, and peak VO2. In an invasive version of the HFSS, pulmonary capillary wedge pressure is included as an eighth variable. The HFSS then stratifies patients into low-, medium-, and high-risk categories, based upon a sum of the variables above multiplied by defined coefficients. Among the patients in the validation sample, one-year survival rates without transplant for these three strata were 88%, 60%, and 35%, respectively. Other risk models exist and are used by various centers to assess patients' candidacy for heart transplantation.

Although the most common indication for heart transplantation is severe HF refractory to medical therapy, the operation may be recommended in other circumstances. These include severely limiting ischemia not amenable to interventional or surgical revascularization, recurrent symptomatic ventricular tachyarrhythmias refractory to medical therapy, an implantable cardioverter-defibrillator, surgery, and rarely, for the management of cardiac tumors.

Even the patient who meets the above requirements must be excluded from transplantation when one or more absolute contraindications are demonstrated by standard evaluation procedures. In addition, relative contraindications must be considered in judging whether a patient is likely to benefit from heart transplantation. Absolute contraindications include pulmonary vascular resistance (PVR) greater than 4−6 Wood units (320−480 dynes-sec/cm^5) (normal 1.5 Wood units [120 dynes-sec/cm^5]) or a transpulmonary gradient (mean pulmonary artery pressure minus mean pulmonary capillary wedge pressure) above 15 mmHg have an increased risk of right ventricular failure in the immediate postoperative period.

Patients whose PVR can be pharmacologically reduced to below 4 Wood units (320 dynes-sec/cm^5) are usually considered acceptable for transplantation. In one report, for example, the 3-month mortality rate was higher in patients whose PVR was above 2.5 Wood units compared to those with lower values (17.9% versus 6.9%). However, the 3-month mortality was only 3.8% in those with initially high values that were reduced by nitroprusside, compared to 41% and 28%, respectively, in those who were resistant to nitroprusside or who only responded at a dose that caused systemic hypotension. None of the patients with reversible pulmonary hypertension developed right ventricular failure after transplantation.

Two other absolute contraindications to transplantation are active infection and malignancy of any kind, both of which can be worsened by the immunosuppressive therapy given to prevent transplant rejection.

Heart transplantation in patients with clinically important chronic viral infection remains a subject of active debate. Individuals with chronic hepatitis B or hepatitis C infections who undergo heart transplantation have an increased frequency of liver disease. However, it has been difficult to show that survival after heart transplantation is poorer in the presence of positive hepatitis B or C serology. As a result, practices of individual centers differ. Since the frequency of progressive liver disease appears to be more common with hepatitis B than with hepatitis C, many transplant programs will accept candidates who are anti-HCV antibody positive, but not those who are HBsAg positive.

HIV infection has been considered to be an absolute contraindication to heart transplantation, primarily because of concerns about the increased frequency of infectious and malignant complications and the previously poor survival of such patients. However, the advent of highly active antiretroviral therapy has changed the prognosis of HIV. As a result, the view has been expressed that HIV infection itself should not be sufficient reason to refuse transplantation.

Among the relative contraindications to cardiac transplantation, age has historically been a major factor. Many programs have routinely excluded patients over the age of 60–65. However, carefully selected patients in this age group or older have a survival rate comparable to that in younger patients. One report compared 63 patients transplanted at 65 years of age or older (mean age 67) to 63 matched younger patients (mean age 48). Survival was comparable for both groups at 1, 3, 5, and 10 years; there was also no significant difference in the incidence of rejection or infection. As a result, most centers now focus on the patient's "physiologic" age, with emphasis on the functional integrity of major organ systems and the absence of exclusionary comorbid diseases.

Heart Transplant: Adult, continued

Diabetes mellitus, even without evidence of significant end-organ damage (neuropathy, retinopathy, or nephropathy), is a relative contraindication to heart transplantation. However, the outcome in properly selected patients is comparable to that in nondiabetics. This was illustrated in an analysis of 345 consecutive heart transplant recipients, 101 with diabetes. Diabetics, compared to nondiabetics, had a non-significant trend toward decreased survival at one year (85% vs. 91%) but comparable survival at 5 years (82%). Rates of infection severe enough to require hospitalization were higher among diabetics at 90 days (14% vs. 3%) and 4 years (29% vs. 15%). There were no differences in the incidence of rejection, transplant coronary disease, or renal dysfunction.

Advanced obstructive and/or restrictive lung disease is associated with a higher incidence of postoperative lung complications, including infection associated with immunosuppressive therapy. Objective exclusion criteria include a forced one-second expiratory volume of less than 1.0 liter; a forced vital capacity of less than 50% of predicted; or a forced expiratory volume-to-vital capacity ratio of less than 1.0. In addition, recent pulmonary embolism with or without infarction should delay transplantation, because secondary infection in the affected lobe may occur postoperatively. Before putting the patient on the transplant list, most centers treat this disorder with systemic anticoagulants for 6−8 weeks or until radiographic evidence of resolution is seen.

A January 2012 Medical Technology Assessment focused on the maximum age at transplant. Nine primary literature articles and no systematic reviews were identified regarding heart transplantation as it relates to upper age limitations. A total of 16,892 patients reported in these studies underwent heart transplantation. Though the articles, in general, did well to report follow-up times and age ranges of participants, few appropriately matched patients for age, sex, weight, or presence of comorbidities such as diabetes mellitus. Only 2 of the 9 papers (Crespo-Leiro et al. and Morgan et al.) specifically addressed the safety and efficacy of heart transplantation in patients > 65 years of age and < 65 years of age. Both groups noted that among carefully selected patients aged more than 65 years, heart transplantation can be performed without incurring a greater risk of rejection or death than in patients aged less than 65 years.

From the literature, it is notable that among the relative contraindications to cardiac transplantation, age has historically been a major factor. Many programs have routinely excluded patients over the age of 60−65 while advocating the view that physiologic age is more important than chronological age.

Speaking to the issue of the outcomes related to transplanting a more mature population, a report based on United Network for Organ Sharing (UNOS) data for 14,401 first-time transplant recipients between 1999 and 2006 demonstrated that patients ≥ 60 years of age more frequently had comorbidities than younger patients and that their overall survival post-transplant was lower than in younger patients. However, cumulative five-year survival for patients ≥ 60 years of age was only slightly lower than that for younger patients (69% vs. 75%). There is no data to suggest that this disparity is a direct result of the heart transplantation itself. Multivariate analysis revealed recipient age ≥ 60 years, donor age, ischemic time, creatinine, hypertension, and diabetes to be independent predictors of mortality.

One of the most significant studies demonstrating the lack of age as being a significant variable affecting outcome was the 2006 ISHLT update on the listing criteria and management of cardiac transplantation candidates. This study suggested that most patients 70 years of age or younger and carefully selected patients over age 70 can be considered for cardiac transplantation. This finding is supported by most additional references cited in this report.

Billing/Coding Information

CPT CODES

33940	Donor cardiectomy (including cold preservation)
33944	Backbench standard preparation of cadaver donor heart allograft prior to transplantation, including dissection of allograft from surrounding soft tissues to prepare aorta, superior vena cava, inferior vena cava, pulmonary artery, and left atrium for implantation
33945	Heart transplant, with or without recipient cardiectomy

Heart Transplant: Adult, continued

HCPCS CODES

Not covered: Investigational/Experimental/Unproven for this indication

S2152 Solid organ(s), complete or segmental, single organ or combination of organs; deceased or living donor(s), procurement, transplantation, and related complications including: drugs; supplies; hospitalization with outpatient follow-up; medical/surgical, diagnostic, emergency, and rehabilitative services; and the number of days of pre- and post-transplant care in the global definition

S9975 Transplant related lodging, meals and transportation, per diem

Key References

1. Botta, DM. (2011) Heart Transplantation. October 26, 2011. Medscape. Available: http://emedicine.medscape.com/article/429816-overview#a0104. Date Accessed: December 30, 2011.
2. Carey, WD. (2010) Current Clinical Medicine. Saunders Elsevier. Available: http://www.mdconsult.com/books/page.do?eid=4-u1.0-B978-1-4160-6643-9.00236-8&isbn=978-1-4160-6643-9&uniqId=310518069-8#4-u1.0-B978-1-4160-6643-9.00236-8. Date Accessed: January 5, 2012.
3. Colucci, Wilson S. UpToDate, (2004). Chapter on Indications and Exclusions for Cardiac Transplantation.
4. Colucci, WS. (2011) Evaluation of the patient with suspected heart failure. March 8, 2010. Up to Date. Available: http://www.uptodate.com/contents/evaluation-of-the-patient-with-suspected-heart-failure?source=search_result&search=heart+failure&selectedTitle=2~150. Date Accessed: January 10, 2012.
5. Colucci, WS. (2011) Indications and contraindications for cardiac transplantation. February 18, 2011. Up to Date. Available: http://www.uptodate.com/contents/indications-and-contraindications-for-cardiac-transplantation. Date Accessed: January 6, 2012.
6. Comparative analysis of outcome. J Thorac Cardiovasc Surg 121.3: 532-41.
7. Crespo-Leiro, MG, Paniagua-Martin, MJ, Muniz, J, et al. (2005). Long-term results of heart transplant in recipients older and younger than 65 years: a comparative study of mortality, rejections, and neoplasia in a cohort of 445 patients. Transplant Proc 37.9: 4031-2.
8. Daneshvar, D, Czer, LS, Phan, A, et al. (2011). Heart transplantation in patients aged 70 years and older: a two-decade experience. Transplant Proc 43.10: Blanche, C, Blanche, DA, Kearney, B, et al. (2001). Heart transplantation in patients seventy years of age and older: A 3851-6.
9. Demers, P, Moffatt, S, Oyer, PE, et al. (2003). Long-term results of heart transplantation in patients older than 60 years. J Thorac Cardiovasc Surg 126.1: 224-31.
10. Eisen, HJ. (2011) Patient information: Heart transplantation. September 29. Up to Date. Available: http://www.uptodate.com/contents/patient-information-heart-transplantation. Date Accessed: January 6, 2012.
11. Forni, A, Faggian, G, Chiominto, B, et al. (2007). Heart transplantation in older candidates. Transplant Proc 39.6: 1963-6.
12. Gibbons, RJ, Balady, GJ, Bricker, JT, et al. (2002). ACC/AHA 2002 guideline update for exercise testing: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1997 Exercise Testing Guidelines). Circulation; 106:1883.
13. Goldman, L. (2011) Goldman's Cecil Medicine. Saunders Elsevier. Available: http://www.mdconsult.com/books/pagedo?eid=4-u1.0-B978-1-4377-1604-7.00082-8&isbn=978-1-4377-1604-7&uniqId=311439269-5#4-u1.0-B978-1-4377-1604-7.00082-8. Date Accessed: January 10, 2012.
14. Heartfailure.org. (2012) Heart Failure. Heartfailure.org. Available: http://www.heartfailure.org/eng_site/hf.asp. Date Accessed: January 10, 2012.
15. Hunt, SA, Abraham, WT, Chin, MH, et al. (2009). 2009 Focused update incorporated into the ACC/AHA 2005 Guidelines for the Diagnosis and Management of Heart Failure in Adults A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines Developed in Collaboration With the International Society for Heart and Lung Transplantation. J Am Coll Cardiol 53.15: e1-e90.
16. ISHLT GUIDELINE The 2016 International Society for Heart Lung, Transplantation listing criteria for heart transplantation: A 10-year update, Mandeep R. Mehra, MD (Chair)et al, The Journal of Heart and Lung Transplantation, Vol 35, No 1, January 2016.
17. Marelli, D, Kobashigawa, J, Hamilton, MA, et al. (2008). Long-term outcomes of heart transplantation in older recipients. J Heart Lung Transplant 27.8: 830-4.
18. Mehra, MR, Kobashigawa, J, Starling, R, et al. (2006). Listing criteria for heart transplantation: International Society for Heart and Lung Transplantation guidelines for the care of cardiac transplant candidates--2006. J Heart Lung Transplant 25.9: 1024-42.
19. Morgan, JA, John, R, Weinberg, AD, et al. (2003). Long-term results of cardiac transplantation in patients 65 years of age and older: a comparative analysis. Ann Thorac Surg 76.6: 1982-7.
20. Nagendran, J, Wildhirt, SM, Modry, D, et al. (2004). A comparative analysis of outcome after heart transplantation in patients aged 60 years and older: the University of Alberta experience. J Card Surg 19.6: 559-62.
21. Pham, Michael X. UpToDate, (2018). Prognosis after cardiac transplantation in adults.
22. Robbins, RC, Barlow, CW, Oyer, PE, et al. (1999). Thirty years of cardiac transplantation at Stanford University. J Thorac. Cardiovasc Surg; 117:939.
23. Steinman, TI, Becker, BN, Frost, AE, et al. (2001). Guidelines for the referral and management of patients eligible for solid organ transplantation. Transplantation 71.9: 1189-204.
24. The Registry of the International Society for Heart and Lung Transplantation: (1992). Ninth official report. J Heart Lung Transplant; 11:599.
25. UCSF Medical Center. (2012). Heart Failure Treatment. January 9, 2012. UCSF Medical Center. Available: http://www.ucsfhealth.org/conditions/heart_failure/treatment.html. Date Accessed: January 10, 2012.

Select Health

Heart Transplant: Adult, continued

26. Weiss, ES, Nwakanma, LU, Patel, ND, et al. (2008). Outcomes in patients older than 60 years of age undergoing orthotopic heart transplantation: an analysis of the UNOS database. J Heart Lung Transplant 27.2: 184-91.
27. Wilbur, J. (2005) Diagnosis and Management of Heart Failure in the Outpatient Setting. Saunders. Available: http://www.mdconsult.com/das/article/body/311439269-3/jorg=journal&source=&sp=15908512&sid=0/N/508069/1.html?issn=0095-4543#S0095454305000801. Date Accessed: January 10, 2012.

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

Heart Transplant: Adult, continued

26. Weiss, ES, Nwakanma, LU, Patel, ND, et al. (2008). Outcomes in patients older than 60 years of age undergoing orthotopic heart transplantation: an analysis of the UNOS database. J Heart Lung Transplant 27.2: 184-91.
27. Wilbur, J. (2005) Diagnosis and Management of Heart Failure in the Outpatient Setting. Saunders. Available: http://www.mdconsult.com/das/article/body/311439269-3/jorg=journal&source=&sp=15908512&sid=0/N/508069/1.html?issn=0095-4543#S0095454305000801. Date Accessed: January 10, 2012.

Select Health

MEDICAL POLICY

HEART TRANSPLANT: CHILDREN (UNDER AGE 18)

Policy # 126

Implementation Date: 2/10/99

Review Dates: 1/4/00, 2/27/01, 5/21/01, 5/13/02, 6/25/03, 4/22/04, 4/19/05, 4/20/06, 8/23/07, 8/21/08, 8/13/09, 9/15/11, 11/29/12, 10/24/13, 10/23/14, 10/15/15, 2/16/17, 2/15/18, 2/5/19, 2/13/20, 2/18/21, 1/13/22, 3/21/23, 2/5/24, 2/11/25

Revision Dates: 9/15/06, 8/19/10, 2/16/16, 2/26/20, 1/27/22, 8/10/23

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

Worldwide, > 700 pediatric heart transplantation procedures are performed each year, representing about 12% of the total number of heart transplants performed. Most pediatric heart transplantation programs now have 5-year survival rates in excess of 80%.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers heart transplant in children under age 18 who meet EITHER of the following criteria (A or B).

A. Approved if recommended by Intermountain Health Transplant Team;
OR
B. For services being requested outside of Intermountain Health, guidelines for coverage include **ALL** the following:

1. The patient was evaluated and accepted for transplant by a paneled transplant team; and
2. The patient has end-stage cardiac disease, which is irreversible and progressive, a limited life expectancy or documented progressive (but partially reversible) pulmonary hypertension, or both, and no available reasonable alternative medical or surgical therapy; and
3. The patient suffers from New York Heart Association (NYHA) Class III or Class IV cardiac dysfunction - when criteria can be applied (see below for definition of NYHA Classes) despite maximal medical therapy; and
4. **ONE** of the following diagnoses:
   a. Cardiomyopathy
   b. Inoperable congenital heart disease, for which there is no reasonable corrective operation
   c. Myocarditis
   d. Cardiac tumor
5. Pulmonary vascular resistance < 6 wood units with Nitroprusside or other vasodilators. (does not apply to infancy); and
6. A reasonable expectation that the patient's quality of life, i.e., physical and social function suited to activities of daily living, will be improved; and

Heart Transplant Children: Under Age 18, continued

7. Strong motivation and emotional stability of parent/guardian, a thorough understanding by the patient and family of the magnitude of the operation and its sequelae, including lifetime follow-up; and
8. Medical assessment that the patient will have a tolerance for immunosuppressive therapy and that no other major system disease or anomaly is present which would preclude surgery or a reasonable survival; and
9. Medical and social assessment that the parent/guardian and child have sufficient social stability to provide assurance that he/she will cooperate with the long-term follow-up and the immunosuppressive program which is required; and
10. No uncontrolled and/or untreated psychiatric disorder that would interfere with compliance to a treatment regimen; and
11. Child is > 36 weeks gestational age and weight > 2.2 kilograms.

Absolute Contraindications

1. Severe pulmonary hypertension (PA pressure) or pulmonary vascular resistance (PVR) greater than 6 Wood units/m2, with inability of medications to reduce PVR and PA pressure to acceptable levels
2. Recent pulmonary infarct
3. Persistent acidosis with pH less than 7.1
4. Irreversible and severe renal, hepatic, CNS or pulmonary dysfunction.
5. Active/unresolved alcohol or drug abuse.
6. Active malignancy or history of malignancy with high rate of recurrence
7. Unclear cardiac diagnosis
8. Active, uncontrolled infection

Relative Contraindications

1. Psychosocial considerations as listed below:
   a. Strong history of parent/guardian alcohol and/or substance abuse.
   b. Documented parent/guardian child abuse or neglect.
   c. Family unable to support long-term medical needs of recipient.
   d. Parent/guardian with cognitive/psychiatric impairment severe enough to limit comprehension of medical regimen.
   e. Documented parent/guardian and/or patient noncompliance with previous medical care.
2. HIV positivity (transplant can be considered if ALL the following)
   a. No active or prior opportunistic infections or CNS lymphoma, or visceral Kaposi sarcoma,
   b. Clinically stable and compliant on combination antiretroviral therapy (cART)
   c. Have undetectable HIV RNA and have CD4 counts > 200 cells/µl for > 3 months.
3. Diabetes mellitus, with A1C levels > 8

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

Heart Transplant Children: Under Age 18, continued

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information
The 4 main etiologies leading to conditions that might require heart transplantation are errors in the formation of the heart, cardiac tumors, infections, and toxins (either endogenous or exogenous), leading to damage to the myocardium. Many of the congenital anomalies, including congenital cardiomyopathy, are now known to have specific chromosomal abnormalities associated with them.

Conditions considered for pediatric heart transplantation include the following:

- Cardiomyopathy (i.e., dilated, hypertrophic, restrictive)
- Anatomically uncorrectable congenital heart disease (e.g., HLHS, pulmonary atresia with intact ventricular septum plus sinusoids, congenitally corrected transposition of the great arteries with single ventricle and heart block, severely unbalanced atrioventricular septal defects)
- Congenital heart disease at high risk for repair (e.g., severe Shone complex, interrupted aortic arch and severe subaortic stenosis, critical aortic stenosis with severe endocardial fibroelastosis, Ebstein anomaly in a symptomatic newborn)
- Refractory heart failure
- Unresectable symptomatic cardiac neoplasms

Billing/Coding Information

33940	Donor cardiectomy (including cold preservation)
33944	Backbench standard preparation of cadaver donor heart allograft prior to transplantation, including dissection of allograft from surrounding soft tissues to prepare aorta, superior vena cava, inferior vena cava, pulmonary artery, and left atrium for implantation
33945	Heart transplant, with or without recipient cardiectomy

HCPCS CODES

S2152	Solid organ(s), complete or segmental, single organ or combination of organs; deceased or living donor(s), procurement, transplantation, and related complications; including: drugs; supplies; hospitalization with outpatient follow-up; medical/surgical, diagnostic, emergency, and rehabilitative services; and the number of days of pre- and post-transplant care in the global definition

Key References

1. ISHLT GUIDELINE. The 2016 International Society for Heart Lung, Transplantation listing criteria for heart transplantation: A 10-year update, Mandeep R. Mehra, MD (Chair)et al, The Journal of Heart and Lung Transplantation, Vol 35, No 1, January 2016.
2. ISHLT website. https://ishltregistries.org/registries/quarterlyDataReportResults.asp?organ=HR&rptType=all&continent=4. Accessed December 31, 2019.
3. Lee, TM, Hsu DT, Kantor P et al. (2017). Pediatric Cardiomyopathies. Circ Res. 121.7: 855-73.
4. Mehra MR, Canter CE, Hannan MM et al. (2016). The 2016 International Society for Heart Lung Transplantation listing criteria for heart transplantation: A 10-year update. J Heart Lung Transplant 35.1: 1-23.
5. Organ Procurement and Transplantation Network. National Data. Available at https://optn.transplant.hrsa.gov/data/view-data-reports/national-data/#. Accessed December 31, 2019.
6. Rossano JW, Singh TP, Cherikh WS et al (2019). The International Thoracic Organ Transplant Registry of the International Society for Heart and Lung Transplantation: Twenty-second pediatric heart transplantation report – 2019; Focus theme: Donor and recipient size match. J Heart Lung Transplant. 38.10: 1028-41.
7. Rossano JW, Shaddy RE. (2014). Heart Failure in Children: Etiology and Treatment. J Pedatr. 165.2: 228-33.

Heart Transplant Children: Under Age 18, continued

8. Rossano JW, Dipchand AI, Edwards LB, et al. The Registry of the International Society for Heart and Lung Transplantation: Nineteenth Pediatric Heart Transplantation Report-2016; Focus Theme: Primary Diagnostic Indications for Transplant. J Heart Lung Transplant. 2016 Oct. 35.10:1185-1195.
9. Thrush PT, Hoffman TM. (2014). Pediatric heart transplantation—indications and outcomes in the current era. J Thorac Dis. 6.8: 1080-1096.

Revision History

Revision Date	Summary of Changes
8/10/23	For Commercial Plan Policy, added "Unclear cardiac diagnosis" and "Active, uncontrolled infection" as absolute contraindications to this procedure.

Select Health

Select Health

MEDICAL POLICY

HEART-LUNG TRANSPLANT

Policy # 127

Implementation Date: 2/10/99

Review Dates: 1/4/00, 2/27/01, 5/21/01, 5/13/02, 6/25/03, 4/22/04, 4/22/05, 8/23/07, 8/21/08, 8/13/09, 8/19/10, 9/15/11, 7/18/13, 6/11/15, 6/16/16, 6/15/17, 7/20/18, 6/13/19, 6/18/20, 3/15/22, 4/20/23, 6/18/24

Revision Dates: 9/15/06, 3/29/22

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Cardiopulmonary transplantation (heart and lung transplantation) is the simultaneous surgical replacement of the heart and lungs in patients with end-stage cardiac and pulmonary disease. This procedure remains a viable therapeutic alternative for patients in specific disease states, although the frequency of application has substantially diminished in recent years.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers heart-lung transplantation *with limitations as outlined below* (either A or B must be met).

A. Heart-Lung Transplantation will be approved if recommended by Intermountain Health Cardiovascular Clinical Program, **or**

B. For clinicians outside of Intermountain Health, all the following criteria must be met:
   1) Provided by In-Network Providers in an In-Network Facility* unless otherwise approved in writing in advance by Select Health; and
   2) Review of the transplant team demonstrates that patient has a significant and reasonable probability for improved health post-transplant; and
   3) Transplant is not being performed as part of an investigational trial; and
   4) Patient has severe cardiopulmonary disease which is refractory to standard-of-care therapy.

**This criterion does not apply to Idaho commercial plans. Members on Idaho commercial plans may use their out-of-network benefits with an out-of-network provider if all other criteria are met.*

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For this policy, specifically, there are no CMS criteria available; therefore, the Select Health Commercial policy or InterQual criteria apply. Select Health

Select Health

Heart-Lung Transplant, continued

applies these requirements after careful review of the evidence that supports the clinical benefits outweigh the clinical risks. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information
The International Society Heart and Lung Transplantation (ISHLT) summarized the distribution of diagnoses leading to heart-lung transplantation from 1988 to 2019. The 3 leading indications were:

- Nonidiopathic pulmonary arterial hypertension due to congenital heart disease or cardiomyopathy: 38 %: 32%
- Idiopathic pulmonary arterial hypertension: 28.4%
- Cystic fibrosis: 15%

Heart-lung transplantation may also be required in patients with end-stage parenchymal lung disease who also have severely compromised left ventricular function (e.g., sarcoidosis).

The updated joint guidelines from the American Thoracic Society/American Society for Transplant Physicians/European Respiratory Society and the ISHLT guidelines does not suggest an age limit for heart-lung transplantation. Among patients with severe lung disease, referral for heart-lung transplantation should be made if the patient is a New York Heart Association (NYHA) class III or IV heart failure despite optimal surgical and medical treatment. Malignant ventricular arrhythmias not amenable to pharmacologic or electrophysiologic interventions (including an implantable cardioverter-defibrillator) in patients with end-stage lung disease are rare indications for the combined procedure.

Patients with idiopathic pulmonary arterial hypertension with preserved left ventricular function are best treated with double-lung transplantation. Combined heart-lung transplantation is the preferred procedure for patients with complex congenital heart disease with Eisenmenger syndrome, including those with single ventricle anatomy, unsuccessfully repaired or uncorrectable lesions, and/or severely depressed left ventricular function.

On the other hand, bilateral lung transplantation with repair of the congenital defect is the recommended procedure in patients with simple congenital heart disease. These lesions include:

- Atrial or ventricular septal defect
- Scimitar syndrome
- Pulmonary venous stenosis
- A functionally inadequate vascular bed as with multiple peripheral pulmonary arterial stenoses or pulmonary arteriovenous malformations.

Orthotopic heart transplantation alone has been performed in patients with congenital heart disease and a physiologic single lung (e.g., unilateral pulmonary venous stenosis, or due to previous aortopulmonary shunt procedure). Heart-lung transplant recipients receive an "en bloc" harvested heart and lung allograft and can be listed under both the lung and heart allocation systems. The lung allocation system in the United States was changed by United Network for Organ Sharing (UNOS) in 2018 (https://optn.transplant.hrsa.gov/policies-bylaws/policies/).

Billing/Coding Information

CPT CODES

33930	Donor cardiectomy-pneumonectomy (including cold preservation)

Heart-Lung Transplant, continued

33933	Backbench standard preparation of cadaver donor heart/lung allograft prior to transplantation, including dissection of allograft from surrounding soft tissues to prepare aorta, superior vena cava, inferior vena cava, and trachea for implantation
33935	Heart-lung transplant with recipient cardiectomy-pneumonectomy
33940	Donor cardiectomy (including cold preservation)
33944	Backbench standard preparation of cadaver donor heart allograft prior to transplantation, including dissection of allograft from surrounding soft tissues to prepare aorta, superior vena cava, inferior vena cava, pulmonary artery, and left atrium for implantation
33945	Heart transplant, with or without recipient cardiectomy

HCPCS CODES

S2054	Transplantation of multivisceral organs
S2055	Harvesting of donor multivisceral organs, with preparation and maintenance of allografts; from cadaver donor
S2060	Lobar lung transplantation
S2061	Donor lobectomy (lung) for transplantation, living donor
S2152	Solid organ(s), complete or segmental, single organ or combination of organs; deceased or living donor(s), procurement, transplantation, and related complications; including: drugs; supplies; hospitalization with outpatient follow-up; medical/surgical, diagnostic, emergency, and rehabilitative services; and the number of days of pre- and post-transplant care in the global definition

Key References

1. Chambers DC, Cherikh WS, Harhay MO et al. The International Thoracic Organ Transplant Registry of the International Society for Heart and Lung Transplantation: Thirty-sixth adult lung and heart-lung transplantation Report-2019; Focus theme: Donor and recipient size match. J Heart Lung Transplant. 2019;38(10):1042.
2. Doyle, RL & Nador, RG. Heart-lung transplantation. 2006. http://www.utdol.com/utd/content/topic.do?topicKey=hrt_tran/5634&type=A&selectedTitle=1~19. Date accessed: 9/15/06.

Select Health

Select Health

MEDICAL POLICY

IMPLANTABLE LOOP RECORDERS (ILR)

Policy # 131
Implementation Date: 7/15/03
Review Dates: 2/26/04, 1/13/05, 1/3/06, 2/16/07, 2/21/08, 2/19/09, 2/18/10, 2/17/11, 2/16/12, 4/25/13, 2/20/14, 3/19/15, 2/11/16, 2/16/17, 2/15/18, 2/5/19, 2/11/20, 2/18/21, 4/18/22, 4/12/23, 2/15/24, 3/23/25
Revision Dates:

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

An implantable loop recorder (ILR) is an implantable, self-sensing, and/or patient-activated monitoring system that records electrocardiography data. It is designed to diagnose a cardiac cause of syncope when the standard work-up, including non-invasive tests, is not diagnostic. Electrodes in the device sense the heart's activity through human tissue, so there is no need for any intravenous leads. When symptoms occur, the patient uses an external activator device to record electrocardiogram (ECG) data for analysis by a physician; the ILR has an autosensing feature that can be programmed to automatically begin recording when it senses an arrhythmia. The monitor can store up to 40 minutes of preceding signals after an episode of spontaneous syncope. The device is removed after the battery has failed, or earlier, if a definitive diagnosis has been established.

The ILR is implanted subcutaneously under local anesthetic in a single incision procedure in a left pectoral or mammary location. The projected life of the battery is approximately 24 to 36 months. The manufacturer recommends that the device be explanted (removed) when it is no longer clinically necessary, or when the battery is depleted. The electrodes are self-contained within the recorder. The ILR system consists of the following elements:

1. A subcutaneously placed, programmable cardiac event recorder with looping memory.
2. A handheld telemetry unit, which is used by the patient to activate electrocardiographic storage.
3. A programmer, which is used to program the event recorder and retrieve, display, and print stored data.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health covers implantable loop recorders.

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

Implantable Loop Recorder (ILR), continued

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

Syncope is the abrupt and transient loss of consciousness associated with absence of postural tone, followed by a rapid and usually complete recovery. This symptom is alarming for the individual, witnesses, family, and physicians. Although syncope can be a harbinger of a multitude of disease processes and can mimic the appearance of a cardiac arrest, it is most often benign and self-limited. Nevertheless, injuries associated with syncopal attacks occur in 35% of patients, and recurrent episodes can be psychologically devastating. In addition, syncope can be a premonitory sign of sudden cardiac death, especially in patients with organic heart disease.

The common pathway of all forms of syncope is a sudden decrease or brief cessation of cerebral blood flow. The causes can be classified into 7 main groups: unknown origin (estimated 34% of total), neurally-mediated (24%), cardiac (18%), neurological (10%), orthostatic hypotension (8%), medication-related (3%), and psychiatric disorders (2%). Since a syncopal episode can occur without warning, it sometimes causes injuries due to falls and other accidents.

Syncope is common and accounts for around 6% of all hospital admissions and 3% of emergency room visits. Forty-three percent of patients experience recurrent syncope with an approximate 5% first year mortality in patients with unexplained syncope. Moreover, it does not always have a benign course, with mortality rates up to 33% at one year in patients with a structural cardiac disorder. In addition, costs for investigation for syncope ("diagnostic odysseys") are substantial, and about 25% of all patients remain undiagnosed.

A major problem in the diagnosis of the underlying cause is that syncope is a transient symptom and not a disease. Typically, patients are asymptomatic at the time of assessment, and thus, the likelihood of capturing a spontaneous event during diagnostic testing is low. The cause of syncope is not determined after history, physical examination, or surface ECG, in 38−47%% of patients. Even after further referral for tilt-table and electrophysiological testing, 10%−26% of patients will remain undiagnosed. Other tests that can be employed include 24−48-hour halter or prolonged cardiac monitoring.

Ambulatory event monitors (AEMs) were developed to provide longer periods of monitoring and may be useful when the initial evaluation is non-diagnostic or when symptoms are infrequent. Ambulatory event monitors (AEMs) are intermittent recorders that can be used for longer periods (weeks to months) of monitoring to provide briefer, intermittent recordings to investigate events that occur infrequently. Intermittent recorders can be worn continuously or be attached by the patient when symptoms occur. They are activated after the onset of symptoms. Some recorders are implanted under the skin for long-term recordings (Medtronic's Reveal implantable loop recorder). Ambulatory event monitors are useful if symptoms are quite brief, or if symptoms include only very brief or no patient incapacitations, so that the patient, or a companion, can activate the recorder. These devices are often capable of downloading data trans-telephonically.

There are several types of AEMs available:

1. Noncontinuous devices with memory. These devices are carried by the patient and applied to the precordial area when symptoms occur (e.g., HeartCard Cardiac Event Recorder). The limitations are that an arrhythmia may be of short duration and not captured by the device or the patient may be incapacitated and unable to apply the device while symptomatic.
2. Continuous memory loop devices. These devices are worn continuously and can continuously store EKG data so that when symptoms occur, the patient activates the device, and the EKG is recorded from the memory loop for the preceding 30−90 seconds and approximately 1 minute after.

Implantable Loop Recorder (ILR), continued

3. Implantable continuous memory loop devices. These devices are inserted under the skin in the chest area during an outpatient surgical procedure. When symptoms occur, the patient activates the handheld activator over the recorder to activate the storage of cardiac rhythms; the newest generation of this device (Medtronic's Reveal Plus) also has an autosensing feature. The device may be used for more than 1 year's duration and has a projected battery life of 14 months, at which time the device must be surgically removed.

Current published evidence consists of numerous case series and studies and several controlled trials; and at least 1 economic study. Additionally, there is an abstract of a relatively large (n = 201) randomized trial (EaSyAS) reported at the European Society of Cardiology annual meeting in 2002.

Except for the EaSyAS trial, virtually all the biggest available trials and several smaller series, including the economic trial, were sponsored by the manufacturer (Medtronic) and the authors are part of the (Medtronic) Reveal Investigators group. Additionally, these studies are not without flaws. Both the RCTs and case studies consistently demonstrate substantial improvements in diagnostic yield (i.e., of recurrent, unexplained syncope), in an assortment of patient populations. However, both the Krahn randomized trial and the EaSyAS trial demonstrated no difference in patient outcomes and the EaSyAS trial also demonstrated increased treatment costs (without a significant difference in outcomes). Krahn et al. demonstrated dramatic improvements in costs per diagnosis; however, this trial must be interpreted with caution due to the authors' affiliations with Medtronic. The EaSyAS trial demonstrated increased costs for treatment in the ILR group vs. conventional work-up of a recurrent, unexplained syncope population. Thus, there are probable substantial improvements in diagnostic yield, possible reductions in diagnostic costs, but with ill-defined patient populations and little direct evidence of improved patient outcomes; and some evidence of increased costs for treatment (without improvements in patient outcomes). Many questions remain, however, about just who these patients are, and which of them would benefit from a diagnosis. As Noll stated in Internal Medicine News: "We have a diagnosis in these patients, but we are not able to treat them appropriately. This raises the question, then, of whether we really need to diagnose these patients if there are no good methods to influence outcome."

Billing/Coding Information

CPT CODES

33282	Implantation of patient-activated cardiac event recorder
33284	Removal of an implantable, patient-activated cardiac event recorder
93285	Programming device evaluation (in person) with iterative adjustment of the implantable device to test the function of the device and select optimal permanent programmed values with physician analysis, review and report; implantable loop recorder system
93291	Interrogation device evaluation (in person) with physician analysis, review and report, includes connection, recording and disconnection per patient encounter; implantable loop recorder system, including heart rhythm derived data analysis
93298	Interrogation device evaluation(s), (remote) up to 30 days; implantable loop recorder system, including analysis of recorded heart rhythm data, physician analysis, review(s) and report(s)
93299	Interrogation device evaluation(s), (remote) up to 30 days; implantable cardiovascular monitor system or implantable loop recorder system, remote data acquisition(s), receipt of transmissions and technician review, technical support and distribution of results

HCPCS CODES

C1764	Event recorder, cardiac (implantable)
E0616	Implantable cardiac event recorder with memory, activator, and programmer

Implantable Loop Recorder (ILR), continued

Key References

1. Armstrong VL, Lawson J, Kamper AM, Newton J, Kenny RA. The use of an implantable loop recorder in the investigation of unexplained syncope in older people. Age Ageing. 2003 Mar;32(2):185-8. PMID: 12615562
2. Ashby DT, Cehic DA, Disney PJ, Mahar LJ, Young GD. A retrospective case study to assess the value of the implantable loop recorder for the investigation of undiagnosed syncope. Pacing Clin Electrophysiol. 2002 Aug;25(8):1200-5. PMID: 12358170
3. Chrysostomakis SI, Klapsinos NC, Simantirakis EN, Marketou ME, Kambouraki DC, Vardas PE. Sensing issues related to the clinical use of implantable loop recorders. Europace. 2003 Apr;5(2):143-8. PMID: 12633638
4. Connolly SJ, Sheldon R, Thorpe KE, Roberts RS, Ellenbogen KA, Wilkoff BL, Morillo C, Gent M; VPS II Investigators. Pacemaker therapy for prevention of syncope in patients with recurrent severe vasovagal syncope: Second Vasovagal Pacemaker Study (VPS II): a randomized trial. JAMA. 2003 5/7;289(17):2224-9. PMID: 12734133
5. Flevari P, Livanis EG, Theodorakis GN, Zarvalis E, Mesiskli T, Kremastinos DT. Vasovagal syncope: a prospective, randomized, crossover evaluation of the effect of propranolol, nadolol and placebo on syncope recurrence and patients' well-being. J Am Coll Cardiol. 2002 Aug 7;40(3):499-504. PMID: 12142117
6. Giancaterino, S., Florentino, L., Nishimura, M., & Hsu, J. (2018). Current and Future Use of Insertable Cardiac Monitors. *J Am Coll Cardiol EP*, 4:1383–1386.
7. Huikuri HV, Mahaux V, Bloch-Thomsen PE. Cardiac arrhythmias and risk stratification after myocardial infarction: results of the CARISMA pilot study. Pacing Clin Electrophysiol. 2003 Jan;26(1 Pt 2):416-9. PMID: 12687857
8. Kenny RA. SAFE PACE 2: Syncope and Falls in the Elderly--Pacing and Carotid Sinus Evaluation: a randomized controlled trial of cardiac pacing in older patients with falls and carotid sinus hypersensitivity. Europace. 1999 Jan;1(1):69-72. PMID: 11220545
9. Krahn AD, Klein GJ, Yee R, Norris C. Final results from a pilot study with an implantable loop recorder to determine the etiology of syncope in patients with negative noninvasive and invasive testing. Am J Cardiol. 1998 Jul 1;82(1):117-9. PMID: 9671019
10. Krahn AD, Klein GJ, Yee R, Manda V. The high cost of syncope: cost implications of a new insertable loop recorder in the investigation of recurrent syncope. Am Heart J. 1999 May;137(5):870-7. PMID: 10220636
11. Krahn AD, Klein GJ, Yee R, Takle-Newhouse T, Norris C. Use of an extended monitoring strategy in patients with problematic syncope. Reveal Investigators. Circulation. 1999 Jan 26;99(3):406-10. PMID: 9918528
12. Krahn AD, Klein GJ, Yee R, Skanes AC. Randomized assessment of syncope trial: conventional diagnostic testing versus a prolonged monitoring strategy. Circulation. 2001 Jul 3;104(1):46-51. PMID: 11435336
13. Krahn AD, Klein GJ, Yee R, Skanes AC; REVEAL Investigators. Predictive value of presyncope in patients monitored for assessment of syncope. Am Heart J. 2001 May;141(5):817-21. PMID: 11320372
14. Krahn AD, Klein GJ, Fitzpatrick A, Seidl K, Zaidi A, Skanes A, Yee R. Predicting the outcome of patients with unexplained syncope undergoing prolonged monitoring. Pacing Clin Electrophysiol. 2002 Jan;25(1):37-41. PMID: 11877934
15. Mieszczanska H, Ibrahim B, Cohen TJ. Initial clinical experience with implantable loop recorders. J Invasive Cardiol. 2001 Dec;13(12):802-4. PMID: 11731693
16. National Horizon Scanning Centre report, The University of Birmingham, UK. Insertable Loop Recorders for the Diagnosis of Syncope. 1/2002.
17. Nierop PR, van Mechelen R, van Elsacker A, Luijten RH, Elhendy A. Heart rhythm during syncope and presyncope: results of implantable loop recorders. Pacing Clin Electrophysiol. 2000 Oct;23(10 Pt 1):1532-8. PMID: 11060875
18. Paylos JM, Aguilar Torresa R. [Usefulness of the implantable subcutaneous recorder in the diagnosis of recurrent syncope of unknown etiology in patients without structural heart disease and negative tilt test and electrophysiological study] Rev Esp Cardiol. 2001 Apr;54(4):431-42. Spanish. Erratum in: Rev Esp Cardiol 2001 Jul;54(7):929. PMID: 11282048
19. Roche F, Gaspoz JM, Pichot V, Costes F, Isaaz K, Ferron C, Roche C, Geyssant A, Lacour JR, Barthelemy JC. Accuracy of an automatic and patient-triggered long-term solid memory ambulatory cardiac event recorder. Am J Cardiol. 1997 Oct 15;80(8):1095-8. PMID: 9352989
20. Seidl K, Rameken M, Breunung S, Senges J, Jung W, Andresen D, van Toor A, Krahn AD, Klein GJ; Reveal-Investigators. Diagnostic assessment of recurrent unexplained syncope with a new subcutaneously implantable loop recorder. Europace. 2000 Jul;2(3):256-62. PMID: 11227598
21. Silva, J., Bromberg, B., Emge, F., Bowman, T., & VanHare, G. (2016). Implantable Loop Recorder Monitoring for Refining Management of Children With Inherited Arhythmia Syndromes. *J Am Heart Assoc*, 5: e003632. doi:10.1161/JAHA.116.003632
22. Sulke N. Eastbourne Syncope Assessment Study (EASYAS). European Society of Cardiology XXIV Annual Meeting. Berlin, 8/31 - 9/4/02

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

INTIMA-MEDIA THICKNESS (IMT) TESTING FOR THE ASSESSMENT OF HEART DISEASE RISK (HEART SCAN)

Policy # 238
Implementation Date: 9/14/04
Review Dates: 10/18/05, 10/19/06, 10/18/07, 12/18/08, 12/17/09, 12/16/10, 12/15/11, 7/18/13, 6/19/14, 6/11/15, 6/16/16, 6/15/17, 7/20/18, 6/13/19, 6/18/20, 6/17/21, 5/19/22, 6/15/23, 6/19/24
Revision Dates:

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Intima-media testing involves the application of B-mode ultrasound to the carotid artery to determine quantitative arterial intima-media wall thickness (IMT) of the artery. This is used as a surrogate to estimate intima-media wall-thickening in the coronary arteries. A relationship between common carotid artery intima-media thickness and angiographic presence and extent of coronary artery disease has been reported in several studies. These studies suggest that patients at high risk for an adverse coronary event can be identified early, before the patient is symptomatic, through use of these surrogate markers. Data from a National Heart Lung and Blood Institute study indicates that carotid arterial IMT incorporates additional, independent information on prediction of coronary events beyond angiographic measurements of lumen narrowing.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health does NOT cover intima-media thickness (IMT) testing to assess cardiovascular risk in any population. This meets the plan's definition of experimental/investigational.

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Intima Media Thickness (IMT) Testing for the Assessment of Heart Disease Risk (Heart Scan), continued

Summary of Medical Information

The current state of the literature has clearly established that carotid IMT, with its numerous methods and sub sites (e.g., common, internal, bifurcation, maximal, mean/average), is highly correlated with a variety of traditional risk factors for coronary artery disease (CAD) as well as for CAD-related events (i.e., MI, stroke). The evidence supporting the belief that carotid IMT provides additional predictive capability beyond traditional risk factors is mixed; however, the weight of the evidence seems to suggest that indeed carotid IMT does provide some additional predictive capability beyond traditional risk factor assessment. One report suggests that IMT is equal in weight/value to nine separate traditional risk factors.

However, the predictive ability of carotid IMT seems to vary widely between subpopulations of people at elevated risk for CAD without a history of CAD-related events as a function of age, gender, and the presence and degree of other risk factors. Additionally, it is not clear whether the additional predictive capacity of carotid IMT would persist when adjusted for the entire risk factor profile now being proposed by Intermountain's Cardiovascular Services group (which includes c-reactive protein and serum homocysteine in addition to the more traditional Framingham risk factors). To date, there have been no studies that have prospectively followed appropriate patient groups to determine whether the addition of carotid IMT to a standard and complete battery of risk factors (comparable to the profile now being advocated by Intermountain's Cardiovascular Services group) changes treatment decisions and/or leads to meaningful improvements in patient outcomes.

Current literature evaluates IMT as a risk factor. However, no studies are available to assess IMT and treatment outcomes to reduce coronary cerebrovascular events or death from atherosclerosis.

Additional concerns/issues include:

- IMT "… is complex and expensive …" (Nichols et al.,1999)
- "The differences in carotid-artery intima-media thickness between patients at high risk and those at low risk are too small for common clinical use (Nichols et al., 1999)."
- There seems to be consensus suggesting that carotid IMT is an early-to-intermediate or late-intermediate marker when atherosclerosis is still limited to the arterial wall, whereas coronary angiography measures atherosclerosis in its late stages. Traditional risk factors generally are measures (albeit indirect and sometimes weak) of the numerous factors at play throughout the process but their predictive capacity may diminish with age and advanced disease. Thus, the value of carotid IMT may be at its highest during the early-to-late "intermediate" period of atherosclerosis and less useful in the earliest and possibly latest stages of disease.
- "The association between IMT and risk of MI did not show a clearly linear pattern (Bots et al., Circulation 1997)." Thus, use of a linear algorithm may not be valid for this indication.
- IMT measurement protocols have yet to be standardized and concerns remain about quality control of IMT facilities outside of tightly-controlled clinical trials.
- Atherosclerosis is a focal, patchy disease, thus, measures of carotid IMT may not reflect the process in other vessels.
- "Whether increased common carotid intima-media thickness itself reflects local atherosclerosis is still a subject of debate. It may merely reflect an adaptive response of the vessel wall to changes in sheer stress, tensile stress, and blood flow and subsequent changes in lumen diameter (Bots et al., Circulation 1997)."
- "We believe that the primary ill effect is not the increase in carotid-artery thickness in itself, but rather the increase in the stiffness of the vessel that results from increased wall thickness and content (Nichols et al., 1999)."

Billing/Coding Information

Not covered for the indications listed above

CPT CODES

93998	Unlisted noninvasive vascular diagnostic study

INtima Media Thickness (IMT) Testing for the Assessment of Heart Disease Risk (Heart Scan), continued

HCPCS CODES

No specific codes identified

Key References

1. American Heart Association Conference Proceedings--Beyond Secondary Prevention: Identifying the High-Risk Patient for Primary Prevention, published in Circulation/00.
2. Bots ML, Hoes AW, Koudstaal PJ, Hofman A, Grobbee DE. Common carotid intima-media thickness and risk of stroke and myocardial infarction: the Rotterdam Study. Circulation. 1997 Sep 2;96(5):1432-7.
3. Bots ML, Hofman A, Grobbee DE. Increased common carotid intima-media thickness. Adaptive response or a reflection of atherosclerosis? Findings from the Rotterdam Study. Stroke. 1997 Dec;28(12):2442-7.
4. Chambless L, Heiss G, Folsom A, Rosamond W, Szklo M, et al. Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: The Atherosclerosis Risk in Communities (ARIC) Study/87 - 1993. Amer J Epidemiol 1997; 146:483-94.
5. Chambless LE, Folsom AR, Davis V, Sharrett R, Heiss G, Sorlie P, Szklo M, Howard G, Evans GW. Risk factors for progression of common carotid atherosclerosis: the Atherosclerosis Risk in Communities Study/87-1998. Am J Epidemiol. 2002 Jan 1;155(1):38-47.
6. del Sol AI, Moons KG, Hollander M, Hofman A, Koudstaal PJ, Grobbee DE, Breteler MM, Witteman JC, Bots ML. Is carotid intima-media thickness useful in cardiovascular disease risk assessment? The Rotterdam Study. Stroke. 2001 Jul;32(7):1532-8.
7. Hodis H, Mack W, et al. The role of carotid-arterial intima-media thickness in predicting clinical coronary events. Annals of Internal Medicine 1998; 128: 262-269.
8. Iglesias del Sol A, Bots ML, Grobbee DE, Hofman A, Witteman JC. Carotid intima-media thickness at different sites: relation to incident myocardial infarction. The Rotterdam Study. Eur Heart J. 2002 Jun;23(12):934-40.
9. Kastelein JJ, Bots, MLD et al. Statin therapy with ezetimibe or niacin in high-risk patients. N Engl Med. 2009 Nov 26;361(22)2180-3.
10. Nichols WW, Pepine CJ, O'Rourke MF. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke. N Engl J Med. 1999 Jun 3;340(22):1762-3.
11. O'Leary D, Polack J, Kronmal R, Manolio T, Burke G, et al. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. N Engl Med Jan 1999;340: 14-22.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

INTRAVASCULAR LITHOTRIPSY (IVL)

Policy # 647
Implementation Date:4/23/21
Review Dates: 5/5/22, 4/20/23, 4/4/24, 4/17/25
Revision Dates: 5/11/23

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description
Intravascular lithotripsy (IVL) is a technology derived from renal lithotripsy, in which multiple emitters mounted on a traditional balloon catheter provide circumferential pulsatile energy to disrupt calcified plaque and improve acute gain while minimizing vessel injury. Over the last 40 years, despite multiple advancements in percutaneous coronary interventions, calcifed lesions remain a challenge for even the most experienced operators, leading to an increase in morbidity and mortality. Most recently, intravascular lithotripsy (IVL) has been shown to be an innovative technology that is designed to address heavily calcifed lesions.

The Shockwave Medical Peripheral IVL System (Shockwave Medical, Fremont, CA) received U.S. Food and Drug Administration (FDA) approval in 2016. This system is a single-use sterile disposable catheter that contains multiple lithotripsy emitters enclosed in an integrated balloon. The emitters create sonic pressure waves in the shape of a sphere, creating a field effect to treat circumferential vascular calcium. These sonic pressure waves selectively disrupt and fracture calcium in situ, altering vessel compliance, while minimizing injury and maintaining the integrity of the fibro-elastic components of the vessel wall.

In addition, Shockwave C2 coronary intravascular Lithotripsy (IVL) catheter has been FDA approved in 2021, for the dilation of a severely stenotic and heavily calcified coronary lesion; its technology is the same as the peripheral IVL system.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers intravascular lithotripsy (IVL) for patients undergoing transfemoral aortic valve replacement (TAVR) with calcified peripheral arterial disease.

Select Health covers IVL for patients undergoing percutaneous coronary intervention with stenotic and heavily calcified coronary lesions.

All other indications utilizing IVL are considered experimental/investigational.

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the

Intravascular Lithotripsy (IVL), continued

Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

IVL, by disrupting intimal and medial calcification, alters vessel compliance to allow for the safe passage of large-bore delivery sheaths. This expands the patient cohort that could be eligible for transfemoral access for TAVR procedures. IVL-enabled transfemoral access offers several advantages. First, it preserves the established benefits of TAVR: decreased morbidity and mortality, fewer hospital days, and reduced cost. Second, although alternative access options exist, they are more invasive and have a significant learning curve (1,5). IVL leverages the familiarity of a balloon-based intervention, minimizing the learning curve, regardless of a center's volume.

IVL may represent a straightforward technique to preserve the benefits of reduced morbidity and mortality of transfemoral TAVR in patients with calcified peripheral arterial disease. IVL in the coronary artery is based on the same technology, as above, but is applied by a specific catheter designed for the coronary artery and is designed to dilate the recalcitrant coronary lesion prior to stenting.

Billing/Coding Information

CPT CODES

C9764 Revascularization, endovascular, open or percutaneous, lower extremity artery(ies), except tibial/peroneal; with intravascular lithotripsy, includes angioplasty within the same vessel(s), when performed

C9765 Revascularization, endovascular, open or percutaneous, lower extremity artery(ies), except tibial/peroneal; with intravascular lithotripsy, and transluminal stent placement(s), includes angioplasty within the same vessel(s), when performed

C9766 Revascularization, endovascular, open or percutaneous, lower extremity artery(ies), except tibial/peroneal; with intravascular lithotripsy and atherectomy, includes angioplasty within the same vessel(s), when performed

C9767 Revascularization, endovascular, open or percutaneous, lower extremity artery(ies), except tibial/peroneal; with intravascular lithotripsy and transluminal stent placement(s), and atherectomy, includes angioplasty within the same vessel(s), when performed

C9772 Revascularization, endovascular, open or percutaneous, tibial/peroneal artery(ies), with intravascular lithotripsy, includes angioplasty within the same vessel(s), when performed

C9773 Revascularization, endovascular, open or percutaneous, tibial/peroneal artery(ies); with intravascular lithotripsy, and transluminal stent placement(s), includes angioplasty within the same vessel(s), when performed

C9774 Revascularization, endovascular, open or percutaneous, tibial/peroneal artery(ies); with intravascular lithotripsy and atherectomy, includes angioplasty within the same vessel(s), when performed

C9775 Revascularization, endovascular, open or percutaneous, tibial/peroneal artery(ies); with the

Intravascular Lithotripsy (IVL), continued

intravascular lithotripsy and transluminal stent placement(s), and atherectomy, includes angioplasty within same vessel(s), when performed

0715T Percutaneous transluminal coronary lithotripsy (list separately in addition the code for the primary procedure)

Key References

1. Aksoy, A., et al. Intravascular Lithotripsy in Calcified Coronary Lesions. (2019). https://doi.org/10.1161/CIRCINTERVENTIONS.119.008154 PMID: 31707803
2. Di Mario, C., et al. A Prospective Registry of Intravascular Lithotripsy-Enabled Vascular Access for Transfemoral Transcatheter Aortic Valve Replacement. *JACC: Cardiovascular Interventions*. (2019). 12(5). doi: 10.1016/j.jcin.2019.01.211
3. FDA approval letter: https://www.accessdata.fda.gov/cdrh_docs/pdf20/P200039A.pdf
4. Safety and efficacy data: https://www.accessdata.fda.gov/cdrh_docs/pdf20/P200039B.pdf
5. Shockwave IVL coronary catheter indication for use: https://www.accessdata.fda.gov/cdrh_docs/pdf20/P200039C.pdf.

Revision History

Revision Date	Summary of Changes
5/11/23	For Commercial Plan Policy, added language regarding an additional qualifying option for this treatment: **"Select Health covers IVL for patients undergoing percutaneous coronary intervention with stenotic and heavily calcified coronary lesions."**

Select Health

Select Health

MEDICAL POLICY

LDL APHERESIS (LIPOSORBER DEVICE, HELP SYSTEM)

Policy # 207

Implementation Date:12/9/03

Review Dates: 2/9/05, 2/16/06, 5/17/07, 4/24/08, 4/23/09, 4/12/12, 6/20/13, 4/17/14, 5/7/15, 4/14/16, 4/27/17, 8/16/18, 4/27/19, 4/15/20, 4/15/21, 3/18/22, 4/18/23, 4/16/24, 4/2/25

Revision Dates: 2/18/10, 4/21/11, 8/19/15, 5/9/19, 7/12/23, 5/1/25

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

Heart disease is the number one cause of morbidity and mortality in the adult US population. Lowering total and LDL cholesterol has been shown to reduce the development and recurrence of heart disease. Consequently, great emphasis is placed on the treatment of elevated total and LDL cholesterol by many physicians. Treatment including diet and multiple medications are used to reach goals published by US multinational cardiovascular society collaborations with approval through the NHLBI (National Heart, Lung, and Blood Institute). However, some patients, primarily those with familial hypercholesterolemia (FH), may require additional therapy.

The dextran sulfate cellulose adsorption system (LipoSorber) uses columns containing dextran sulfate immobilized on cellulose beads to remove LDL from the plasma. Typically, there are 2 such columns in the plasma circuit, which are regenerated automatically during LDL apheresis. The system has a high selectivity for the removal of apolipoprotein B (apo B)-containing lipoproteins, removing LDL, very low-density lipoprotein, and lipoprotein (a). Binding of LDL appears to depend on electrostatic interaction between dextran sulfate and apo B and is inhibited by acetylation of LDL. Interestingly, dextran sulfate columns can remove LDL of patients with familial defective apo B-100 with equal efficacy. Although some molecules with similar properties are absorbed into the column, this is not associated with any adverse clinical consequences. The columns are discarded after each apheresis procedure, and this contributes to the high running costs of this system.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers LDL apheresis in *limited* circumstances when certain criteria have been met.

A. Patients must meet ALL the following criteria (1−5):

1. Patient has one of the following diagnoses and associated LDL cholesterol levels:
   a) Functional homozygous FH patients with LDL-C ≥ 300 mg/dL (or non-high density lipoprotein cholesterol [HDL-C] 330 mg/dL); or
   b) Functional heterozygous FH patients with LDL-C ≥ 300 mg/dL (or non-HDL cholesterol 330 mg/dL and 0 to 1 risk factors); or

LDL Apheresis (Liposorber Device, Help System), continued

c) Functional heterozygous FH patients with LDL-C ≥ 200 mg/dL (or non-HDL cholesterol 230 mg/dL) and high-risk characteristics such as ≥ 2 risk factors or high lipoprotein (a) ≥ 50 mg/dL using an isoform insensitive assay; or

d) Functional heterozygous FH patients with LDL-C ≥ 160 mg/dL* (or non-HDL cholesterol ≥ 190 mg/dL) and very high-risk characteristics, such as established coronary heart disease or other cardiovascular disease.

AND

2. Patient has documented medical nutritional therapy consultation and has shown active efforts at appropriate lifestyle modifications.

AND

3. Supporting documentation demonstrates the patient to have attempted or is currently on maximal medical therapy. Maximal medical therapy is defined as the following:
4. Maximal doses, for at least 6 months, of high-potency HMG-CoA reductase inhibitor (statin) therapy (unless intolerant, complete or incomplete; or detrimental side effects are documented) with ezetimibe or bempedoic acid, and a PCSK9 interfering agent, with compliance to that therapy (unless intolerant or detrimental side effects are documented).

AND

5. Absence of other severe co-morbid conditions such as end-stage renal disease, emphysema, or other life-threatening conditions.

AND

6. LDL apheresis is to be performed with an FDA approved device.

*Patients with familial heterozygous traits with LDL cholesterol ≥ 160 must have documented atherosclerosis. The patient should have known coronary artery disease, cerebrovascular disease, or peripheral vascular disease documented prior to apheresis therapy.

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

The evidence permits the conclusion that lipid apheresis consistently reduces total cholesterol and LDL cholesterol. Three randomized, controlled trials (RCTs) report that the reductions in LDL cholesterol are clinically and statistically significantly greater than those achieved by medication alone for patients with refractory hypercholesterolemia. The evidence suggests that lipid apheresis may reduce arterial morphologic change and improve hemodynamic flow in diseased arteries; however, this has not been consistently demonstrated in RCTs. The RCTs are insufficient to provide direct evidence that lipid apheresis reduces adverse cardiovascular events as compared to maximal medical management. The results of these RCTs are also limited by the fact that 2 of the 3 trials included some patients who may have been responsive to medical management, rather than limiting the population to those patients who are truly refractory to maximal medical management.

LDL Apheresis (Liposorber Device, Help System), continued

In most of the nonrandomized studies, the patients had failed diet and drug therapy, and correspond more closely to the target group of refractory patients. The efficacy of LDL lowering is of similar magnitude in these studies as compared to the RCTs. In uncontrolled studies using pre-and post-treatment coronary angiography, the reductions in cholesterol appear to be associated with non-progression of atherosclerosis. Therefore, the evidence supports the effectiveness of lipid apheresis for providing a long-term lipid-lowering benefit and suggests that lipid apheresis may be associated with non-progression of coronary artery disease. Given the established causal relationship between LDL cholesterol and cardiac events, it is likely that lipid apheresis will reduce cardiovascular events for patients with hypercholesterolemia who are refractory or intolerant to maximal drug therapy.

Lipid apheresis improves health outcomes by lowering cholesterol in patients with refractory hypercholesterolemia, leading to a reduction in adverse cardiovascular events. However, the available evidence has not quantified the magnitude of this benefit. Given that the magnitude of LDL lowering by apheresis is large, 50%−70% or greater, the magnitude of reduction in adverse cardiovascular events is also likely to be clinically meaningful.

Lipid apheresis is a relatively safe treatment, although it occasionally may be associated with significant hypotension and anaphylactic reactions. Most adverse effects are minor. Hypotension may be more likely with dextran sulfate chemo adsorption, especially in patients receiving concomitant angiotensin-converting enzyme (ACE) inhibitors. There are no adequate trials of direct comparison of the techniques to quantitatively determine treatment or safety advantages. All 3 techniques are relatively selective for LDL cholesterol but may remove some other molecules non-selectively. The clinical effect of removal of other factors such as HDL, fibrinogen, and bradykinin are unclear at this time, but there is no evidence to suggest that adverse effects have resulted from this phenomenon.

For the indicated patient group with refractory hypercholesterolemia despite maximal medical therapy, the alternatives are very limited. Radical treatments such as portocaval shunt or liver transplantation have been tried but are not yet established in clinical practice. Intensification of medical therapy is an option as newer and more potent cholesterol lowering agents are introduced, but only a minority of patients will remain refractory. Lipid apheresis is likely the best option for many of these patients.

Billing/Coding Information
Covered: For the conditions outlined above

CPT CODES

0342T	Therapeutic apheresis with selective HDL delipidation and plasma reinfusion
36516	Therapeutic apheresis; with extracorporeal selective adsorption or selective filtration and plasma reinfusion

HCPCS CODES

S2120	Low density lipoprotein (LDL) apheresis using heparin-induced extracorporeal LDL precipitation
E88.89	Other specified metabolic disorders

Key References

1. Bambauer R, Schiel R, Latza R, Klinkmann J, Schneidewind JM. LDL apheresis in clinical practice: long-term treatment of severe hyperlipidemia. Ther Apher. 1997 Feb;1(1):49-54. PMID: 10225781
2. Bambauer R, Schneidewind JM, Latza R. Apheresis technologies for prevention and regression of atherosclerosis: clinical results. ASAIO J. 1999 Sep-Oct;45(5):403-7. PMID: 10503615
3. Bambauer R, Schiel R, Latza R. Low-density lipoprotein apheresis: an overview. Therap Apher Dial. 2003 Aug;7(4):382-90.
4. BCBS TEC — Lipid apheresis in the treatment of patients with severe, refractory hypercholesterolemia. 4/1999.
5. Borberg H. Results of an open, longitudinal multicenter LDL-apheresis trial. Transfus Sci. 1999 Feb;20(1):83-94. PMID: 10621566
6. Jovin IS, Taborski U, Muller-Berghaus G. Analysis of the long-term efficacy and selectivity of immunoadsorption columns for low density lipoprotein apheresis. ASAIO J. 2000 May-Jun;46(3):298-300. PMID: 10826740
7. Kereiakes DJ, Robinson JG, Cannon CP, Lorenzato C, Pordy R, Chaudhari U, Colhoun HM. Efficacy and safety of the proprotein convertase subtilisin/kexin type 9 inhibitor alirocumab among high cardiovascular risk patients on maximally tolerated statin therapy: The ODYSSEY COMBO I study. Am Heart J. 2015 Jun;169(6):906-915.e13. doi: 10.1016/j.ahj.2015.03.004. Epub 2015 Mar 13.

Acute Inpatient Rehabilitation LDL Apheresis (Liposorber Device, Help System), continued

8. Mabuchi H, Koizumi J, Shimizu M, Kajinami K, Miyamoto S, Ueda K, Takegoshi T. Long-term efficacy of low-density lipoprotein apheresis on coronary heart disease in familial hypercholesterolemia. Hokuriku-FH-LDL-Apheresis Study Group. Am J Cardiol. 1998 Dec 15;82(12):1489-95. PMID: 9874053
9. Matsuzaki M, Hiramori K, Imaizumi T, Kitabatake A, Hishida H, Nomura M, Fujii T, Sakuma I, Fukami K, Honda T, Ogawa H, Yamagishi M. Intravascular ultrasound evaluation of coronary plaque regression by low density lipoprotein-apheresis in familial hypercholesterolemia: the Low Density Lipoprotein-Apheresis Coronary Morphology and Reserve Trial (LACMART). J Am Coll Cardiol. 2002 Jul 17;40(2):220-7. PMID: 12106923
10. Nishimura S, Sekiguchi M, Kano T, Ishiwata S, Nagasaki F, Nishide T, Okimoto T, Kutsumi Y, Kuwabara Y, Takatsu F, Nishikawa H, Daida H, Yamaguchi H. Effects of intensive lipid lowering by low-density lipoprotein apheresis on regression of coronary atherosclerosis in patients with familial hypercholesterolemia: Japan Low-density Lipoprotein Apheresis Coronary Atherosclerosis Prospective Study (L-CAPS). Atherosclerosis. 1999 Jun;144(2):409-17. PMID: 10407502
11. Robinson JG, Colhoun HM, Bays HE, Jones PH, Du Y, Hanotin C, Donahue S. Efficacy and safety of alirocumab as add-on therapy in high-cardiovascular-risk patients with hypercholesterolemia not adequately controlled with atorvastatin (20 or 40 mg) or rosuvastatin (10 or 20 mg): design and rationale of the ODYSSEY OPTIONS Studies. Clin Cardiol. 2014 Oct;37(10):597-604. doi: 10.1002/clc.22327. Epub 2014 Sep 30.
12. Robinson JG, Farnier M, Krempf M, Bergeron J, Luc G, Averna M, Stroes ES, Langslet G, Raal FJ, El Shahawy M, Koren MJ, Lepor NE, Lorenzato C, Pordy R, Chaudhari U, Kastelein JJ; ODYSSEY LONG TERM Investigators. Efficacy and safety of alirocumab in reducing lipids and cardiovascular events. N Engl J Med. 2015 Apr 16;372(16):1489-99. doi: 10.1056/NEJMoa1501031. Epub 2015 Mar 15. PMID:25773378
13. Rosenson, R.S., de Ferranti, S.D., & Durrington, P. Treatment of drug-resistant hypercholesterolemia. *UpToDate*. June 2023.
14. Thompson GR. LDL apheresis. Atherosclerosis. 2003 Mar;167(1):1-13. Review.

Revision History

Revision Date	Summary of Changes
7/12/23	For Commercial Plan Policy, updated diagnoses and associated LDL cholesterol levels in criteria section #1 to align with current clinical guidelines: "a) Functional homozygous FH patients with LDL-C ≥300 mg/dL (or non-high density lipoprotein cholesterol [HDL-C] 330 mg/dL). b) Functional heterozygous FH patients with LDL-C ≥300 mg/dL (or non-HDL cholesterol 330 mg/dL and 0 to 1 risk factors). c) Functional heterozygous FH patients with LDL-C ≥200 mg/dL (or non-HDL cholesterol 230 mg/dL) and high-risk characteristics such as ≥2 risk factors or high lipoprotein (a) ≥50 mg/dL using an isoform insensitive assay. d) Functional heterozygous FH patients with LDL-C ≥160 mg/dL (or non-HDL cholesterol 190 mg/dL) and very high-risk characteristics such as established coronary heart disease, other cardiovascular disease."
5/1/25	For Commercial Plan Policy, modified requirements in criterion #A-4: "Maximal doses, for at least 6 months, of high-potency HMG-CoA reductase inhibitor (statin) therapy (unless intolerant, ***complete or incomplete***; or detrimental side effects are documented) with ezetimibe ***or bempedoic acid***, and a ***PCSK9 interfering agent***, with compliance to that therapy (***unless intolerant or detrimental side effects are documented***)."

Select Health

Select Health

MEDICAL POLICY

LEADLESS PACEMAKERS

Policy # 670
Implementation Date: 8/21/23
Review Dates: 8/11/24
Revision Dates:11/20/23, 8/23/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Bradycardia (defined as resting heart rate < 60 beats per minute [bpm] on electrocardiography [ECG]) due to AV node dysfunction or sinus node dysfunction (SND), commonly called sick sinus syndrome, may result in hemodynamic consequences. A permanent cardiac pacemaker (PM) is the definitive treatment for symptomatic bradycardia that is not caused by underlying disorders or medication. More than 1 million PMs are implanted annually worldwide, with 200,000 implanted in the United States each year. Up to 12% of patients with transvenous PMs experience a complication, including lead- and pocket-related events, within 2 months of implantation, and nearly 20% of patients experience a complication within 5 years. The transvenous leads are the most common source of complication, removal, and revision surgery.

The Micra transcatheter pacing system (TPS) (Medtronic) is a single-chamber right ventricular pacing device. The device senses electrical activity of the heart via electrodes within the device's titanium capsule. Heart rhythm is monitored for bradycardia. Rate-adaptive pacing therapy is provided based on programmed pacing parameters. The Micra TPS is self-contained and does not require a surgical incision in the chest or intravascular leads. It is inserted via a 23-French catheter placed in the femoral vein and held in place within the right ventricle of the heart via nitinol tines that attach to the myocardium.

The Aveir VR Leadless Pacemaker (Abbott) is designed to provide bradycardia pacing as a pulse generator with built-in battery and electrodes for implantation in the right ventricle. The leadless pacemaker is intended to provide sensing of intrinsic cardiac signals and delivery of cardiac pacing therapy to the target patient population. Using the power of leadless technology, the Aveir VR Leadless Pacemaker is implanted in the heart through a minimally invasive catheter procedure. The mapping capability is designed to help reduce the number of repositioning attempts (Aveir VR Brochure). A protective sleeve fully covers the Aveir VR Leadless Pacemaker's helix during catheter navigation to reduce the risk of damaging the helix or an injury to cardiovascular structures. The Aveir VR Leadless Pacemaker's active fixation helix uses a screw-in mechanism to enable both implantation and chronic retrieval of the leadless pacemaker.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health considers the Micra single-chamber transcatheter pacing system or the Aveir single-chamber VR leadless pacemaker system as medically necessary for members who meet both of the following criteria:

Leadless Pacemakers, continued

1. Has symptomatic paroxysmal or permanent high-grade arteriovenous block, or symptomatic bradycardia-tachycardia syndrome or sinus node dysfunction (sinus bradycardia or sinus pauses) requiring a single chamber ventricular pacemaker; and

2. Has a significant contraindication precluding placement of conventional single-chamber ventricular pacemaker leads, such as any of the following:

a) History of an endovascular or cardiovascular implantable electronic device (CIED) infection or who are at high risk for infection; or
b) Limited access for transvenous pacing given venous anomaly, occlusion of axillary veins, or planned use of such veins for a semi-permanent catheter; or current or planned use of an AV fistula for hemodialysis; or
c) Presence of a bioprosthetic tricuspid valve.

Select Health considers the Micra single-chamber transcatheter pacing system and the Aveir single-chamber VR leadless pacemaker system as experimental/investigational in all other situations in which the above criteria are not met.

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Billing/Coding Information
Covered when the above criteria are met

CPT CODES

33274 Transcatheter insertion or replacement of permanent leadless pacemaker, right ventricular, including imaging guidance (eg, fluoroscopy, venous ultrasound, ventriculography, femoral venography) and device evaluation (eg, interrogation or programming), when performed

Key References

1. Hayes, Inc. Evidence Analysis Research Brief. Aveir VR Leadless Pacemaker (Abbott) for Single-Chamber Pacemaker Indications. Mar 6, 2023.
2. Hayes, Inc. Health Technology Assessment. Micra Transcatheter Pacing System (Medtronic Inc.) for Single-Chamber Pacemaker Indications. Jun 3, 2022.

Revision History

Revision Date	Summary of Changes
11/20/23	For Commercial Plan Policy, revised to include coverage criteria for the Aveir leadless pacemaker system (previously considered experimental/investigational).
8/23/24	For Commercial Plan Policy, clarified in criteria #1, this requirement pertains to members

Select Health

	"requiring a single chamber ventricular pacemaker."

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

PERCUTANEOUS LEFT ATRIAL APPENDAGE CLOSURE (LAAC) DEVICES

Policy # 430

Implementation Date:12/28/09

Review Dates: 5/19/11, 6/19/14, 10/20/16, 10/19/17, 10/3/18, 10/15/19, 10/15/20, 11/18/21, 5/1/22, 6/15/23, 12/6/24

Revision Dates: 6/21/12, 4/13/13, 8/6/15, 6/3/22, 5/1/25, 6/12/25

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

Atrial fibrillation (AF) is a common dysrhythmia. It is characterized as paroxysmal if the duration is less than one week, persistent if the duration is between 7 days, and 1 year and permanent if greater than one year in duration with failure of conversion. AF can have adverse consequences related to a reduction in cardiac output and thrombus formation that can lead to systemic embolization and strokes.

Most embolized thrombi are felt to develop in the left atrial appendage (LAA). In patients with atrial fibrillation, blood tends to pool and form clots in this appendage. To prevent clot formation and subsequent embolization in patients with AF, current guidelines recommend anticoagulation with warfarin. Management of anticoagulation with warfarin is cumbersome due to the need for frequent monitoring as warfarin is subject to significant drug-drug and drug-food interactions which can result in excessive or inadequate anticoagulation. Though warfarin is the long-term oral antithrombotic of choice in patients at high risk of embolism in association with non-valvular atrial fibrillation, especially after an ischemic cerebrovascular event, many patients cannot achieve anticoagulation targets. It is estimated that therapeutic INRs are reached less than 60% of the time, and only 50% of high-risk atrial fibrillation patients are treated with warfarin. In some of these instances, alternative treatment with medications such as aspirin or clodipogrel are used, though, studies have shown these therapies to be inferior to warfarin.

To overcome many of the alternative issues surrounding anticoagulation therapy, the WATCHMAN LAA Closure Technology (Boston Scientific Corporation, Maple Grove, MN) consists of a delivery catheter and a device that is permanently implanted in the left atrial appendage (LAA) of the heart. The device, often referred to as the WATCHMAN, which received FDA approval on March 13, 2015, prevents LAA blood clots from entering the bloodstream and potentially causing a stroke. It is made of a self-expanding, nickel-titanium (Nitinol) frame with an attached woven plastic cap. The physician inserts the delivery catheter into the body through a vein in the leg. The catheter is advanced through the bloodstream until it reaches the upper right chamber of the heart (right atrium). The physician makes a small hole through the wall between the two upper chambers of the heart (atrial septum) so that the catheter reaches the LAA. The physician then pushes the WATCHMAN through the delivery catheter into the LAA where it opens like an umbrella and is permanently implanted. Once the WATCHMAN is in place, a thin layer of tissue grows over it in about 45 days. This keeps blood clots in the LAA from entering the bloodstream.

The Amplatzer Amulet Left Atrial Appendage Occluder (LAAO) is a permanent implant that is placed in the patient's left atrial appendage, which is a pouch-like part of the heart. The device is intended to prevent blood clots formed in the LAA from entering the bloodstream and potentially causing a stroke. The device is made of a Nitinol (nickel-titanium) mesh with polyester fabric cover.

Percutaneous Left Atrial Appendage Closure (LAAC) Devices, continued

Percutaneous LAA closure with Watchman or Amulet may reduce the risk of stroke in some patients with AF and high risk of stroke with contraindications to oral anticoagulation (OAC) or unwillingness to adhere to long-term OAC therapy.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers percutaneous left atrial appendage closure with an FDA approved device (e.g., Watchman or Amulet) in *limited* circumstances as outlined below. Select Health covers this procedure when either A or B are met.

A. Will be approved If recommended by Intermountain Health Cardiovascular Clinical Program,

OR

B. For all other clinicians, criteria for coverage must be met (must meet 1 and 2 **AND** either 3, 4, or 5).
   1. For patients with $CHA_2DS_2VAsc \geq 2$, who have contraindications to anticoagulation therapy; and
   2. The patient does not have rheumatic mitral stenosis;

      AND
   3. For patients who are unable to take long-term oral anticoagulation due to occupational risks, or who are intolerant to oral anticoagulation due to side effects or prior bleeding experience; or
   4. For patients in whom administering oral anticoagulation long-term is deemed clinically high risk, or contraindicated, because of fall-risk or other predisposition to bleeding complications; or
   5. For patients in need of secondary prevention who had a therapeutic INR or verified medication compliance with novel oral anticoagulation (NOAC) medications at the time of their embolic event.

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

The WATCHMAN was evaluated in four clinical studies, in which two studies compared the WATCHMAN to warfarin. Many patients with atrial fibrillation take warfarin or other FDA approved blood thinning

Percutaneous Left Atrial Appendage Closure (LAAC) Devices, continued

medicines to prevent a stroke caused by a blood clot in the brain. However, warfarin can increase the risk of bleeding anywhere in the body. If bleeding happens in the brain, this can also cause a stroke.

These two clinical studies suggested that warfarin was better than the WATCHMAN in preventing strokes caused by a blocked blood vessel in the brain. However, the number of strokes caused by bleeding in the brain was lower in the WATCHMAN patients compared to the warfarin patients.

The overall rate of serious bleeding was similar in the WATCHMAN and warfarin patients. Within several months after the device was implanted, the rate of serious bleeding was higher in the WATCHMAN patients as compared to warfarin patients. However, beginning six months after the device implant procedure, the rate of serious bleeding was lower in WATCHMAN patients.

In one of the clinical studies that evaluated the WATCHMAN, 226 out of 246 WATCHMAN patients (approximately 92%) were able to stop taking their warfarin 45 days after the device was implanted. Within a year after the implant procedure, 231 out of 234 patients remaining in the study (over 99%) were able to stop taking warfarin.

To prevent strokes, most atrial fibrillation patients can safely take blood thinning medicines (like warfarin) without serious side effects. However, in some patients, blood thinning medicines can be difficult to use due to bleeding concerns. In choosing a treatment, physicians should consider the risks and benefits of blood thinning medicines compared to the WATCHMAN or Amulet for each individual patient. This includes the risk that either kind of stroke (caused by a blocked blood vessel or by bleeding) might occur.

Billing/Coding Information

Covered: For the conditions outlined above

CPT CODES

33340	Percutaneous transcatheter closure of the left atrial appendage with endocardial implant, including fluoroscopy, transseptal puncture, catheter placement(s), left atrial angiography, left atrial appendage angiography, when performed, and radiological supervision and interpretation

HCPCS CODES

No specific codes identified

Key References

1. Arnsdorf M, Podrid. Overview of the evaluation and management of atrial fibrillation. 17.2. February 4, 2009. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/4832&selectedTitle=1~150&source=search_result. Date Accessed: November 9, 2009.
2. Arnsdorf M. Electrocardiographic and electrophysiologic features of atrial fibrillation. 17.2. June 12, 2009. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/4532&selectedTitle=1~150&source=search_result. Date Accessed: November 9, 2009.
3. Arnsdorf M. Patient information: atrial fibrillation. 17.2. August 28, 2009. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=hrt_dis/4882&selectedTitle=1~150&source=search_result. Date Accessed: November 9, 2009.
4. Atritech. The WATCHMAN System. 2008 2009. Atritech. Available: http://www.atritech.net/device.aspx?id=107. Date Accessed: November 9, 2009.
5. Bayard YL, Ostermayer SH, Sievert H. "Transcatheter occlusion of the left atrial appendage for stroke prevention." Expert Rev Cardiovasc Ther 3.6 (2005): 1003-8.
6. Block PC, Burstein S, Casale PN, et al. "Percutaneous left atrial appendage occlusion for patients in atrial fibrillation suboptimal for warfarin therapy: 5-year results of the PLAATO (Percutaneous Left Atrial Appendage Transcatheter Occlusion) Study." JACC Cardiovasc Interv 2.7 (2009): 594-600.
7. Calkins H, Brugada J, Packer DL, et al. "HRS/EHRA/ECAS expert Consensus Statement on catheter and surgical ablation of atrial fibrillation: recommendations for personnel, policy, procedures and follow-up. A report of the Heart Rhythm Society (HRS) Task Force on catheter and surgical ablation of atrial fibrillation." Heart Rhythm 4.6 (2007): 816-61.
8. Cheng J, Arnsdorf M. Surgical approaches to prevent recurrent atrial fibrillation. 17.2. July 17, 2008. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/55314&selectedTitle=1~150&source=search_result. Date Accessed: November 9, 2009.
9. Cheng J, Hijazi Z. Left atrial appendage amputation, ligation, or occulsion in patients with atrial fibrillation. 17.2. April 19, 2007. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/20022&selectedTitle=1~45&source=search_result. Date Accessed: November 9, 2009.
10. Chiam PT, Ruiz CE. "Percutaneous transcatheter left atrial appendage exclusion in atrial fibrillation." J Invasive Cardiol 20.4 (2008): E109-13.

Select Health

Percutaneous Left Atrial Appendage Closure (LAAC) Devices, continued

11. Crandall MA, Bradley DJ, Packer DL, Asirvatham SJ. "Contemporary management of atrial fibrillation: update on anticoagulation and invasive management strategies." Mayo Clin Proc 84.7 (2009): 643-62.
12. Food and Drug Administration. Summary from the Circulatory System Meeting - April 23, 2009. June 18. 2009 2009. FDA. Available: http://www.fda.gov/AdvisoryCommittees/CommitteesMeetingMaterials/MedicalDevices/MedicalDevicesAdvisoryCommittee/CirculatorySystemDevicesPanel/ucm142895.htm. Date Accessed: November 9, 2009.
13. Food and Drug Administration. Amplatzer Amulet Left Atrial Appendage Occluder – P200049. Available: https://www.fda.gov/medical-devices/recently-approved-devices/amplatzer-amulet-left-atrial-appendage-occluder-p200049#:~:text=The%20Amplatzer%20Amulet%20Left%20Atrial,and%20potentially%20causing%20a%20stroke.
14. Fountain R, Holmes DR, Jr., Hodgson PK, Chandrasekaran K, Van Tassel R, Sick P. "Potential applicability and utilization of left atrial appendage occlusion devices in patients with atrial fibrillation." Am Heart J 152.4 (2006): 720-3.
15. Friedman PA, Asirvatham SJ, Dalegrave C, et al. "Percutaneous epicardial left atrial appendage closure: preliminary results of an electrogram guided approach." J Cardiovasc Electrophysiol 20.8 (2009): 908-15.
16. Fuster V, Ryden LE, Cannom DS, et al. "ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society." Circulation 114.7 (2006): e257-354.
17. Hayes, Inc. Health Technology Assessment. Feb. 8, 2022. Percutaneous Left Appendage Closure to Reduce Closure to Reduce Stroke Risk in Patients with Atrial Fibrillation.
18. Holmes DR, Reddy VY, Turi ZG, et al. "Percutaneous closure of the left atrial appendage versus warfarin therapy for prevention of stroke in patients with atrial fibrillation: a randomised non-inferiority trial." Lancet 374.9689 (2009): 534-42.
19. Kanderian AS, Gillinov AM, Pettersson GB, Blackstone E, Klein AL. "Success of surgical left atrial appendage closure: assessment by transesophageal echocardiography." J Am Coll Cardiol 52.11 (2008): 924-9.
20. Lerakis S, Synetos A. "Left atrial appendage exclusion system for stroke prevention in atrial fibrillation: a percutaneous device delivery approach." Minerva Cardioangiol 56.6 (2008): 667-70.
21. Lerakis S, Synetos A. "WATCHMAN((R)) left atrial appendage system for stroke prevention in atrial fibrillation: a percutaneous-device delivery approach." Future Cardiol 3.5 (2007): 507-9.
22. Mannin W, Singer D, Lip G, Hart R. Antithrombotic therapy to prevent embolization in nonvalvular atrial fibrillation. 17.2. June 19, 2009. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/50724&selectedTitle=1~150&source=search_result. Date Accessed: November 9, 2009.
23. Manning W, Singer D, Hart R. Risk stratification for embolization in atrial fibrillation. 17.2. October 24, 2008. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/61906. Date Accessed: November 9, 2009.
24. Matsuo Y, Sandri M, Mangner N et al. Interventional Closure of the Left Atrial Appendage for Stroke Prevention. Circ J 2014; 78(3):619-24. https://www.jstage.jst.go.jp/article/circj/78/3/78_CJ-13-0828/_pdf
25. Mobius-Winkler S, Schuler GC, Sick PB. "Interventional treatments for stroke prevention in atrial fibrillation with emphasis upon the WATCHMAN device." Curr Opin Neurol 21.1 (2008): 64-9.
26. Onalan O, Crystal E. "Left atrial appendage exclusion for stroke prevention in patients with nonrheumatic atrial fibrillation." Stroke 38.2 Suppl (2007): 624-30.
27. Reddy VY, Homes D, Doshi HK, et al. (2011). Safety of percutaneous left atrial appendage closure: results from the Watchman Left Atrial Appendage System for Embolic Protection in Patients with AF (PROTECT AF) clinical trial and the Continued Access Registry. Circulation. Feb 1;123(4):417-24.
28. Reddy VY, Möbius-Winkler S, Miller MA et al. Left Atrial Appendage Closure With the Watchman Device in Patients With a Contraindication for Oral Anticoagulation The ASAP Study (ASA Plavix Feasibility Study With Watchman Left Atrial Appendage Closure Technology). J Am Coll Cardiol 2013; 61(25):2551-56. http://www.sciencedirect.com/science/article/pii/S0735109713014150
29. Sick PB, Schuler G, Hauptmann KE, et al. "Initial worldwide experience with the WATCHMAN left atrial appendage system for stroke prevention in atrial fibrillation." J Am Coll Cardiol 49.13 (2007): 1490-5.
30. Ussia GP, Mule M, Cammalleri V, et al. "Percutaneous closure of left atrial appendage to prevent embolic events in high-risk patients with chronic atrial fibrillation." Catheter Cardiovasc Interv 74.2 (2009): 217-22.
31. Wazni, O.M., Boersma, L., Healey, J.S., et al. Comparison of anticoagulation with left atrial appendage closure after atrial fibrillation ablation: Rationale and design of the OPTION randomized trial. Am Heart J. 2022 Sep; 251:35-42. doi: 10.1016/j.ahj.2022.05.003. Epub 2022 May 6. PMID: 35526570.
32. Weimar C, Weber R, Diener HC. "Antithrombotic medication for stroke prevention." Expert Rev Cardiovasc Ther 7.10 (2009): 1245-54.

Revision History

Revision Date	Summary of Changes
5/1/25	For Commercial Plan Policy, modified both coverage criteria and title of policy to clarify this policy applies to, Percutaneous Left Atrial Appendage Closure (LAAC) Devices.
6/12/25	For Commercial Plan Policy, clarified requirements in criterion #B-3: “For patients who are unable to take long-term oral anticoagulation ***due to occupational risks*** …”

Percutaneous Left Atrial Appendage Closure (LAAC) Devices, continued

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

PECTUS EXCAVATUM SURGERY

Policy # 160

Implementation Date: 4/22/02

Review Dates: 6/25/03, 6/24/04, 5/20/05, 5/4/06, 7/12/07, 6/19/08, 6/11/09, 5/19/11, 7/18/13, 6/19/14, 6/11/15, 6/16/16, 6/15/17, 6/21/18, 6/19/19, 6/18/20, 10/15/21, 11/10/22, 4/20/23, 4/18/24

Revision Dates: 4/22/02, 7/24/06, 1/12/10, 11/17/21, 6/30/22

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Pectus excavatum deformity is a congenitally acquired deformity of the front portion of the chest wall caused by the backward displacement of the xiphoid cartilage. In this condition, the anterior chest wall cartilages develop abnormally, leaving a "dent" in the chest wall. Though it causes a cosmetic deformity, it can also cause reduced function of an individual if the depression in the chest wall impairs the ability of the lungs or heart to work normally. Surgery can be done to correct the deformity while the child is still growing. Surgery is usually done prior to puberty with its associated bone growth and maturing of the child's skeletal structure.

The degree of anticipated functional impairment a patient with pectus excavatum deformity may expect is measured indirectly with the Haller Index. This value reflects the ratio of the transverse diameter (the horizontal distance of the inside of the ribcage) to the anterior-posterior diameter (the shortest distance between the vertebrae and sternum) of the chest and is calculated from a single CT scan of the chest performed through the deepest portion of a pectus excavatum deformity.

Haller Index

The Haller index (HI), also known as the pectus index, is a simple mathematical way to assess and describe the chest cage on CT of the thorax and is used in the detection and pre/postoperative assessment of pectus excavatum.

The Haller index is calculated by dividing the transverse diameter of the chest by the anterior-posterior distance on CT of the chest on the axial slice that demonstrates the smallest distance between the anterior surface of the vertebral body and the posterior surface of the sternum. Some authors have found that both radiographic- and CT-calculated Haller indices are strongly correlated and thus recommend the use of chest radiography instead of CT to minimize the radiation exposure.

The Haller index is affected by the vertebral level at which it is measured and is largest cranially. For consistency, therefore, it is recommended to calculate the largest Haller index in pectus excavatum patients by obtaining the AP diameter at the deepest point of the sternum.

The following values are used:

normal chest: < 2.0 cm
mild excavatum: 2.0−3.2 cm
moderate excavatum: 3.2−3.5 cm
severe excavatum: > 3.5 cm

Pectus Excavatum Surgery, continued

Corrective pectus excavatum surgery is considered with a Haller index ≥ 3.25 cm.

Correction Index

To assess a correction index (CI), a virtual correction of the pectus is performed, and a horizontal line is drawn across the posterior aspect of the corrected sternum. CIs are calculated by measuring the distance between the posterior aspect of the corrected sternum and anterior aspect of the vertebra. This number is subtracted by the distance between the posterior aspect of the sternum at the site of deepest depression and anterior vertebra. This difference is divided by the first measurement and multiplied by 100 to represent the percentage of chest depression and, therefore, potential correction.

The CI provides an accurate assessment of pectus severity, and by the nature of the measurement, reflects the potential degree of operative repair. The Haller index correlates well with the correction index in pectus patients with standard chest wall dimensions, but is quite discrepant in the nonstandard chest. We recommend operative repair for pectus excavatum with a correction index of 28% or more, because this value correlates with the long-accepted standard (Haller index ≥ 3.25 cm) and this index remains accurate even in nonstandard chest morphologies.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers repair of pectus excavatum deformities when the following criteria are met:

1. Patient is > 7 years of age; **AND** one of the following**:**
2. Haller index ≥ 3.25 cm or correction index ≥ 28% by non-contrast chest CT; **OR**
3. Serial PA and lateral chest x-rays taken at mid-respiration and at least 6 months apart revealing a persistent ratio of the external skeletal AP diameter of the chest at the angle of Lewis compared to the distance between the anterior surface of the vertebral body and the gladiolas-xiphoid junction is < 0.3 cm; **OR**
4. Serial PA and lateral chest x-rays taken at mid-respiration and at least 6 months apart revealing a persistent ratio of the external skeletal AP diameter of the chest at the angle of Lewis compared to the distance between the anterior surface of the vertebral body and the gladiolas-xiphoid junction is < 0.5 cm and > 0.3 cm, **AND either of the following:**
    a) Pulmonary function tests consistent with restrictive lung disease without other underlying lung disease which explains the abnormalities; **OR**
    b) An echocardiogram performed with in the last 3 months reveals reduced cardiovascular function attributable to the chest wall deformity.

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For this policy, specifically, there are no CMS criteria available; therefore, the Select Health Commercial policy or InterQual criteria apply. Select Health applies these requirements after careful review of the evidence that supports the clinical benefits outweigh the clinical risks. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

Pectus Excavatum Surgery, continued

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

Patients with chest wall deformities often have poor body image and self-esteem. Some individuals attempt to cover the defect with clothing and avoid activities that may require exposure of the chest. Indications for surgical correction are controversial and vary widely. Surgical repair is offered primarily as a method of improving cosmesis and psychological factors but may be necessary to improve cardiopulmonary function in some patients, as the disfigurement may be accompanied by physiologic impairment.

The scientific literature is controversial as to whether pectus excavatum is primarily cosmetic or whether it results in actual physiological impairment of function. Patients with mild pectus excavatum deformity may be treated with posture and exercise. Most surgical corrections are performed for cosmetic reasons, and in some cases to improve functional impairment. Authors agree that if patients with severe deformities do not undergo surgical repair in childhood, their symptoms worsen in adulthood.

If surgical repair is performed at an early age, it has been reported there is a high recurrence rate due to periods of rapid bone growth. While the optimal age for surgical repair is generally between the ages of 11 and 18 years, each case must be reviewed individually for the presence of impaired cardiopulmonary symptoms. In some cases, surgery may be performed in adults to correct pectus deformities. Adults who have uncorrected pectus excavatum deformity and experience symptoms of activity limitation may undergo surgical repair with low morbidity, short-term limitation of activities, and improvement of symptoms.

Surgery for pectus excavatum may be performed using any of several techniques, including a sternal osteotomy (i.e., a modified osteotomy that involves supporting, removing and repositioning the sternum) or implantation of a Silastic mold in the subcutaneous space to fill the defect without altering the thoracic cage. Surgical correction often employs a metal bar behind the sternum; the bar may be removed in 1 to 2 years, after remolding has occurred. The standard surgical procedure is the open Ravitch procedure, which involves extensive dissection, cartilage resection, and sternal osteotomy. More recently, minimally invasive techniques, including the Nuss procedure (i.e., a minimally invasive repair of pectus excavatum [MIR pectus excavatum]), have been utilized that involve the insertion of a convex steel bar beneath the sternum through small thoracic incisions. These recently developed minimally invasive methods do not require cartilage resection or osteotomy.

Billing/Coding Information

CPT CODES

21740	Reconstructive repair of pectus excavatum or carinatum; open
21742	; minimally invasive approach (Nuss procedure), without thoraoscopy
21743	; minimally invasive approach (Nuss procedure), with thoracoscopy

HCPCS CODES

No specific codes identified

Key References

1. Haller, Alex J., Journal of Pediatric Surgery, Vol 22, No 10: pp. 904-906.
2. Haller, et al., Journal of Thoracic and Cardiovascular Surgery, Vol 60, No 3 (September), 1970: pp. 375−383.
3. Haller, Alex J., Turner, Charles S. Diagnosis and Operative Management of Chest Wall Deformities in Children, Surgical Clinics of North America, Vol. 61, No. 5, (October) 1981.
4. https://radiopaedia.org/articles/haller-index?lang=us

Pectus Excavatum Surgery, continued

5. Poston, PM, Patel, SP, Rajput, M, Rossi, NO, Ghanamah, MS, Davis, JE, & Turek, JW. The Correction Index: Setting the Standard for Recommending Operative Repair of Pectus Excavatum. Ann Thorac Surg. 2014 Apr;97(4):1176-9; discussion 1179-80. doi: 10.1016/j.athoracsur.2013.12.050. Epub 2014 Feb 26.
6. Stedman's Medical Dictionary, 26th Edition.
7. Welch, Kenneth J., Satisfactory Surgical Correction of Pectus Excavation Deformity in Childhood, A Limited Opportunity. J. Thoracic Surgery, Vol. 36, No 5 (November), 1958: pp. 697−713.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

Select Health

MEDICAL POLICY

TRANSCATHETER EDGE-TO-EDGE REPAIR (TEER)

Policy # 464
Implementation Date: 10/19/10
Review Dates: 12/15/11, 10/20/16, 10/19/17, 10/3/18, 10/15/19, 10/15/20, 11/25/21, 9/14/22, 10/19/23, 10/1/24
Revision Dates: 5/30/13, 1/10/14, 1/23/14, 12/13/21, 1/10/22, 10/1/22, 7/3/23, 10/17/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

The mitral valve insufficiency occurs when the mitral valve fails to close properly causing blood to flow backward into the atrium (regurgitation), leaving the left ventricle, the heart's primary pumping chamber, with too little blood. To compensate, the ventricle stretches and overworks. Over time, it becomes enlarged, distorted, and weak.

Historically, 3 options have been available to treat symptomatic mitral valve regurgitation: medications (such as diuretics to alleviate fluid retention), valve repair, and valve replacement. Valve surgery is a highly invasive procedure which many fragile patients may not be able to undertake due to their associated comorbidities. Consequently, new approaches to mitral valve repair/replacement are under development.

One such development is the MitraClip (Abbott Laboratories, Chicago, IL). The MitraClip procedure is performed in a cardiac catheterization laboratory with the patient under general anesthesia. A thin catheter is inserted through a small incision in the groin and guided through the femoral vein to the affected area of the heart. A smaller catheter holding the clip is slipped through the first catheter. After the clip is attached to the valve leaflets, the catheters are removed. The patient is released from the hospital within a day or two. If the clip is not placed ideally on the first attempt, it can be reset. If it does not sufficiently correct the regurgitation, surgical repair or replacement of the valve remains an option.

The MitraClip system received FDA approval on October 24, 2013, for use in the United States. The FDA approved indication is for the percutaneous reduction of significant symptomatic mitral regurgitation (MR is for the percutaneous reduction of significant symptomatic mitral regurgitation (> 3+) due to primary abnormality of the mitral apparatus [degenerative MR] in patients who have been determined to be at a prohibitive risk for mitral valve surgery by a heart team. This team includes a cardiac surgeon experienced in mitral valve surgery and a cardiologist experienced in mitral valve disease, and this procedure is intended for patients in whom existing comorbidities would not preclude the expected benefit from the reduction of the mitral regurgitation.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers transcatheter edge-to-edge repair (TEER) for patients who meet *specific* coverage criteria.

Transcatheter Edge-to-Edge Repair, continued

Coverage criteria:

A. Mitral Valve

Must meet one of the following:

1. Primary degenerative mitral valve disease*, moderate-to-severe, or severe mitral regurgitation with high operative risk; or
2. Secondary moderate-to-severe or severe mitral regurgitation**, regardless of operative risk.

B. Tricuspid Valve

1. Select Health will cover tricuspid TEER with primary or secondary tricuspid regurgitation that is severe or greater#.

*Degenerative mitral valve disease is classified as degeneration of the mitral valve due to changes in the connective tissue of the valve or mitral chordae causing weakness and redundancy of the leaflets and their supporting structures. Degenerative mitral valve disease does not include infective causes, or mitral regurgitation due to ischemia or problems intrinsic with the left ventricle causing the mitral annulus to dilate and creating the mitral insufficiency. The most common finding in patients with degenerative valve disease is leaflet prolapse due to elongation or rupture of the chordal apparatus.

**Secondary means patients with normal mitral valves, who develop heart failure symptoms with moderate-to-severe or severe functional mitral regurgitation (MR), when the patient remains symptomatic despite stable doses of maximally tolerated guideline-directed medical therapy (GDMT).

#**Echocardiographic Parameters and Relative Cutoffs for TR 5-Tier Grading (Hahn, R. T., et al.)**

	Mild (1+)	**Moderate (2+)**	**Severe (3+)**	**Massive (4+)**	**Torrential (5+)**
Qualitative					
Tricuspid morphology	Normal or mildly abnormal	Moderately abnormal	Severely abnormal (flat, leaflet, large coaptation gap, marked tethering)	Severely abnormal (flat, leaflet, large coaptation gap, marked tethering)	Severely abnormal (flat, leaflet, large coaptation gap, marked tethering)
Color-flow jet area	Small, narrow; central	Moderate central	Large central; or eccentric, wall impinging	Large central; or eccentric, wall impinging	Large central; or eccentric, wall impinging
Flow convergence zone	Not visible, transient, or small	Intermediate in size and duration	Large throughout systole	Large throughout systole	Large throughout systole
CWD contour	Faint, partial, parabolic	Dense, parabolic	Dense, parabolic or triangular	Dense, often triangular, may have low peak velocity	Dense, usually triangular, often low peak velocity

Transcatheter Edge-to-Edge Repair, continued

Right heart dimensions	Usually normal	Normal or mild dilation	Usually dilated	Dilated	Dilated
Semiquantitative					
VCW (biplane), mm[a]	<3	3-6.9	7-13.9	14-20.9	≥21
PISA, radius, mm[b]	≤5.4	5.5-8.9	≥9	≥9	≥9
Hepatic vein flow[c]	Systolic dominant	Systolic blunting	Systolic flow reversal	Systolic flow reversal	Systolic flow reversal
Tricuspid inflow (PWD)	A-wave dominant	Variable	E-wave dominant (≥1 m/s)	E-wave dominant (≥1 m/s)	E-wave dominant (≥1 m/s)
Quantitative					
PISA EROA, mm^2	<20	20-39	40-59	60-79	≥80
Regurgitant volume (2D PISA), mL	<30	30-44	45-59	60-74	≥75
New quantitative methods					
Regurgitant fraction, %	≤15	16-49	≥50	≥50	≥50
3D VCA, mm^2	—	—	75-94.9	95-114.9	≥115
2D Doppler EROA, mm^2	—	—	75-94.9	95-114.9	≥115

[a]At a color Doppler scale between 40 and 60 cm/s. Note that some studies suggest an average VCW of >9 mm should define severe TR. [b]Color Doppler Nyquist shift down toward 20 cm/s, until the hemispherical flow convergence zone is clearly visualized. [c]Unless other reason for flow reversal (ie, atrial fibrillation, right atrial elevated pressures/noncompliance). Adapted from: 1) Zoghbi et al; 2) Lancellotti et al; and 3) Hahn et al.
CWD = continuous-wave Doppler; EROA = effective regurgitant orifice area; PISA = proximal isovelocity surface area; PWD = pulsed-wave Doppler; VCA = vena contracta area; VCW = vena contracta width.

Transcatheter Edge-to-Edge Repair, continued

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

In December 2013, an updated review of this technology was undertaken. This review identified one systematic review and 41 primary literature articles which met the inclusion criteria for review. Most of the authors conducted and published their results after the EVEREST I Trial, EVEREST II RCT, EVEREST II HRR, and REALISM HR trials were completed. The only systematic review included only 12 studies and stated that no RCTs comparing MitraClip to non-surgical therapies were identified. This is the same finding illustrated in this report. They also noted that only one paper published between 2000 and 2013 reported outcomes beyond 12 months; however, 11 papers identified for this review followed-up with patients after 12 months between only 2010 and 2013; with regards to the primary literature articles, 5,575 patients have been enrolled in published studies in the last three years. Outcomes from the studies showed successful implantation ranging from 85% to 100% with reduction in mitral regurgitation ranging from a low 41.4% to 65%. These studies also showed improvement in NYHA categorization of at least 1 grade, ranging from 34.4% to 66.7%. In the 9 studies which assessed patients out to at least 12 months post-procedure, survival ranged from 71.1% to 87.5%. Like the findings reported in the systematic review, the average follow-up time was just over a year in the primary literature. Dissimilar to that review, however, 5 papers followed patients for 2 years and one paper followed patients out to 4 years.

An important observation from the studies relates to the number of MitraClips used. 11 of the 41 studies used either more than 2 clips or no clip at all in each procedure. Paranskaya et al., for example, reported that 19 patients (22.3%) received 3 clips, 4 patients (4.7%) received 4 clips, and 1 patient (1.2%) received 5 clips. For studies where clips had to be removed because of perioperative complications, the authors reported that zero clips were used. Also, some of the 11 studies did not report what number of clips over 2 were used during the procedure so it is difficult to estimate what number of procedures used 3, 4, or 5 clips.

Feldman et al. and Mauri et al. were the only two to have randomized their surgical repair patients from their MitraClip patients. This is important to note as the remaining three studies explicitly note that MitraClip patients were significantly older, had lower LVEF, had a higher EuroSCORE I, had higher LV diameter, and had more comorbidities in general.

Five studies compared MitraClip surgery to standard valvular surgery. Feldman et al. and Mauri et al. were the only two to have randomized their surgical repair patients from their MitraClip patients. This is important to note, as the remaining three studies explicitly note that MitraClip patients were significantly older, had lower LVEF, had a higher EuroSCORE I, had higher LV diameter, and had more comorbidities in general. These studies did not demonstrate a statistically significant difference between percutaneous MitraClip procedures to valvular surgery for endpoints which included 30-day survival, longer term mortality, and improvement in cardiovascular function.

In conclusion, the current body of evidence regarding MitraClip demonstrates that MitraClip may reasonably improve patient outcomes, especially for patients who otherwise could not undergo surgery. Though only a few papers thoroughly examine patient outcomes in the long-term (> 2 years), what

Transcatheter Edge-to-Edge Repair, continued

evidence does exist, shows similar outcomes as compared to standard surgical methods in appropriately selected patients. (GRADE 1B).

Billing/Coding Information
Covered: for the conditions outlined above

CPT CODES

0345T	Transcatheter mitral valve repair percutaneous approach via the coronary sinus [MitraClip]
0545T	Transcatheter tricuspid valve annulus reconstruction with implantation of adjustable annulus reconstruction device, percutaneous approach
0569T	Transcatheter tricuspid valve repair, percutaneous approach; initial prosthesis
0570T	Transcatheter tricuspid valve repair, percutaneous approach; each additional prosthesis during same session (List separately in addition to code for primary procedure)
0646T	Transcatheter tricuspid valve implantation/replacement (TTVI) with prosthetic valve, percutaneous approach, including right heart catheterization, temporary pacemaker insertion, and selective right ventricular or right atrial angiography, when performed
33418	Transcatheter mitral valve repair, percutaneous approach, including transseptal puncture when performed; initial prosthesis
33419	; additional prosthesis (es) during the same session (List separately in addition to code for primary procedure)
93590	Percutaneous transcatheter closure of paravalvular leak; initial occlusion device, mitral valve
93592	Percutaneous transcatheter closure of paravalvular leak; each additional occlusion device (List separately in addition to code for primary procedure)

HCPCS CODES

No specific codes identified

Key References

1. Alegria-Barrero, E, Chan, PH, Foin, N, et al. (2013). Concept of the central clip: when to use one or two MitraClips(R). EuroIntervention.
2. Armoiry, X, Brochet, E, Lefevre, T, et al. (2013). Initial French experience of percutaneous mitral valve repair with the MitraClip: a multicentre national registry. Arch Cardiovasc Dis 106.5: 287-94.
3. Armstrong, EJ, Rogers, JH, Swan, CH, et al. (2013). Echocardiographic predictors of single versus dual MitraClip device implantation and long-term reduction of mitral regurgitation after percutaneous repair. Catheter Cardiovasc Interv 82.4: 673-9.
4. Auricchio, A, Schillinger, W, Meyer, S, et al. (2011). Correction of mitral regurgitation in nonresponders to cardiac resynchronization therapy by MitraClip improves symptoms and promotes reverse remodeling. J Am Coll Cardiol 58.21: 2183-9.
5. Capomolla, S, Febo, O, Gnemmi, M, et al. (2000). Beta-blockade therapy in chronic heart failure: diastolic function and mitral regurgitation improvement by carvedilol. Am Heart J 139.4: 596-608.
6. Cedars-Sinai. (2010). Cedars-Sinai Heart Institute And The Evalve Mitraclip: Clinical Trial Of Non-Surgical Repair For Severe Mitral Valve Regurgitation. Cedars-Sinai. Available: http://www.cedars-sinai.edu/About-Us/News/News-Releases-2008/Cedars-Sinai-Heart-Institute-And-The-Evalve-Mitraclip-Clinical-Trial-Of-Non-Surgical-Repair-For-Severe-Mitral-Valve-Regurgitation-.aspx. Date Accessed: August11, 2010.
7. Chan, PH, She, HL, Alegria-Barrero, E, et al. (2012). Real-world experience of MitraClip for treatment of severe mitral regurgitation. Circ J 76.10: 2488-93.
8. Colucci, WS. (2013) Predictors of survival in heart failure due to systolic dysfunction. UptoDate. Available: http://www.uptodate.com/contents/predictors-of-survival-in-heart-failure-due-to-systolic-dysfunction?source=search_result&search=nyha&selectedTitle=1~150#H3. Date Accessed: December 5, 2013.
9. Conradi, L, Treede, H, Rudolph, V, et al. (2013). Surgical or percutaneous mitral valve repair for secondary mitral regurgitation: comparison of patient characteristics and clinical outcomes. Eur J Cardiothorac Surg 44.3: 490-6; discussion 496.
10. Conradi, L, Treede, H, Franzen, O, et al. (2011). Impact of MitraClip therapy on secondary mitral valve surgery in patients at high surgical risk. Eur J Cardiothorac Surg 40.6: 1521-6.

Select Health

Transcatheter Edge-to-Edge Repair, continued

11. Divchev, D, Kische, S, Paranskaya, L, et al. (2012). In-hospital outcome of patients with severe mitral valve regurgitation classified as inoperable and treated with the MitraClip(R) device. J Interv Cardiol 25.2: 180-9.
12. Estevez-Loureiro, R, Franzen, O, Winter, R, et al. (2013). Echocardiographic and clinical outcomes of central versus non-central percutaneous edge-to-edge repair of degenerative mitral regurgitation. J Am Coll Cardiol.
13. Feldman, T, Foster, E, Glower, DD, et al. (2011). Percutaneous repair or surgery for mitral regurgitation. N Engl J Med 364.15: 1395-406.
14. Foster, E. (2010). Clinical practice. Mitral regurgitation due to degenerative mitral-valve disease. N Engl J Med 363.2: 156-65.
15. Foster, E, Kwan, D, Feldman, T, et al. (2013). Percutaneous mitral valve repair in the initial EVEREST cohort: evidence of reverse left ventricular remodeling. Circ Cardiovasc Imaging 6.4: 522-30.
16. Franzen, O, van der Heyden, J, Baldus, S, et al. (2011). MitraClip(R) therapy in patients with end-stage systolic heart failure. Eur J Heart Fail 13.5: 569-76.
17. Gaemperli, O, Moccetti, M, Surder, D, et al. (2012). Acute haemodynamic changes after percutaneous mitral valve repair: relation to mid-term outcomes. Heart 98.2: 126-32.
18. Gaasch, WH. (2013) Functinoal mitral regurgitation. September 17, 2012. Up to Date. Available: http://www.uptodate.com/contents/functional-mitral-regurgitation. Date Accessed: November 29, 2013.
19. Gaasch, WH. (2013) Overview of the management of chronic mitral regurgitation. August 12, 2013. Up to Date. Available: http://www.uptodate.com/contents/overview-of-the-management-of-chronic-mitral-regurgitation?source=search_result&search=mitraclip&selectedTitle=1~2. Date Accessed: November 28, 2013.
20. Gaemperli, O, Biaggi, P, Gugelmann, R, et al. (2013). Real-time left ventricular pressure-volume loops during percutaneous mitral valve repair with the MitraClip system. Circulation 127.9: 1018-27.
21. Glower, D, Ailawadi, G, Argenziano, M, et al. (2012). EVEREST II randomized clinical trial: predictors of mitral valve replacement in de novo surgery or after the MitraClip procedure. J Thorac Cardiovasc Surg 143.4 Suppl: S60-3.
22. Grigioni, F, Avierinos, JF, Ling, LH, et al. (2002). Atrial fibrillation complicating the course of degenerative mitral regurgitation: determinants and long-term outcome. J Am Coll Cardiol 40.1: 84-92.
23. Guarracino, F, Ferro, B, Baldassarri, R, et al. (2013). Non invasive evaluation of cardiomechanics in patients undergoing MitrClip procedure. Cardiovasc Ultrasound 11: 13.
24. Hahn, R. T., et al. Tricuspid Valve Academic Research Consortium Definitions for Tricuspid Regurgitation and Trial Endpoints. *Eur Heart J*. 2023 Nov 14;44(43):4508-4532. doi: 10.1093/eurheartj/ehad653. PMID: 37793121; PMCID: PMC10645050.
25. Hanson, I. (2013). Mitral Regurgiatation Medication,Medscape. Available: http://emedicine.medscape.com/article/155618-medication#showall. Date Accessed: December 5, 2013.
26. Herrmann, HC, Gertz, ZM, Silvestry, FE, et al. (2012). Effects of atrial fibrillation on treatment of mitral regurgitation in the EVEREST II (Endovascular Valve Edge-to-Edge Repair Study) randomized trial. J Am Coll Cardiol 59.14: 1312-9.
27. Hsieh, LHT. (2013). The MitraClip Miricle. The Globe and Mail. Available: http://www.theglobeandmail.com/life/health-and-fitness/advsunnybrook/sunnybrookfeatures/the-mitraclip-miracle/article12038536/. Date Accessed: December 6, 2013.
28. Lim, DS, Reynolds, MR, Feldman, T, et al. (2013). Improved Functional Status and Quality of Life in Prohibitive Surgical Risk Patients With Degenerative Mitral Regurgitation Following Transcatheter Mitral Valve Repair with the MitraClip(R) System. J Am Coll Cardiol.
29. Maisano, F, Franzen, O, Baldus, S, et al. (2013). Percutaneous mitral valve interventions in the real world: early and 1-year results from the ACCESS-EU, a prospective, multicenter, nonrandomized post-approval study of the MitraClip therapy in Europe. J Am Coll Cardiol 62.12: 1052-61.
30. Mauri, L, Foster, E, Glower, DD, et al. (2013). 4-year results of a randomized controlled trial of percutaneous repair versus surgery for mitral regurgitation. J Am Coll Cardiol 62.4: 317-28.
31. Mayo Clinic. (2010). Mitral Valve Regurgitation. Mayo Clinic. Available: http://www.mayoclinic.com/health/mitral-valve-regurgitation/DS00421. Date Accessed: August 11, 2010.
32. Mealing, S, Feldman, T, Eaton, J, et al. (2013). EVEREST II high risk study based UK cost-effectiveness analysis of MitraClip(R) in patients with severe mitral regurgitation ineligible for conventional repair/replacement surgery. J Med Econ 16.11: 1317-26.
33. Munkholm-Larsen, S, Wan, B, Tian, DH, et al. (2013). A systematic review on the safety and efficacy of percutaneous edge-to-edge mitral valve repair with the MitraClip system for high surgical risk candidates. Heart.
34. Orban, M, Braun, D, Sonne, C, et al. (2013). Dangerous liaison: successful percutaneous edge-to-edge mitral valve repair in patients with end-stage systolic heart failure can cause left ventricular thrombus formation. EuroIntervention.
35. Otto, CM, Salerno, CT. (2005). Timing of surgery in asymptomatic mitral regurgitation. N Engl J Med 352.9: 928-9.
36. Paranskaya, L, D'Ancona, G, Bozdag-Turan, I, et al. (2013). Percutaneous mitral valve repair with the MitraClip system: perioperative and 1-year follow-up results using standard or multiple clipping strategy. Catheter Cardiovasc Interv 81.7: 1224-31.
37. Paranskaya, L, D'Ancona, G, Bozdag-Turan, I, et al. (2013). Residual mitral valve regurgitation after percutaneous mitral valve repair with the MitraClip(R) system is a risk factor for adverse one-year outcome. Catheter Cardiovasc Interv 81.4: 609-17.
38. Paranskaya, L, D'Ancona, G, Bozdag-Turan, I, et al. (2013). Percutaneous vs surgical repair of mitral valve regurgitation: single institution early and midterm outcomes. Can J Cardiol 29.4: 452-9.
39. Perl, L, Vaturi, M, Assali, A, et al. (2013). Preliminary experience using the transcatheter mitral valve leaflet repair procedure. Isr Med Assoc J 15.10: 608-12.
40. Pleger, ST, Mereles, D, Schulz-Schonhagen, M, et al. (2011). Acute safety and 30-day outcome after percutaneous edge-to-edge repair of mitral regurgitation in very high-risk patients. Am J Cardiol 108.10: 1478-82.
41. Pleger, ST, Schulz-Schonhagen, M, Geis, N, et al. (2013). One year clinical efficacy and reverse cardiac remodelling in patients with severe mitral regurgitation and reduced ejection fraction after MitraClip implantation. Eur J Heart Fail 15.8: 919-27.
42. Puls, M, Tichelbacker, T, Bleckmann, A, et al. (2013). Failure of acute procedural success predicts adverse outcome after percutaneous edge-to-edge mitral valve repair with MitraClip. EuroIntervention.
43. Reichenspurner, H, Schillinger, W, Baldus, S, et al. (2013). Clinical outcomes through 12 months in patients with degenerative mitral regurgitation treated with the MitraClip(R) device in the ACCESS-EUrope Phase I trial. Eur J Cardiothorac Surg 44.4: e280-8.

Transcatheter Edge-to-Edge Repair, continued

44. Reynolds, M, Galper, B, Apruzzese, P, et al. (2012). TCT-786 Cost Effectiveness of the MitraClip® Compared with Mitral Valve Surgery: 12-month Results from the EVEREST II Randomized Controlled Trial. J Am Coll Cardiol 60.17_S.
45. Rosenthal, L. (2013) Atrial Fibrillation Medication. December 3, 2013. Medscape. Available: http://emedicine.medscape.com/article/151066-medication. Date Accessed: December 5, 2013.
46. Rudolph, V, Knap, M, Franzen, O, et al. (2011). Echocardiographic and clinical outcomes of MitraClip therapy in patients not amenable to surgery. J Am Coll Cardiol 58.21: 2190-5.
47. Rudolph, V, Lubos, E, Schluter, M, et al. (2013). Aetiology of mitral regurgitation differentially affects 2-year adverse outcomes after MitraClip therapy in high-risk patients. Eur J Heart Fail 15.7: 796-807.
48. Scandura, S, Ussia, GP, Capranzano, P, et al. (2012). Left cardiac chambers reverse remodeling after percutaneous mitral valve repair with the MitraClip system. J Am Soc Echocardiogr 25.10: 1099-105.
49. Schillinger, W, Hunlich, M, Baldus, S, et al. (2013). Acute outcomes after MitraClip therapy in highly aged patients: results from the German TRAnscatheter Mitral valve Interventions (TRAMI) Registry. EuroIntervention 9.1: 84-90.
50. Seneviratne, B, Moore, GA, West, PD. (1994). Effect of captopril on functional mitral regurgitation in dilated heart failure: a randomised double blind placebo controlled trial. Br Heart J 72.1: 63-8.
51. Siegel, RJ, Biner, S, Rafique, AM, et al. (2011). The acute hemodynamic effects of MitraClip therapy. J Am Coll Cardiol 57.16: 1658-65.
52. Smith, T, McGinty, P, Bommer, W, et al. (2012). Prevalence and echocardiographic features of iatrogenic atrial septal defect after catheter-based mitral valve repair with the MitraClip system. Catheter Cardiovasc Interv 80.4: 678-85.
53. Surder, D, Pedrazzini, G, Gaemperli, O, et al. (2013). Predictors for efficacy of percutaneous mitral valve repair using the MitraClip system: the results of the MitraSwiss registry. Heart 99.14: 1034-40.
54. Taggart NW, Cabalka AK, Eicken A, Aboulhosn JA, Thomson JDR, Whisenant B, et al. Outcomes of Transcatheter Tricuspid Valve-in-Valve Implantation in Patients With Ebstein Anomaly. VIVID Registry. *Am J Cardiol*. 2018 Jan 15;121(2):262-268.
55. Taramasso, M, Denti, P, Buzzatti, N, et al. (2012). Mitraclip therapy and surgical mitral repair in patients with moderate to severe left ventricular failure causing functional mitral regurgitation: a single-centre experience. Eur J Cardiothorac Surg 42.6: 920-6.
56. TCT Daily Staff. (2012). Extended EVEREST II Data Examine Safety, Efficacy of MitraClip as Well as Cost Effectiveness. October 25, 2012. TCT MD. Available: http://www.tctmd.com/show.aspx?id=115915. Date Accessed: December 6, 2013.
57. Teufel, T, Steinberg, DH, Wunderlich, N, et al. (2012). Percutaneous mitral valve repair with the MitraClip(R) system under deep sedation and local anaesthesia. EuroIntervention 8.5: 587-90.
58. U.S. Food and Drug Administration. (2013). MitraClip Clip Delivery System Labelling. November 15, 2013. U.S. Department of Health & Human Services. Available: http://www.accessdata.fda.gov/cdrh_docs/pdf10/P100009c.pdf. Date Accessed: November 28, 2013.
59. U.S. Food and Drug Administration (2019). FDA News Release. FDA approves new indication for valve repair device to treat certain heart failure patients with mitral regurgitation. Available: https://www.fda.gov/news-events/press-announcements/fda-approves-new-indication-valve-repair-device-treat-certain-heart-failure-patients-mitral. Date Accessed: December 2, 2021.
60. Ussia, GP, Cammalleri, V, Sarkar, K, et al. (2012). Quality of life following percutaneous mitral valve repair with the MitraClip System. Int J Cardiol 155.2: 194-200
61. Vascular, A. (2013). MitraClip Percutaneous Mitral Valve Repair System. Abbott Vascular. Available: http://www.abbottvascular.com/int/mitraclip.html#about-mitral-regurgitation. Date Accessed: November 29, 2013.
62. Whisenant B, Kapadia SR, Eleid MF, Kodali SK, McCabe JM, Krishnaswamy A, Morse M, Smalling RW, Reisman M, Mack M, O'Neill WW, Bapat VN, Leon MB, Rihal CS, Makkar RR, Guerrero M. One-Year Outcomes of Mitral Valve-in-Valve Using the SAPIEN 3 Transcatheter Heart Valve. *JAMA Cardiol*. 2020 Jul 29: e202974. doi: 10.1001/jamacardio.2020.2974
63. Whitlow, PL, Feldman, T, Pedersen, WR, et al. (2012). Acute and 12-month results with catheter-based mitral valve leaflet repair: the EVEREST II (Endovascular Valve Edge-to-Edge Repair) High Risk Study. J Am Coll Cardiol 59.2: 130-9.
64. Zoghbi, WA, Enriquez-Sarano, M, Foster, E, et al. (2003). Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. J Am Soc Echocardiogr 16.7: 777-802.

Select Health

Transcatheter Edge-to-Edge Repair, continued

Select Health

MEDICAL POLICY

PERCUTANEOUS TRANSCATHETER CLOSURE FOR THE TREATMENT OF ATRIAL SEPTAL DEFECTS (ASD) AND PATENT FORAMEN OVALE (PFO)

Policy # 174
Implementation Date: 4/21/02
Review Dates: 10/23/03, 11/18/04, 3/25/05, 6/19/08, 6/11/09, 6/17/10, 5/3/11, 7/18/13, 6/19/14, 6/11/15, 6/16/16, 6/15/17, 6/28/18, 6/13/19, 6/18/20, 6/17/21, 5/6/22, 6/15/23, 6/20/24
Revision Dates: 11/18/04, 8/4/06, 10/31/06, 2/5/07, 6/30/07, 12/5/11, 8/14/18, 5/13/22, 5/2/24, 5/17/24, 11/6/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Atrial septal defect (ASD) is the most common congenital lesion in adults after bicuspid aortic valve. Although the defect is often asymptomatic until adulthood, potential complications of an undetected ASD include right ventricular failure, atrial arrhythmias, paradoxical embolization, cerebral abscess, and pulmonary hypertension that can become irreversible and lead to right-to-left shunting (Eisenmenger syndrome).

In approximately 70% of individuals, the primum and secundum septa fuse after birth, creating an intact interatrial septum. However, in a significant proportion of the population, the septae do not fuse. If the foramen ovale is completely covered but not sealed, it is called a "probe patent" or simply "patent" foramen ovale (PFO), indicating that the foramen can be opened by a reversal of the interatrial pressure gradient or by an intracardiac catheter. Less commonly, an open communication persists between the atria after septation. Such a communication is called an atrial septal defect (ASD).

The various types of ASDs are classified according to their location and the nature of the embryologic defect. Isolated ASDs include: PFO, ASD at the fossa ovalis (secundum ASD), a defect superior to the fossa ovalis (superior sinus venosus type ASD, superior vena caval defect), a defect inferior to the fossa ovalis (inferior sinus venosus type ASD, inferior vena caval defect), and coronary sinus defects.

The standard of care for the treatment of significant or symptomatic ASDs is percutaneous closure using several FDA-approved devices. Recently, this same therapy has been investigated for closure of physiologically significant PFOs. No devices are currently FDA approved that are specific to PFO-closure, however, this therapy is being performed using devices that are FDA approved for ASD-closure in patients with cryptogenic stroke. PFO-closure is also being investigated as a treatment of migraine headaches.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers percutaneous transcatheter closure of symptomatic atrial septal defect (ASD) in secundum position or for the closure of the fenestration in individuals who have undergone a fenestrated Fontan procedure. These procedures are considered medically necessary

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

when using a device that has been FDA approved for that purpose and used according to the labeled indications.

Select Health covers percutaneous transcatheter closure of patent foramen ovale (PFO) using FDA approved closure devices *in limited circumstances*.

Criteria for coverage (must meet both 1 and 2):

1. Patient has a documented history of cryptogenic, clinically-evident transient ischemic episode or cryptogenic stroke, which has been verified by an independent, qualified neurologic specialist documenting the PASCAL classification system* and RoPE scores**; and
2. Patient has demonstrated the PFO to be hemodynamically significant as defined by either one of the following:
   a. Right-sided pressure or volume overload changes on imaging studies along with evidence of a large shunt (typically permanent right-to-left shunt); or
   b. Documented orthodeoxia-platypnea (resting or exercise induced)

Select Health does NOT cover percutaneous transcatheter closure of PFO for migraine prophylaxis or for any other indications because its effectiveness for these indications has not been established. It is considered experimental/investigational in these circumstances.

* PFO-Associated Stroke Causal Likelihood (PASCAL) Classification System (UpToDate)

(Proposed flexible clinical practice approach to classifying patent foramen ovale causal association in patients with embolic infarct topography and without other major stroke sources†)

Risk source	Features	RoPE score	
		Low††	High††
Very High	A PFO and a straddling thrombus	Definite	Definite
High	(1) Concomitant pulmonary embolism or deep venous thrombosis preceding an index infarct combined with either (2a) a PFO and an atrial septal aneurysm or (2b) a large-shunt PFO	Probable	Highly probably
Medium	Either (1) a PFO and an atrial septal aneurysm or (2) a large-shunt PFO	Possible	Probable
Low	A small-shunt PFO without an atrial septal aneurysm	Unlikely	Possible

†The algorithm in this table is proposed for use in flexible clinical practice when application of an entire formal classification system is not being conducted.

††The RoPE score includes points for 5 age categories, cortical infarct, absence of hypertension, diabetes, prior stroke or transient ischemic attack, and smoking. A higher RoPE score (≥ 7 points) increases probability of causal association.

** Risk of Paradoxical Embolism (RoPE) score (UpToDate)

Characteristic	Points	RoPE score
No history of hypertension	1	
No history of diabetes	1	
No history of stroke or transient ischemic attack	1	
Nonsmoker	1	

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

Cortical Infarct on imaging	1	
Age, years		
18 to 29	5	
30 to 39	4	
40 to 49	3	
50 to 59	2	
60 to 69	1	
≥ 70	0	
Total score (sum of individual points)		
Maximum score (a patient < 30 years with no hypertension, no diabetes, no history of stroke or TIA, nonsmoker, and cortical infarct)		10
Minimum score (a patient ≥ 70 years with hypertension, diabetes, prior stroke, current smoker, and no cortical infarct)		0

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

There are several devices approved by the FDA for closure of ASDs. These include the AMPLATZER Septal Occluder, HELEX Septal Occluder, and the NMT Medical CardioSEAL STARFlex Septal Occlusion System. All these devices are inserted via a catheter in collapsed form, then once at the defect, the device opens, and occludes the defect.

Prior to October 31, 2006, the FDA approved 2 catheter systems under the Human Device Exemption (HDE) for patients with patent foramen ovale (PFO) and cryptogenic stroke due to presumed paradoxical embolism through a PFO and who have failed conventional drug therapy. The previously approved devices were the NMT Medical CardioSEAL STARFlex Septal Occlusion System and the AGA Medical Amplatzer PFO Occluder. As it was felt that these devices no longer met the criteria for the HDE exemption and needed to prove their efficacy through the PMA approval process, both manufacturers agreed to cease marketing their devices effective October 31, 2006.

Though no randomized, controlled trials have proven the PFO specific percutaneous devices to be safe and effective, the literature suggests that currently approved FDA devices are effective in closing both ASD and PFO. Anzola et al., for example, studied 140 patients who underwent percutaneous closure for PFO. At the 12-month follow-up, 91% had not detectable right-to-left shunt and only one CVA-like event had occurred. Bruch et al. reported on 66 patients with ASD, none of whom had residual shunting after 12 months. No evidence of recurrent thromboembolic events was observed in this population.

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

A 2004 study of 28 patients with ASD by Khositseth et al. found a 7% residual shunt rate at 12 months. A 3.6% recurrence rate for recurrent thromboembolic events was observed at 23 months. Yew et al. reported 5-year data on patients who underwent percutaneous ASD-closure. All patients experienced complete closure.

Subsequently, as the questions around the similarity between PFO and ASD physiology have been addressed in the above literature, one may be able to extrapolate that use of ASD-closure approved devices will be effective in PFOs. Supporting this supposition, several conference abstracts provide additional support for ASD-closure devices for treating PFO. In 58 patients, closed with either the Amplatzer PFO device or the Amplatzer Septal Occluder, residual shunt at 3 months was lower in the latter group. In 1000 patients treated with either device, serious adverse events were uncommon and the relative risk of CVA was 0.15 compared with historical controls.

It was noted that a Hayes Directory published in 2002 gave percutaneous closure of PFO and ASD a 'B' rating indicating a device with some proven benefit (i.e., use of the technology is supported by a moderate level of published evidence, but further research is required to fully clarify clinical indications, contraindications, treatment parameters, comparison with other technologies, and/or impact on health outcomes). The report cited a lack of long-term follow-up data as a weakness in the literature.

These data suggest that ASD devices are effective at closing PFO and reducing risk for recurrent stroke, at least in the short-term. Long-term data are still lacking as well as comparative trials to examine the relative efficacy between percutaneous devices and medical therapy on stroke outcomes. Ongoing clinical trials may provide additional insight pertinent to this question.

In 2011, a literature review was performed, and guidelines provided by the Intermountain Medical Center Heart Institute identified a science advisory from the American College of Cardiology Foundation (ACCF), American Heart Association (AHA), American Stroke Association (ASA), and the American Academy of Neurology published in 2010. The advisory concluded the optimal therapy for prevention of recurrent stroke or transient ischemic attack in patients with cryptogenic stroke and PFO has not been defined. Although numerous observational studies have suggested a strong association between PFO and cryptogenic stroke, a causal relationship has not been convincingly established for the majority of affected patients. Treatment choices include medical therapy with antiplatelet agents or vitamin K antagonists, percutaneous device closure, or open surgical repair. Whereas suture closure of an incidental PFO is performed routinely during an operation undertaken for another indication, primary surgical repair is rarely advocated in the current era. The choice between medical therapy and percutaneous device closure has been the subject of intense debate over the past several years, albeit one that has not been adequately informed by randomized, prospective clinical trial data to permit an objective comparison of the relative safety and efficacy of these respective approaches. Enrollment in clinical trials has lagged considerably despite frequent calls for participation from the US Food and Drug Administration and major professional societies. Completion and peer review of ongoing trials are critical steps to establish an evidence base from which clinicians can make informed decisions regarding the best therapy for individual patients. The present advisory strongly encourages all clinicians involved in the care of appropriate patients with cryptogenic stroke and patent foramen ovale-cardiologists, neurologists, internists, radiologists, and surgeons-to consider referral for enrollment in these landmark trials to expedite their completion and help resolve the uncertainty regarding optimal care for this condition.

Based on a guideline for healthcare professionals from the American Heart Association/American Stroke Association published in 2011, the importance of PFO with or without atrial septal aneurysm for a first stroke or recurrent cryptogenic stroke, remains in question. No randomized controlled clinical trials comparing different medical therapies, medical versus surgical closure, or medical versus transcatheter closure, have been reported, although several studies are ongoing. Non-randomized comparisons of various closure techniques with medical therapy have generally shown reasonable complication rates and recurrence risk with closure at or below those reported with medical therapy. One study suggested a particular benefit in patients with > 1 stroke at baseline.

There is still debate on the possible mechanism of formation of WMLs in migraine. Subjects with migraine with aura (MA) have a two-fold risk of being a carrier of a cardiac right-to-left shunt (RILES) due to PFO compared with the general population. Patent foramen ovale, which can be detected by transcranial Doppler (TCD), is a risk factor for cryptogenic ischemic stroke in young patients, and its prevalence in patients with MA is about 45%.

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

In 2010, Ueno et al. assessed the contribution of embolic etiologies, PFO and atrial septal aneurysm (ASA), to cerebral white matter lesions (WMLs) in ischemic stroke patients. They enrolled 115 patients (age, 69 +/- 11 years; 41 females); 49 (43%) were in the PFO group, 4 (3%) were in the ASA group, 23 (20%) were in the PFO-ASA group, and 39 (34%) were in the non-SA group. The PFO-ASA group had significantly increased WMLs compared to the other three groups ($p = 0.004$). On multiple logistic regression analysis, the coexistence of PFO and ASA was significantly associated with the degree of WMLs (odds ratio: 2.40; 95% confidence interval: 1.11-5.17; $p = 0.026$) when the PFO-ASA and non-SA groups were compared. They concluded that coexistence of PFO with ASA could play an important pathogenic role in WML severity.

Adami et al. performed the Shunt Associated Migraine (SAM) study. One hundred eighty-five patients (77% women) underwent a standardized headache and vascular risk factors questionnaire, contrast-enhanced transcranial Doppler, blood coagulation tests, and brain MRI. RLS was categorized into four grades: no shunt, < 10 microbubbles (mb), > 10 mb single spikes pattern, and > 10 mb shower/curtain pattern. Standard and fluid-attenuated inversion recovery T2- weighted MRI sequences were inspected for WMLs by three independent raters blinded to RLS grade. WML load was scored in the periventricular areas (PV-WMLs) with the Fazekas scale and in the deep white matter (D-WMLs) with the Scheltens scale. Interobserver agreement was good to excellent ($k = 0.64$ to 0.96, $p < 0.0001$). WML load was then correlated between patients with and without RLS. They concluded that the presence of right-to-left shunt does not increase white matter lesion load in patients who have migraine with aura.

In summary, these studies provide new information on options for closure of PFO and generally indicate that short-term complications with these procedures are rare and for the most part minor. Unfortunately, long-term follow-up is lacking. Event rates over 1 to 2 years after transcatheter closure ranged from 0%–3.4%. Studies in which closure was compared with medical treatment alone indicate trends toward better outcomes with closure. Windecker et al. reported a very high 3-year event rate of 33.2% in 44 medically treated patients compared with 7.3% in 59 similar patients treated with PFO-closure. The generally low rates of stroke in the closure series, the lack of robust outcome differences in the 3 non-randomized comparison studies, and the overall absence of controlled comparisons of closure strategies with medical treatment alone, reinforce the need to complete randomized clinical trials comparing closure with medical therapy. A 2009 statement from the AHA/ASA/ACC strongly encourages all clinicians involved in the care of appropriate patients with cryptogenic stroke and PFO-cardiologists, neurologists, internists, radiologists, and surgeons, to consider referral for enrollment in these landmark trials to expedite their completion and help resolve the uncertainty regarding optimal care for this condition.

Billing/Coding Information

CPT CODES

Covered: For the indications outlined above

93580	Percutaneous transcatheter closure of congenital interatrial communication (i.e., Fontan fenestration, atrial septal defect) with implant

HCPCS CODES

C1760	Closure device, vascular (implantable/insertable)
C1817	Septal defect implant system, intracardiac

Key References

1. Adami A, Rossato G, Cerini R, et al. (2008). Right-to-left shunt does not increase white matter lesion load in migraine with aura patients. Neurol 71; 101-107.
2. Alaeddini J, Feghali G, Jenkins S, Ramee S, White C, Abi-Samra F. Frequency of atrial tachyarrhythmias following transcatheter closure of patent foramen ovale. J Invasive Cardiol 18.8 (2006): 365-8.
3. American Academy of Neurology. Recurrent stroke in patients with patent foramen ovale and atrial septal aneurysm. Available: http://www.aan.com/professionals/practice/pdfs/ClinicianStrokeD3.pdf. Date Accessed: June 14, 2007.
4. Anzai H, Child J, Natterson B, et al. Incidence of thrombus formation on the CardioSEAL and the Amplatzer interatrial closure devices. Am J Cardiol 93.4 (2004): 426-31.
5. Anzola GP, Morandi E, Casilli F, Onorato E. Does transcatheter closure of patent foramen ovale really shut the door? A prospective study with transcranial Doppler. Stroke 35.9 (2004): 2140-4.
6. Aslam F, Iliadis AE, Blankenship JC. Percutaneous closure of patent foramen ovale: success and outcomes of a low-volume procedure at a rural medical center. J Invasive Cardiol 19.1 (2007): 20-4.

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

7. Bogousslavsky J, Garazi S, Jeanrenaud X, Aebischer N, Van Melle G. Stroke recurrence in patients with patent foramen ovale: the Lausanne Study. Lausanne Stroke with Paradoxal Embolism Study Group. Neurology 46.5 (1996): 1301-5.
8. Brandt RR, Neumann T, Neuzner J, Rau M, Faude I, Hamm CW. Transcatheter closure of atrial septal defect and patent foramen ovale in adult patients using the Amplatzer occlusion device: no evidence for thrombus deposition with antiplatelet agents. J Am Soc Echocardiogr 15.10 Pt 1 (2002): 1094-8.
9. Braun MU, Fassbender D, Schoen SP, et al. Transcatheter closure of patent foramen ovale in patients with cerebral ischemia. J Am Coll Cardiol 39.12 (2002): 2019-25.
10. Bruch L, Parsi A, Grad MO, et al. Transcatheter closure of interatrial communications for secondary prevention of paradoxical embolism: single-center experience. Circulation 105.24 (2002): 2845-8.
11. Burian M, Neumann T, Weber M, et al. Nickel release, a possible indicator for the duration of antiplatelet treatment, from a nickel cardiac device in vivo: a study in patients with atrial septal defects implanted with an Amplatzer occluder. Int J Clin Pharmacol Ther 44.3 (2006): 107-12.
12. Buscheck F, Sievert H, Kleber F, et al. Patent foramen ovale using the Premere device: the results of the CLOSEUP trial. J Interv Cardiol 19.4 (2006): 328-33.
13. Carandang R, Seshadri S, Beiser A, et al. Trends in incidence, lifetime risk, severity, and 30-day mortality of stroke over the past 50 years. JAMA 296.24 (2006): 2939-46.
14. Cardoso CO, Rossi Filho RI, Machado PR, Francois LM, Horowitz ES, Sarmento-Leite R. Effectiveness of the Amplatzer device for transcatheter closure of an ostium secundum atrial septal defect. Arq Bras Cardiol 88.4 (2007): 384-9.
15. Chatterjee T, Petzsch M, Ince H, et al. Interventional closure with Amplatzer PFO occluder of patent foramen ovale in patients with paradoxical cerebral embolism. J Interv Cardiol 18.3 (2005): 173-9.
16. De Castro S, Cartoni D, Fiorelli M, et al. Morphological and functional characteristics of patent foramen ovale and their embolic implications. Stroke 31.10 (2000): 2407-13.
17. De Ridder S, Suttorp MJ, Ernst SM, et al. Percutaneous transcatheter closure of atrial septal defects: initial single-centre experience and follow-up results. Initial experience with three-dimensional echocardiography. Acta Cardiol 60.2 (2005): 171-8.
18. Del Sette M, Dinia L, Bonzano L, et al. (2008). White matter lesions in migraine and right-to-left shunt: a conventional and diffusion MRI study. Ceph28; 376-382.
19. Demkow M, Ruzyllo W, Kepka C, et al. Transcatheter closure of patent foramen ovale in patients with cryptogenic stroke. Kardiol Pol 61.8 (2004): 101-9; discussion 109.
20. Donti A, Giardini A, Salomone L, Formigari R, Picchio FM. Transcatheter patent foramen ovale closure using the premere PFO occlusion system. Catheter Cardiovasc Interv 68.5 (2006): 736-40.
21. Dorenbeck U, Simon B, Skowasch D, et al. Cerebral embolism with interventional closure of symptomatic patent foramen ovale: an MRI-based study using diffusion-weighted imaging. Eur J Neurol 14.4 (2007): 451-4.
22. Du ZD, Cao QL, Joseph A, et al. Transcatheter closure of patent foramen ovale in patients with paradoxical embolism: intermediate-term risk of recurrent neurological events. Catheter Cardiovasc Interv 55.2 (2002): 189-94.
23. El Said HG, McMahon CJ, Mullins CE, et al. Patent foramen ovale morphology and impact on percutaneous device closure. Pediatr Cardiol 26.1 (2005): 62-5.
24. Food and Drug Administration FDA Panel Summary-PFO Trial Design Issues. 2007. Available: http://www.fda.gov/ohrms/dockets/ac/07/briefing/2007-4284b201.pdf. Date Accessed: May 16, 2007.
25. Food and Drug Administration Information for Physicians and Patients on the Withdrawal of Two Humanitarian Device Exemptions (HDEs) For Patent Foramen Ovale (PFO) Occluders. 2006. Available: http://www.fda.gov/cdrh/ode/h000007-h990011withdraw.html. Date Accessed: December 6, 2006.
26. Furie KL, Kasner SE, Adams RJ, et al. (2011). Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. Jan;42(1):227-76. Epub 2010 Oct 21.
27. Gentile S, Rainero I, Diniele D, et al. (2009). Reversible MRI abnormalities in a patient with recurrent status migrainous. Cephalgia 29. 687-690.
28. Giardini A, Donti A, Formigari R, et al. Comparison of results of percutaneous closure of patent foramen ovale for paradoxical embolism in patients with versus without thrombophilia. Am J Cardiol 94.8 (2004): 1012-6.
29. Goldstein LB, Bushnell CD, Adams RJ, et al. (2011). Guidelines for the primary prevention of stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. Feb;42(2):517-84. Epub 2010 Dec 2.
30. Hara H, Schwartz RS. Patent foramen ovale. 2006. UpToDate. Available: http://www.utdol.com/utd/content/topic.do?topicKey=cong_adu/5471&type=A&selectedTitle=1~26. Date.
31. Hara H, Virmani R, Ladich E, et al. Patent foramen ovale: current pathology, pathophysiology, and clinical status. J Am Coll Cardiol 46.9 (2005): 1768-76.
32. Harms V, Reisman M, Fuller CJ, et al. Outcomes after transcatheter closure of patent foramen ovale in patients with paradoxical embolism. Am J Cardiol 99.9 (2007): 1312-5.
33. Hayes Directory. Transcatheter Closure of Septal Defects. Lansdale, PA: Winifred S. Hayes, Inc., 2002.
34. Herrmann HC, Silvestry FE, Glaser R, et al. Percutaneous patent foramen ovale and atrial septal defect closure in adults: results and device comparison in 100 consecutive implants at a single center. Catheter Cardiovasc Interv 64.2 (2005): 197-203.
35. Homma S, Sacco RL, Di Tullio MR, Sciacca RR, Mohr JP. Effect of medical treatment in stroke patients with patent foramen ovale: patent foramen ovale in Cryptogenic Stroke Study. Circulation 105.22 (2002): 2625-31.
36. Hong TE, Thaler D, Brorson J, Heitschmidt M, Hijazi ZM. Transcatheter closure of patent foramen ovale associated with paradoxical embolism using the amplatzer PFO occluder: initial and intermediate-term results of the U.S. multicenter clinical trial. Catheter Cardiovasc Interv 60.4 (2003): 524-8.
37. Ilkhanoff L, Naidu SS, Rohatgi S, Ross MJ, Silvestry FE, Herrmann HC. Transcatheter device closure of interatrial septal defects in patients with hypoxia. J Interv Cardiol 18.4 (2005): 227-32.
38. Kannan BR, Francis E, Sivakumar K, Anil SR, Kumar RK. Transcatheter closure of very large (>or= 25 mm) atrial septal defects using the Amplatzer septal occluder. Catheter Cardiovasc Interv 59.4 (2003): 522-7.
39. Khositseth A, Cabalka AK, Sweeney JP, et al. Transcatheter Amplatzer device closure of atrial septal defect and patent foramen ovale in patients with presumed paradoxical embolism. Mayo Clin Proc 79.1 (2004): 35-41.

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

40. Kiblawi FM, Sommer RJ, Levchuck SG. Transcatheter closure of patent foramen ovale in older adults. Catheter Cardiovasc Interv 68.1 (2006): 136-42; discussion 143-4.
41. Krumsdorf U, Ostermayer S, Billinger K, et al. Incidence and clinical course of thrombus formation on atrial septal defect and patient foramen ovale closure devices in 1,000 consecutive patients. J Am Coll Cardiol 43.2 (2004): 302-9.
42. Lee PH, Song J-K, Kim JS, Heo R, Lee S, Kim D-H, Song J-M, Kang D-H, Kwon SU, Kang D-W, Lee D, Kwon HS, Yun S-C, Sun BJ, Park J-H, Lee J-H, Jeong HS, Song H-J, Kim J, Park S-J, Cryptogenic Stroke and High-Risk Patent Foramen Ovale: The DEFENSE-PFO Trial, *Journal of the American College of Cardiology* (2018), doi: 10.1016/j.jacc.2018.02.046.
43. Lopez K, Dalvi BV, Balzer D, et al. Transcatheter closure of large secundum atrial septal defects using the 40 mm Amplatzer septal occluder: results of an international registry. Catheter Cardiovasc Interv 66.4 (2005): 580-4.
44. Martin F, Sanchez PL, Doherty E, et al. Percutaneous transcatheter closure of patent foramen ovale in patients with paradoxical embolism. Circulation 106.9 (2002): 1121-6.
45. Mas JL, Arquizan C, Lamy C, et al. Recurrent cerebrovascular events associated with patent foramen ovale, atrial septal aneurysm, or both. N Engl J Med 345.24 (2001): 1740-6.
46. Messé SR, Schwartz RS, Perloff JK. Treatment of atrial septal abnormalities (PFO, ASD, and ASA) for prevention of recurrent stroke in adults. 2006. UpToDate. Available: http://www.utdol.com/utd/content/topic.do?topicKey=cva_dise/19543. Date Accessed: May 15, 2007.
47. Messe SR, Silverman IE, Kizer JR, et al. Practice parameter: recurrent stroke with patent foramen ovale and atrial septal aneurysm: report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 62.7 (2004): 1042-50.
48. Messe, S. R. & Brecker, S. JD. Stroke associated with patent foramen ovale (PFO): Management. UpToDate. Last Review: April 2024.
49. Onorato E, Melzi G, Casilli F, et al. Patent foramen ovale with paradoxical embolism: mid-term results of transcatheter closure in 256 patients. J Interv Cardiol 16.1 (2003): 43-50.
50. Overell JR, Bone I, Lees KR. Interatrial septal abnormalities and stroke: a meta-analysis of case-control studies. Neurology 55.8 (2000): 1172-9.
51. O'Gara PT, Messe SR, Tuzcu EM, et al. (2009). Percutaneous device closure of patent foramen ovale for secondary stroke prevention: a call for completion of randomized clinical trials. A science advisory from the American Heart Association/American Stroke Association and the American College of Cardiology Foundation. J Am Coll Cardiol. May 26;53(21):2014-8.
52. Patel A, Lopez K, Banerjee A, Joseph A, Cao QL, Hijazi ZM. Transcatheter closure of atrial septal defects in adults > or =40 years of age: immediate and follow-up results. J Interv Cardiol 20.1 (2007): 82-8.
53. Post MC, Van Deyk K, Budts W. Percutaneous closure of a patent foramen ovale: single-centre experience using different types of devices and mid-term outcome. Acta Cardiol 60.5 (2005): 515-9.
54. Purcell IF, Brecker SJ, Ward DE. Closure of defects of the atrial septum in adults using the Amplatzer device: 100 consecutive patients in a single center. Clin Cardiol 27.9 (2004): 509-13.
55. Sacco RL, Adams R, Albers G, et al. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: a statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke: co-sponsored by the Council on Cardiovascular Radiology and Intervention: the American Academy of Neurology affirms the value of this guideline. Circulation 113.10 (2006): e409-49.
56. Schneider B, Bauer R. Is surgical closure of patent foramen ovale the gold standard for treating interatrial shunts? An echocardiographic follow-up study. J Am Soc Echocardiogr 18.12 (2005): 1385-91.
57. Schwerzmann M, Windecker S, Wahl A, et al. Percutaneous closure of patent foramen ovale: impact of device design on safety and efficacy. Heart 90.2 (2004): 186-90.
58. Silverman IE, Kiernan FJ, Kelsey AM, et al. Initial experience with a transcatheter septal closure system for secondary stroke prevention in patients with interatrial septal defects. Conn Med 67.3 (2003): 135-44.
59. Slavin L, Tobis JM, Rangarajan K, Dao C, Krivokapich J, Liebeskind DS. Five-year experience with percutaneous closure of patent foramen ovale. Am J Cardiol 99.9 (2007): 1316-20.
60. Spies C, Strasheim R, Timmermanns I, Schraeder R. Patent foramen ovale closure in patients with cryptogenic thrombo-embolic events using the Cardia PFO occluder. Eur Heart J 27.3 (2006): 365-71.
61. Spies C, Timmermanns I, Schrader R. Transcatheter closure of secundum atrial septal defects in adults with the Amplatzer septal occluder: Intermediate and long-term results. Clin Res Cardiol 96.6 (2007): 340-6.
62. Thanopoulos BV, Dardas PD, Karanasios E, Mezilis N. Transcatheter closure versus medical therapy of patent foramen ovale and cryptogenic stroke. Catheter Cardiovasc Interv 68.5 (2006): 741-6.
63. Varma C, Benson LN, Warr MR, et al. Clinical outcomes of patent foramen ovale closure for paradoxical emboli without echocardiographic guidance. Catheter Cardiovasc Interv 62.4 (2004): 519-25.
64. Wang JK, Tsai SK, Wu MH, Lin MT, Lue HC. Short- and intermediate-term results of transcatheter closure of atrial septal defect with the Amplatzer Septal Occluder. Am Heart J 148.3 (2004): 511-7.
65. Wiegers SE, Sutton MGSJ. Pathophysiology and clinical features of atrial septal defects in adults. 2006. UpToDate. Available: http://www.utdol.com/utd/content/topic.do?topicKey=congadu/4835&type=A&selectedTitle=3~42. Date Accessed: June 4, 2007.
66. Windecker S, Wahl A, Nedeltchev K, et al. Comparison of medical treatment with percutaneous closure of patent foramen ovale in patients with cryptogenic stroke. J Am Coll Cardiol 44.4 (2004): 750-8.
67. Woods TD, Patel A. A critical review of patent foramen ovale detection using saline contrast echocardiography: when bubbles lie. J Am Soc Echocardiogr 19.2 (2006): 215-22.
68. Yew G, Wilson NJ. Transcatheter atrial septal defect closure with the Amplatzer septal occluder: five-year follow-up. Catheter Cardiovasc Interv 64.2 (2005): 193-6.
69. Zajarias A, Thanigaraj S, Lasala J, Perez J. Predictors and clinical outcomes of residual shunt in patients undergoing percutaneous transcatheter closure of patent foramen ovale. J Invasive Cardiol 18.11 (2006): 533-7.

Select Health

Percutaneous Transcatheter Closure for the Treatment of Atrial Septal Defects (ASD) and Patent Foramen Ovale (PFO), continued

Revision History

Revision Date	Summary of Changes
5/2/24	For Commercial Plan Policy, clarified requirements in criterion #1: "Patient has a documented history of ***recurrent, cryptogenic, clinically-evident transient ischemic episode or recurrent, cryptogenic stroke***, which has been verified by an independent, qualified neurologic specialist."
5/17/24	For Commercial Plan Policy, removed "recurrent" as a requirement in criterion #1, and included requirement for provider to document the PASCAL classification system and RoPE scores.
11/6/24	For Commercial Plan Policy, clarified that both criterion #1 and #2 must be met to qualify for coverage.

Select Health

Select Health

MEDICAL POLICY

PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY STENTING (PTAS) FOR THE TREATMENT OF INTRACEREBRAL DISEASE

Policy # 495
Implementation Date:12/5/11
Review Dates: 7/18/13, 6/19/14, 6/11/15, 6/16/16, 6/15/17, 9/18/18, 8/7/19, 8/20/20, 8/19/21, 7/21/22, 7/28/23, 8/22/24
Revision Dates:

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description
Cerebrovascular disease (CVD) refers to a group of conditions that affect the circulation of blood to the brain, potentially limiting blood flow to affected areas of the brain. This can manifest itself as either a stroke or transient ischemic attack (TIA).

Standard therapy for patients with atherosclerotic disease is antiplatelet therapy with aspirin or other antiplatelet agents and control of other risk factors. Intracerebral stenting of identified atherosclerotic lesions is also offered to some patients. The stent and delivery catheter consist of an expandable stainless-steel device that provides structural support for a blood vessel, helping to keep it open. The stent is a self-expanding, metal (nitinol) mesh in the shape of a tube. It is intended for use in the treatment of patients with recurrent intracranial stroke due to atherosclerotic disease who did not respond to medical therapy.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health does NOT cover percutaneous transluminal angioplasty stenting (PTAS) in the treatment of intracranial disease. The current evidence has not demonstrated this treatment as a clinical benefit for the initial therapy of intracerebral atherosclerotic disease.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Percutaneous Transluminal Angioplasty Stenting (PTAS) for the Treatment of Intracerebral Disease, continued

Summary of Medical Information

A Medical Technology Assessment performed in October 2011 identified 4 systematic reviews. All systematic reviews were published in 2006 or 2007 and no updates are available and long-term outcomes were not presented. Questions concerning durability and success of 2 different stents were raised. All concluded that current evidence at the time of their reviews was insufficient to warrant broad coverage outside an investigational setting.

Seventeen peer-reviewed papers studying PTAS were identified. These studies were small in size and supported the use of stenting in patients with > 50% atherosclerotic narrowing of any intracerebral vessel with significant success and relatively low risk of complications. For example, the largest study by Jiang, involved 637 patients and 670 lesions. It was a retrospective study comparing 2 different stents. A relatively low risk of complications, 6.1% within 30 days of the procedure was demonstrated. This study's limitations include the short duration of follow-up (1 month), and the lack of randomization or blinding.

The most recent article on this topic was a randomized controlled multi-centered study published in September 2011 by Chimowitz et al. This study compared percutaneous transluminal angioplasty and stenting (PTAS) with aggressive medial management in patients with 70%–99% narrowing. The 2 groups had similar initial clinical findings, and both achieved similar outcomes to medical management including improved blood pressure control, LDL reduction, and diabetic control. Aspirin and clopidogrel were used. The interventional group developed a much higher rate of complications including hemorrhagic stroke and death within the first 30 days. The medical group also achieved a lower rate of stroke reduction than what was expected compared with previous studies evaluating the role of medical therapy in the prevention of recurrent stroke. "The 30-day rate of stroke or death in the PTAS group (14.7) is substantially higher than the rates previously reported with the use of the Wingspan stent in the Phase I trial and in 2 registries (rates ranging from 4.4-9.6)." The rate of stroke was much lower at 5.5% with expected rate of 10.7%. The expected 1-year rate of stroke or death was 25% and realized rate in this study was 12.2%. These results were carefully analyzed to attempt to explain the difference in realized and expected outcomes.

Despite experienced interventional radiologists, similar initial clinical parameters, and similar results in aggressive medical management between the 2 groups, the much higher complication rate within the first 30 days of the procedure which included stroke and death led to the early suspension of this trial.

Also discouraging was that 4% of patients (total of 9) who crossed over from the medical group to the PTAS group due to recurrent strokes also had a high rate of complications. Three of the 9 patients suffered a stroke after PTAS within 30 days. Despite attempts from stent manufacturers to minimize restenosis the rate of restenosis occurs "23%-30% within 6 months after intracranial PTAS and could also lead to later stroke."

In conclusion, the most recent large clinical trial, Stenting and Aggressive Medical Management for Preventing Recurrent stroke in Intracranial Stenosis (SAMMPRIS), 14% of patients treated with angioplasty combined with stenting experienced a stroke or died within the first 30 days after enrollment compared with 5.8% of patients treated with medical therapy alone. Even though there have been many studies dating back to 2002, the literature has shown for patients with intracranial arterial stenosis, aggressive medical management was superior to PTAS with the use of either the Wingspan or the Neurolink stent systems. The risks of early stroke after PTAS were high and the risk of stroke with aggressive medical therapy alone was lower than expected. Therefore, the use of PTAS with or without medical management does not seem to be a reliable form of treatment for intracranial stroke.

Billing/Coding Information

Not covered: Investigational/Experimental/Unproven for this indication

CPT CODES

61635	Transcatheter placement of intravascular stent(s), intracranial (e.g., atherosclerotic stenosis), including balloon angioplasty, if performed

HCPCS CODES

C1874	Stent, coated/covered, with delivery system
C1875	Stent, coated/covered, without delivery system

Percutaneous Transluminal Angioplasty Stenting (PTAS) for the Treatment of Intracerebral Disease, continued

C1876 Stent, non-coated/non-covered, with delivery system

C1877 Stent, non-coated/non-covered, without delivery system

Key References

1. Abruzzo, TA, Tong, FC, Waldrop, AS, et al. (2007). Basilar artery stent angioplasty for symptomatic intracranial athero-occlusive disease: complications and late midterm clinical outcomes. AJNR Am J Neuroradiol 28.5: 808-15.
2. Albers, GW, Caplan, LR, Easton, JD, et al. (2002). Transient ischemic attack--proposal for a new definition. N Engl J Med 347.21: 1713-6.
3. Australia and New Zealand Horizon Scanning Network (ANZHSN). (2007). Intracranial angioplasty and stenting (WingSpan™) for cerebral atherosclerotic stenosis.
4. Bang, JS, Oh, CW, Jung, C, et al. (2010). Intracranial stent placement for recanalization of acute cerebrovascular occlusion in 32 patients. AJNR Am J Neuroradiol 31.7: 1222-5.
5. Bennett, CL, Connors, JM, Carwile, JM, et al. (2000). Thrombotic thrombocytopenic purpura associated with clopidogrel. N Engl J Med 342.24: 1773-7.
6. Caplan, LR. (2011) Overview of the evaluation of stroke. March 24, 2011. Up to Date. Available: http://www.uptodate.com/contents/overview-of-the-evaluation-of-stroke?source=search_result&search=cerebrovascular+disease&selectedTitle=1~150. Date Accessed: September 28, 2011.
7. Chimowitz, MI, Lynn, MJ, Derdeyn, CP, et al. (2011). Stenting versus aggressive medical therapy for intracranial arterial stenosis. N Engl J Med 365.11: 993-1003.
8. Creager, MA. (2006) Vascular Medicine: A Companion to Braunwald's Heart Disease. Saunders Elseiver. Available: http://www.mdconsult.com/books/page.do?eid=4-u1.0-B978-0-7216-0284-4.50001-4&isbn=978-0-7216-0284-4&uniqId=283830556-8#4-u1.0-B978-0-7216-0284-4.50001-4. Date Accessed: September 26, 2011.
9. Cronenwett, JL. (2010) Cronenwett: Rutherford's Vascular Surgery, 7th ed. Saunders Elseiver. Available: http://www.mdconsult.com/books/page.do?eid=4-u1.0-B978-1-4160-5223-4.00092-5--s0015&isbn=978-1-4160-5223-4&sid=1210424548&uniqId=283830556-5#4-u1.0-B978-1-4160-5223-4.0092-5--s0015. Date Accessed: September 26, 2011.
10. Cruz-Flores, S, Diamond, AL. (2006). Angioplasty for intracranial artery stenosis. Cochrane Database Syst Rev 3: CD004133.
11. Cucchiara, BL, Messé, SR. (2011) Antiplatelet therapy for secondary prevention of stroke. 19.2. April 4, 2011. UpToDate. Available: http://www.uptodate.com/contents/antiplatelet-therapy-for-secondary-prevention-of-stroke?source=search_result&search=stroke+prevention&selectedTitle=1%7E150#H17. Date Accessed: October 23, 2011
12. Del Zoppo, GJ, Saver, JL, Jauch, EC, et al. (2009). Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: a science advisory from the American Heart Association/American Stroke Association. Stroke 40.8: 2945-8.
13. Demarin, V, Lisak, M, Morovic, S, et al. (2010). Low high-density lipoprotein cholesterol as the possible risk factor for stroke. Acta Clin Croat 49.4: 429-39.
14. Endres, M, Heuschmann, PU, Laufs, U, et al. (2011). Primary prevention of stroke: blood pressure, lipids, and heart failure. Eur Heart J 32.5: 545-52.
15. FDA. (2009) NEUROLINK® System - H010004. September 8, 2009. FDA. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm083307.htm. Date Accessed: September 30, 2011.
16. FDA. (2009) Wingspan™ Stent System with Gateway™ PTA Balloon Catheter - H50001. June 30, 2009. FDA. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm078508.htm. Date Accessed: September 30, 2011.
17. Foundation for Education and Research in Neurological Emergencies. (2011) Efficient ED Neurological Exam, CT Diagnosis, and Documentation. FERNE. Available: http://www.ferne.org/Lectures/acep2005_spring/sloan_acep2005_spring_neuroexam.htm. Date Accessed: October 13, 2011.
18. Gupta, R, Schumacher, HC, Mangla, S, et al. (2003). Urgent endovascular revascularization for symptomatic intracranial atherosclerotic stenosis. Neurology 61.12: 1729-35.
19. Hauth, EA, Gissler, HM, Drescher, R, et al. (2004). Angioplasty or stenting of extra- and intracranial vertebral artery stenoses. Cardiovasc Intervent Radiol 27.1: 51-7.
20. Higashida, RT, Meyers, PM, Connors, JJ, 3rd, et al. (2005). Intracranial angioplasty & stenting for cerebral atherosclerosis: a position statement of the American Society of Interventional and Therapeutic Neuroradiology, Society of Interventional Radiology, and the American Society of Neuroradiology. AJNR Am J Neuroradiol 26.9: 2323-7.
21. International Stroke Trial (IST). (1997).: a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischaemic stroke. International Stroke Trial Collaborative Group. Lancet 349.9065: 1569-81.
22. Jeon, P, Kim, BM, Kim, DI, et al. (2010). Emergent self-expanding stent placement for acute intracranial or extracranial internal carotid artery dissection with significant hemodynamic insufficiency. AJNR Am J Neuroradiol 31.8: 1529-32.
23. Jiang, WJ, Cheng-Ching, E, Abou-Chebl, A, et al. (2011). Multi-Center Analysis of Stenting in Symptomatic Intracranial Atherosclerosis. Neurosurgery.
24. Kelly, PJ. Mild-to-moderate hyperhomocystin(e)inemia and risk of stroke. Abstracts of the International Stroke Conference. Presentation at the International Stroke Conference: Stroke; 2000: 366.
25. Kernan, W. N., B. Ovbiagele, H. R. Black, D. M. Bravata, M. I. Chimowitz, M. D. Ezekowitz, M. C. Fang, M. Fisher, K. L. Furie, D. V. Heck, S. C. Johnston, S. E. Kasner, S. J. Kittner, P. H. Mitchell, M. W. Rich, D. Richardson, L. H. Schwamm, J. A. Wilson, C. o. C. American Heart Association Stroke Council, C. o. C. C. Stroke Nursing and D. Council on Peripheral Vascular (2014). "Guidelines for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association." Stroke 45(7): 2160-2236.
26. Lai, Z., et al. Percutaneous transluminal angioplasty and stenting vs aggressive medical management on stroke or intracranial atherosclerotic stenosis: a systematic review and meta-analysis. *Sci Rep*. 2023; 13: 7567.
27. Life Extension Foundation. (2011) Stroke and cerebrovascular disease stroke and prevention. Life Extension Foundation. Available: http://www.lef.org/protocols/heart_circulatory/stroke_cerebrovascular_disease_01.htm. Date Accessed: September 28, 2011.

Select Health

Percutaneous Transluminal Angioplasty Stenting (PTAS) for the Treatment of Intracerebral Disease, continued

28. Lin, R, Aleu, A, Jankowitz, B, et al. (2010). Endovascular Revascularization of Chronic Symptomatic Vertebrobasilar Occlusion. J Neuroimaging.
29. Lylyk, P, Vila, JF, Miranda, C, et al. (2005). Endovascular reconstruction by means of stent placement in symptomatic intracranial atherosclerotic stenosis. Neurol Res 27 Suppl 1: S84-8.
30. Marks, MP, Marcellus, ML, Do, HM, et al. (2005). Intracranial angioplasty without stenting for symptomatic atherosclerotic stenosis: long-term follow-up. AJNR Am J Neuroradiol 26.3: 525-30.
31. Mayo Clinic. (2011) Stroke. Mayo Clinic. Available: http://www.mayoclinic.com/health/stroke/DS00150/DSECTION=prevention. Date Accessed: September 28, 2011.
32. Meyers, PM, Schumacher, HC, Higashida, RT, et al. (2009). Indications for the performance of intracranial endovascular neurointerventional procedures: a scientific statement from the American Heart Association Council on Cardiovascular Radiology and Intervention, Stroke Council, Council on Cardiovascular Surgery and Anesthesia, Interdisciplinary Council on Peripheral Vascular Disease, and Interdisciplinary Council on Quality of Care and Outcomes Research. Circulation 119.16: 2235-49.
33. National Institute for Health (NIH). (2011) Clinical Alert: Angioplasty Combined with Stenting Plus Aggressive Medical Therapy vs. Aggressive Medical Therapy Alone for Intracranial Arterial Stenosis: NINDS Stops Trial Enrollment Due to a Higher Risk of Stroke and Death in the Stented Group. June 16, 2011. US National Library of Medicine. Available: http://www.nlm.nih.gov/databases/alerts/intracranial_arterial_stenosis.html. Date Accessed: October 24, 2011.
34. National Institute for Health and Clinical Excellence (NICE). (2007). Endovascular stent insertion for intracranial atherosclerotic disease.
35. National Stroke Association. (2011) High Blood Pressure (Hypertension). National Stroke Association. Available: http://www.stroke.org/site/PageServer?pagename=highbloodpressure. Date Accessed: September 30, 2011.
36. Park, SI, Kim, BM, Kim, DI, et al. (2009). Clinical and angiographic follow-up of stent-only therapy for acute intracranial vertebrobasilar dissecting aneurysms. AJNR Am J Neuroradiol 30.7: 1351-6.
37. Povedano, G, Zuberbuhler, P, Lylyk, P, et al. (2010). Management strategies in posterior circulation intracranial atherosclerotic disease. J Endovasc Ther 17.3: 308-13.
38. Rakel, R. (2011) Rakel: Textbook of Family Medicine, 8th ed. 6. Elseiver Saunders. Available: http://www.mdconsult.com/books/page.do?eid=4-u1.0-B978-1-4377-1160-8.10042-9--s0130&isbn=978-1-4377-1160-8&uniqId=284399105-13#4-u1.0-B978-1-4377-1160-8.10042-9--s0130. Date Accessed: September 30, 2011.
39. Ridker, PM, Cushman, M, Stampfer, MJ, et al. (1997). Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. N Engl J Med 336.14: 973-9.
40. Riedel, CH, Tietke, M, Alfke, K, et al. (2009). Subacute stent thrombosis in intracranial stenting. Stroke 40.4: 1310-4.
41. Roth, C, Papanagiotou, P, Behnke, S, et al. (2010). Stent-assisted mechanical recanalization for treatment of acute intracerebral artery occlusions. Stroke 41.11: 2559-67.
42. Sacco, RL, Prabhakaran, S, Thompson, JL, et al. (2006). Comparison of warfarin versus aspirin for the prevention of recurrent stroke or death: subgroup analyses from the Warfarin-Aspirin Recurrent Stroke Study. Cerebrovasc Dis 22.1: 4-12.
43. Sainani, GS, Sainani, R. (2002). Homocysteine and its role in the pathogenesis of atherosclerotic vascular disease. J Assoc Physicians India 50 Suppl: 16-23.
44. Saver, JL, Kalaft, M. (2011) Thrombolytic Therapy in Stroke March 29, 2011. Medscape Reference. Available: http://emedicine.medscape.com/article/1160840-overview. Date Accessed: October 18, 2011.
45. Suh, DC, Kim, JK, Choi, JW, et al. (2008). Intracranial stenting of severe symptomatic intracranial stenosis: results of 100 consecutive patients. AJNR Am J Neuroradiol 29.4: 781-5.
46. Technology Assessment Committee. (2006) Carotid, Vertebral and Intracranial Artery Angioplasty and Stenting TA #93.
47. Toole, JF, Malinow, MR, Chambless, LE, et al. (2004). Lowering homocysteine in patients with ischemic stroke to prevent recurrent stroke, myocardial infarction, and death: the Vitamin Intervention for Stroke Prevention (VISP) randomized controlled trial. JAMA 291.5: 565-75.
48. Vajda, Z, Miloslavski, E, Guthe, T, et al. (2010). Treatment of intracranial atherosclerotic arterial stenoses with a balloon-expandable cobalt chromium stent (Coroflex Blue): procedural safety, efficacy, and midterm patency. Neuroradiology 52.7: 645-51.
    Wald, DS, Law, M, Morris, JK. (2002). Homocysteine and cardiovascular disease: evidence on causality from a meta-analysis. BMJ 325.7374: 1202.
49. Wehrschuetz, M, Wehrschuetz, E, Augustin, M, et al. (2011). Early single center experience with the solitaire thrombectomy device for the treatment of acute ischemic stroke. Interv Neuroradiol 17.2: 235-40.
50. Zhang, J, Zhang, R, Wu, Z, et al. (2010). Results of endovascular management for mid-basilar artery aneurysms. Interv Neuroradiol 16.3: 249-54.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Select Health

MEDICAL POLICY

SUBCUTANEOUS IMPLANTABLE DEFIBRILLATOR

Policy # 535
Implementation Date:8/9/13
Review Dates: 8/28/14, 8/20/15, 8/25/16, 8/17/17, 7/25/18, 6/13/19, 6/18/20, 8/11/21, 7/21/22, 8/17/23, 8/11/24
Revision Dates: 8/24/21

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Tachyarrhythmias are broadly characterized as supraventricular tachycardia (SVT), defined as a tachycardia in which the driving circuit or focus originates, at least in part, in tissue above the level of the ventricle (i.e., sinus node, atria, AV node, or the bundle of His) and ventricular tachycardia (VT), defined as a tachycardia in which the driving circuit or focus solely originates in ventricular tissue or Purkinje fibers. Sustained arrhythmias are an important cause of sudden death. Ventricular arrhythmias that occur in the absence of structural heart disease or a defined ion channel abnormality are referred to as *idiopathic* and are usually benign. Ventricular arrhythmias are responsible for most of the 150,000 to 350,000 sudden deaths that occur annually in the United States and account for approximately 13% of all mortality. Without immediate action, ventricular tachycardia can degenerate to ventricular fibrillation and lead to sudden death. Short-term goals of treatment are to stabilize hemodynamically unstable patients, transfer them to a hospital immediately, and stop ventricular tachycardia.

Transvenous implantable cardiac defibrillators (ICDs) have been used for years and are designed to detect a life-threatening, rapid heartbeat emanating from the lower chamber of the heart, and then deliver an electrical discharge intended to terminate the rhythm so that it is converted back to normal. Conventional ICDs consist of a generator, which is usually implanted in a pocket in the pectoral region below the left shoulder. The transvenous right ventricular lead contains the shock coils and pacing electrode. Additional leads may be connected for right atrial or left ventricular pacing, sensing, and defibrillation. The ICD can be implanted under local anesthesia with the leads inserted through an incision into a vein and guided to the heart under fluoroscopy. The lead is attached to the heart muscle, while the other end of the lead is attached to the pulse generator.

Subcutaneous implantable defibrillators (S-ICD) have recently been approved in an effort to reduce morbidity and procedure-related complications. The S-ICD consists of an electrically active pulse generator, which is implanted near the left mid-axillary line and a subcutaneous lead, consisting of sensing electrodes and a shocking coil, which is tunneled 1 to 2 cm to the left of the mid-sternal line. The current FDA approved S-ICD weighs 145 grams and has a lithium battery with a projected life of 5 years; therapy consists of 80-joule biphasic transthoracic shocks and 30 seconds of post-shock pacing.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers subcutaneous implantable defibrillators for members 10 years of age and older, who require a transvenous cardiac defibrillator for ventricular tachyarrhythmias for the treatment of life-threatening ventricular tachyarrhythmias.

Subcutaneous Implantable Defibrillator, continued

Select Health does not cover subcutaneous implantable defibrillators for patients who have symptomatic bradycardia, incessant ventricular tachycardia, or spontaneous, frequently recurring ventricular tachycardia, that is reliably terminated with antitachycardia pacing.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

A recent review of subcutaneous implantable cardiac defibrillators (S-ICDs) identified eight peer-reviewed published studies that met inclusion criteria for this report. No systematic reviews on the safety, efficacy, or cost-effectiveness of subcutaneous implantable defibrillators have been published. These studies had a mean duration ranging from 7.2 to 22.3 months. The studies documented efficacy of 89.6% to 100% in aborting dysrhythmias. These studies also identified inappropriate shocks occurring from 0% to 22% of the time. These numbers compare favorably to historical rates for efficacy and inappropriate shocks for transvenous ICDs which have demonstrated inappropriate shock rates as high as 30%.

In general, single shocks, whether preceded by symptoms or not, are most often caused by the appropriate detection and treatment of a ventricular tachyarrhythmia. Conversely, multiple transvenous ICD discharges often result from the detection of other arrhythmias or signals that are inaccurately classified as a ventricular tachyarrhythmia. Thus, transvenous ICD therapy often is inappropriately delivered for sinus tachycardia, or other supraventricular tachycardias with rapid AV conduction. To this point, Jarman et al., Kobe et al., and Olde Nordkamp et al., all noted that inappropriate shocks (5.6% of all shocks) were associated with T-wave oversensing in patients with S-ICDs. During the MADIT II Trial of transvenous ICDs, Daubert et al. found that one or more inappropriate shocks occurred in 11.5% of the 719 patients enrolled. No cost-effectiveness studies have been completed comparing S-ICD to either transvenous ICD or to medical treatment.

Using the Grade evidence system, which rates the body of evidence Grade 1−3, with additional rankings of A−C, the current body of evidence has a Grade evidence level of 2B. This is primarily due to the limitations in evidence, where risks and magnitude of benefits appear to be finely balanced in comparison to medical treatment, or conventional ICD placement, and where the quality of evidence is moderate (cohort studies with short follow-up times).

Billing/Coding Information

CPT CODES

33240 Insertion of implantable defibrillator pulse generator only; with existing single lead

33241 Removal of implantable defibrillator pulse generator only

33262 Removal of implantable defibrillator pulse generator with replacement of implantable defibrillator pulse generator; single lead system

33263 Removal of implantable defibrillator pulse generator with replacement of implantable defibrillator

Subcutaneous Implantable Defibrillator, continued

pulse generator; dual lead system

33264 Removal of implantable defibrillator pulse generator with replacement of implantable defibrillator pulse generator; multiple lead system

33270 Insertion or replacement of permanent subcutaneous implantable defibrillator system, with subcutaneous electrode, including defibrillation threshold evaluation, induction of arrhythmia, evaluation of sensing for arrhythmia termination, and programming or reprogramming of sensing or therapeutic parameters, when performed

33271 Insertion of subcutaneous implantable defibrillator electrode

33272 Removal of subcutaneous implantable defibrillator electrode

33273 Repositioning of previously implanted subcutaneous implantable defibrillator electrode

93260 Programming device evaluation (in person) with iterative adjustment of the implantable device to test the function of the device and select optimal permanent programmed values with analysis, review and report by a physician or other qualified health care professional; implantable subcutaneous lead defibrillator system

93261 Interrogation device evaluation (in person) with analysis, review and report by a physician or other qualified health care professional, includes connection, recording and disconnection per patient encounter; implantable subcutaneous lead defibrillator system

HCPCS CODES

C1721 Cardioverter-defibrillator, dual chamber (implantable)

C1722 Cardioverter-defibrillator, single chamber (implantable)

C1882 Cardioverter-defibrillator, other than single or dual chamber (implantable)

C1899 Lead, pacemaker/cardioverter-defibrillator combination (implantable)

G0448 Insertion or replacement of a permanent pacing cardioverter-defibrillator system with transvenous lead(s), single or dual chamber with insertion of pacing electrode, cardiac venous system, for left ventricular pacing

Key References

1. A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. (1997). N Engl J Med. 337.22: 1576-83.
2. ACCF/HRS/AHA/ASE/HFSA/SCAI/SCCT/SCMR. (2013). Appropriate use criteria for implantable cardioverter-defibrillators and cardiac resynchronization therapy. American College of Cardiology.
3. Administration, USFaD. (2012) Subcutaneous Implantable Defibrillator (S-ICD) System - P110042. November 7, 2012. U.S. Department of Health & Human Services. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm326541.htm. Date Accessed: April 19, 2013.
4. Aydin, A, Hartel, F, Schluter, M, et al. (2012). Shock efficacy of subcutaneous implantable cardioverter-defibrillator for prevention of sudden cardiac death: initial multicenter experience. Circ Arrhythm Electrophysiol 5.5: 913-9.
5. Bardy, GH, Hofer, B, Johnson, G, et al. (1993). Implantable transvenous cardioverter-defibrillators. Circulation 87.4: 1152-68.
6. Bardy, GH, Smith, WM, Hood, MA, et al. (2010). An entirely subcutaneous implantable cardioverter-defibrillator. N Engl J Med 363.1: 36-44.
7. Borek, PP. (2011) Ventricular Tachycardia. October 7, 2011. Elsevier. Available: https://www.clinicalkey.com/#!/ContentPlayerCtrl/doPlayContent/21-s2.0-1011028/{"scope":"all","query":"ventricular tachyarrhythmia"}. Date Accessed: April 17, 2013.
8. Boston Scientific. (2013) Introducing the S-ICD® System, the world's first and only Subcutaneous Implantable Defibrillator. Boston Scientific. Available: http://www.bostonscientific.com/cardiac-rhythm-resources/cameron-health/sicd-system.html. Date Accessed: April 19, 2013.
9. British Cardiovascular Society. (2013) The Entirely Subcutaneous ICD (S-ICD). January 11, 2013. British Cardiovascular Society. Available: http://www.bcs.com/pages/news_full.asp?NewsID=19792120. Date Accessed: April 10, 2013.
10. Callans, DJ, Hook, BG, Marchlinski, FE. (1991). Use of bipolar recordings from patch-patch and rate sensing leads to distinguish ventricular tachycardia from supraventricular rhythms in patients with implantable cardioverter defibrillators. Pacing Clin Electrophysiol 14.11 Pt 2: 1917-22.
11. Cameron Health. (2013) About the Procedure. Cameron Health. Available: http://www.cameronhealth.com/understand-your-options/about-the-procedure/. Date Accessed: May 31, 2013.
12. Compton, SJ. (2013) Ventricular Tachycardia Clinical Presentation. February 25, 2013. Medscape. Available: http://emedicine.medscape.com/article/159075-clinical. Date Accessed: May 20, 2013.

Subcutaneous Implantable Defibrillator, continued

13. Dabiri Abkenari, L, Theuns, DA, Valk, SD, et al. (2011). Clinical experience with a novel subcutaneous implantable defibrillator system in a single center. Clin Res Cardiol 100.9: 737-44.
14. Daubert, JP, Zareba, W, Cannom, DS, et al. (2008). Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: frequency, mechanisms, predictors, and survival impact. J Am Coll Cardiol 51.14: 1357-65.
15. EuroScan International Network. (2013) Subcutaneous Implantable Cardioverter-defibrillator (ICD). EuroScan International Network. Available: http://euroscan.org.uk/technologies/technology/view/1704. Date Accessed: May 24, 2013.
16. Goldman, L. (2012) Cecil Medicine. 24. Elseiver. Available: https://www.clinicalkey.com/#!/ContentPlayerCtrl/doPlayContent/3-s2.0-B9781437716047000658/{"scope":"all","query":"Ventricular Tachycardia"}. Date Accessed: April 17, 2013.
17. Greene, HL, Roden, DM, Katz, RJ, et al. (1992). The Cardiac Arrhythmia Suppression Trial: first CAST ... then CAST-II. J Am Coll Cardiol 19.5: 894-8.
18. Griksaitis, MJ, Rosengarten, JA, Gnanapragasam, JP, et al. (2013). Implantable cardioverter defibrillator therapy in paediatric practice: a single-centre UK experience with focus on subcutaneous defibrillation. Europace 15.4: 523-30.
19. Hauser, RG. (2013). The subcutaneous implantable cardioverter-defibrillator: should patients want one? J Am Coll Cardiol 61.1: 20-2.
20. Hohnloser, SH, Kuck, KH, Dorian, P, et al. (2004). Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med 351.24: 2481-8.
21. Jarman, JW, Todd, DM. (2013). United Kingdom national experience of entirely subcutaneous implantable cardioverter-defibrillator technology: important lessons to learn. Europace.
22. Kaltman, JR, Gaynor, JW, Rhodes, LA, et al. (2007). Subcutaneous array with active can implantable cardioverter defibrillator configuration: a follow-up study. Congenit Heart Dis 2.2: 125-9.
23. Kobe, J, Reinke, F, Meyer, C, et al. (2013). Implantation and follow-up of totally subcutaneous versus conventional implantable cardioverter-defibrillators: a multicenter case-control study. Heart Rhythm 10.1: 29-36.
24. Medicine, HDAToC. (2012) Heart Disease: A Textbook of Cardiovascular Medicine. 9. Elsevier. Available: https://www.clinicalkey.com/#!/ContentPlayerCtrl/doPlayContent/3-s2.0-B9781437703986000391/{"scope":"all","query":"Ventricular Tachycardia"}. Date Accessed: April 17, 2013.
25. Olde Nordkamp, LR, Dabiri Abkenari, L, Boersma, LV, et al. (2012). The entirely subcutaneous implantable cardioverter-defibrillator: initial clinical experience in a large Dutch cohort. J Am Coll Cardiol 60.19: 1933-9.
26. Russo, AM, Stainback, RF, Bailey, SR, et al. (2013). ACCF/HRS/AHA/ASE/HFSA/SCAI/SCCT/SCMR 2013 appropriate use criteria for implantable cardioverter-defibrillators and cardiac resynchronization therapy: a report of the American College of Cardiology Foundation appropriate use criteria task force, Heart Rhythm Society, American Heart Association, American Society of Echocardiography, Heart Failure Society of America, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, and Society for Cardiovascular Magnetic Resonance. J Am Coll Cardiol 61.12: 1318-68.
27. Saksena, S. (2012) Electrophysiological Disorders of the Heart, Second Edition. 2. Elsevier. Available: https://www.clinicalkey.com/#!/ContentPlayerCtrl/doPlayContent/3-s2.0-B9781437702859000879/{"scope":"onlyThisSpecificBookChapter","query":"effectiveness"}. Date Accessed: May 23, 2013.
28. Silvetti, M. S., Pazzano, V., Verticelli, L., Battipaglia, I., Saputo, F. A., Albanese, S. ... Drago, F. (2018). Subcutaneous implantable cardioverter-defibrillator: is it ready for use in children and young adults? A single-centre study. *European Society of Cardiology*. 20: 1966-1973. doi:10.1093/europace/euy139
29. Waldo, AL, Camm, AJ, deRuyter, H, et al. (1996). Effect of d-sotalol on mortality in patients with left ventricular dysfunction after recent and remote myocardial infarction. The SWORD Investigators. Survival With Oral d-Sotalol. Lancet 348.9019: 7-12.
30. W., S. (2012) Subcutaneous Implantable Cardiac Defibrillator FDA Approved. October 1, 2012. MedGadget. Available: http://www.medgadget.com/2012/10/subcutaneous-implantable-cardiac-defibrillator-fda-approved.html. Date Accessed: April 19, 2013.

Select Health

Select Health

MEDICAL POLICY

TOTAL ARTIFICIAL HEART

Policy # 436
Implementation Date: 3/22/10
Review Dates: 8/16/11, 8/15/13, 8/28/14, 8/20/15, 8/25/16, 12/15/16, 8/17/17, 7/20/18, 6/13/19, 6/18/20, 7/12/21, 7/21/22, 8/17/23, 8/9/24
Revision Dates: 8/16/12, 10/19/15

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description
Heart failure (HF) is a common complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.

Heart failure is classified based upon the history, including assessment of New York Heart Association (NYHA) functional class, and physical examination in conjunction with certain diagnostic tests. Both establish the primary cause of the heart failure and provide a reasonable estimate of its severity. The NYHA classification system, most used to quantify the degree of functional limitation imposed by HF, was first developed by the New York Heart Association (NYHA). This system assigns patients to one of four functional classes, depending on the degree of effort needed to elicit symptoms.

- Class I - symptoms of HF only at activity levels that would limit normal individuals
- Class II - symptoms of HF with ordinary exertion
- Class III - symptoms of HF with less than ordinary exertion
- Class IV - symptoms of HF at rest

Additionally, heart failure has 4 stages (Stage A – Stage D) that reflect the development and progression of the condition:

- Stage A: at high risk for HF but without structural heart disease or symptoms of HF, recommended treatment to control the risk factor and close monitoring.
- Stage B: Structural heart disease but without signs or symptoms of HF, recommended treatment is to start heart failure guided medical therapies.
- Stage C: Structural heart disease with prior or current symptoms of HF, recommended treatment is guided medical therapies and devices therapy if indicated.
- Stage D: Refractory HF requiring specialized interventions, recommended treatments are Heart transplantation and mechanical circulatory support in selected patients and palliative care.

In addition to left ventricular assist devices, total artificial heart transplants (TAH-t) are used in patients with end-stage heart disease who need a heart transplant, who have failed optimal medical therapy, and for who no other reasonable medical or surgical treatment options are available. TAH-t is typically chosen in patients with biventricular failure rather than left ventricular failure only or who are ineligible for other ventricular devices when a donor organ is not immediately available. Total artificial hearts have been used as a bridge to transplant (temporary) or as a destination therapy (permanent).

Total Artificial Heart, continued

The SynCardia Total Artificial Heart (SynCardia Systems Inc., Tucson, AZ) is a pulsating is a pneumatically driven, biventricular replacement device with 2 mechanical valves in each ventricle and 2 exiting drivelines. large power-generating console, which operates and monitors the device. A smaller portable controller, the Freedom Driver, is also available to help to discharge selected patients.

The total artificial heart continues to be studied extensively, as both a bridge to transplant and a destination therapy in patients with intractable (stage 4) heart failure, failing on medication therapy alone.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers the SynCardia total artificial heart as a bridge to transplant in *limited circumstances.*

Members must meet ALL the following criteria:

1. Intractable biventricular failure unresponsive to guideline-directed medical therapy; **AND**
2. Member is an approved candidate for donor heart transplantation and has been registered as a transplant candidate; **AND**
3. Member is not a candidate for left ventricular assist device (LVAD) implantation; **AND**
4. Member has adequate space in the chest area where the ventricles will be removed. This is generally defined as a body surface area (BSA) > 1.7 m^2 and a distance between the sternum and the 10th anterior vertebral body measured by CT is > or = 10 cm.

Select Health does NOT cover any other total artificial heart device (e.g., AbioCor). The lack of evidence does not support adequate safety or efficacy. This meets the plan's definition of experimental/investigational.

Select Health does NOT cover *any* total artificial heart device as destination therapy. The lack of evidence does not support adequate safety or efficacy. This meets the plan's definition of experimental/investigational.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

The number of studies available assessing total artificial heart transplant (TAH-t) as a bridge to transplant or destination therapy is quite limited. Only 3 studies met the inclusion criteria for review, and none were randomized to include an arm comparing the efficacy and safety of TAH-t to left ventricular assist device (LVAD) implantation. None of the reviews assessed the AbioCor device.

Total Artificial Heart, continued

These studies, as expected, tended to have small study numbers, though, the largest by Platis et al. published in 2009 assessed the outcomes of 715 SynCardia transplants over 16 years. Overall, the studies demonstrated improved mortality as compared to watchful waiting but also demonstrated significant complications. From the studies included in this report, the table below provides a parallel comparison of the various trials and the outcomes as they compare to LVAD used as a bridge to transplant.

Characteristic	Total Artificial Heart Device	Left Ventricular Assist Device
Survival to transplant post-implantation	71.5%–79%	69%–86.9%
1-year survival post donor heart transplant	57%–90%	63%–66.6%
5-year survival post donor heart transplant	34%–81%	
Re-exploration for bleeding	47%	15.6%–33.3%
Respiratory failure	4.7%–44%	
Renal failure requiring temporary dialysis	40%	
Infection	33%–85%	8%–19%
Neurological event	7%–16%	4.8%-8.0%
GI bleeding		15.6%
Device failure	2.3%	0%–3.1%

The most recent update was from Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) report, of 450 implants, 266 were transplanted and 162 died on support. One-year survival depended on center experience; for centers that had implanted more than 10 TAHs, survival was near 70%, vs. 40% for centers with 10 or less. Combining all centers, competing outcomes analysis demonstrated an overall likelihood of transplantation of 53%, mortality of 34%, and 13% alive on a device by 12 months. The most common cause of death was multisystem organ failure (36% of deaths), followed by neurologic injury (18%) and elective withdrawal of support (12%).

Billing/Coding Information

Covered: For the conditions outlined above

CPT CODES

33927 Implantation of a total replacement heart system (artificial heart) with recipient cardiectomy

33928 Removal and replacement of total replacement heart system (artificial heart)

33929 Removal of a total replacement heart system (artificial heart) for heart transplantation (List separately in addition to code for primary procedure)

HCPCS CODES

No specific codes identified

Key References

1. Arabia FA, Cantor RS, Koehl DA, et al. Interagency Registry for mechanically assisted circulatory support report on the total artificial heart. J Heart Lung Transplant 2018; 37:1304-1312.
2. Colucci WS. Evaluation of the patient with heart failure or cardiomyopathy. 17.3. October 8, 2009. UpToDate. Available: http://www.uptodate.com/online/content/topic.do?topicKey=hrt_fail/20066&source=preview&anchor=H3#H3. Date Accessed: February 2, 2010.
3. Copeland JG, Smith RG, Arabia FA, et al. "Cardiac replacement with a total artificial heart as a bridge to transplantation." N Engl J Med 351.9 (2004): 859-67.
4. Copeland JG, Smith RG, Bose RK, Tsau PH, Nolan PE, Slepian MJ. "Risk factor analysis for bridge to transplantation with the CardioWest total artificial heart." Ann Thorac Surg 85.5 (2008): 1639-44.
5. El-Hamamsy I, Jacques F, Perrault LP, et al. "Results following implantation of mechanical circulatory support systems: the Montreal Heart Institute experience." Can J Cardiol 25.2 (2009): 107-10.
6. FDA. AbioCor® Implantable Replacement Heart - H040006. June 30, 2009. Food and Drug Administration. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm077536.htm. Date Accessed: January 30, 2010.
7. FDA. Syncardia Temporary CardioWest Total Artificial Heart (TAH-t) - P030011. June 29, 2009. Food and Drug Administration. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm080816.htm. Date Accessed: January 30, 2010.
8. Hayes Inc. Total Artificial Heart, Temporary or Permanent Biventricular Support Device. Lansdale: Hayes Inc., 2005.

Total Artificial Heart, continued

9. HeartReplacement.com. The AbiCor System. 2009. AbioMed. Available: http://www.heartreplacement.com/abiocore.html. Date Accessed: January 10, 2010.
10. Hutchinson J, Scott DA, Clegg AJ, et al. "Cost-effectiveness of left ventricular-assist devices in end-stage heart failure." Expert Rev Cardiovasc Ther 6.2 (2008): 175-85.
11. John R, Kamdar F, Liao K, Colvin-Adams M, Boyle A, Joyce L. "Improved survival and decreasing incidence of adverse events with the HeartMate II left ventricular assist device as bridge-to-transplant therapy." Ann Thorac Surg 86.4 (2008): 1227-34; discussion 1234-5.
12. Lahpor J, Khaghani A, Hetzer R, et al. "European results with a continuous-flow ventricular assist device for advanced heart-failure patients." Eur J Cardiothorac Surg. (2009).
13. Loforte A, Montalto A, Ranocchi F, et al. "Long-term mechanical support with the HeartMate II LVAS." Transplant Proc 41.4 (2009): 1357-9.
14. MD Consult. Heart Failure. 5/13/2009. A.D.A.M. Inc. Available: http://www.mdconsult.com/das/patient/body/183362523-4/952480291/10041/9436.html. Date Accessed: January 30, 2010.
15. Platis A, Larson DF. "CardioWest temporary total artificial heart." Perfusion 24.5 (2009): 341-6.
16. Roussel JC, Senage T, Baron O, et al. "CardioWest (Jarvik) total artificial heart: a single-center experience with 42 patients." Ann Thorac Surg 87.1 (2009): 124-9; discussion 130.
17. Slaughter MS, Rogers JG, Milano CA, et al. "Advanced heart failure treated with continuous-flow left ventricular assist device." N Engl J Med 361.23 (2009): 2241-51.
18. Slaughter MS, Tsui SS, El-Banayosy A, et al. "Results of a multicenter clinical trial with the Thoratec Implantable Ventricular Assist Device." J Thorac Cardiovasc Surg 133.6 (2007): 1573-80.
19. Struber M, Sander K, Lahpor J, et al. "HeartMate II left ventricular assist device; early European experience." Eur J Cardiothorac Surg 34.2 (2008): 289-94.
20. SynCardia. SynCardia Total Artificial Heart. 2009. Available: http://www.syncardia.com/SynCardia-Total-Artificial-Heart.html. Date Accessed: January 30, 2010
21. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. Circulation. 2013 Oct 15;128(16): e240-327.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

Select Health

MEDICAL POLICY

TRANSCATHETER AORTIC VALVE IMPLANT (TAVI) TRANSCATHETER AORTIC VALVE REPLACEMENT (TAVR)

Policy # 444

Implementation Date:5/26/10

Review Dates: 4/21/11, 12/18/14, 10/20/16, 9/19/18, 12/1/18, 12/18/19, 12/23/20, 12/18/21, 1/14/23, 12/21/23, 12/5/24

Revision Dates: 8/5/13, 11/06/13, 4/3/15, 7/10/15, 12/9/16, 1/20/17, 12/21/17, 9/24/18, 8/23/19, 1/4/22, 11/11/24, 12/16/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

There are 3 primary causes of valvular aortic stenosis (AS): a congenitally abnormal valve with superimposed calcification (unicuspid or bicuspid), calcific disease of a trileaflet valve, and rheumatic valve disease. In North America and Europe, aortic valve disease is primarily due to calcific disease of a native trileaflet or a congenital bicuspid valve. Worldwide, rheumatic valve disease is most common; mitral valve involvement invariably accompanies rheumatic aortic valve disease.

Many patients do not develop symptoms until severe valve obstruction (valve area < 1.0 cm^2) is present, while some patients become symptomatic when the stenosis is less severe (1.5−2.0 cm^2), particularly, if there is coexisting aortic regurgitation. Replacement of the aortic valve is the only effective treatment for severe AS. Standard aortic valve replacement surgery involves the patient undergoing a sternotomy and being placed on cardio bypass. Not all patients can tolerate such an extensive procedure.

Less invasive transcatheter techniques for aortic valve replacements have been developed. Currently, FDA approved transcatheter valves include the balloon expandable Sapien valve and self-expandable CoreValve systems. Both Sapien and CoreValve consist of metal frames which support tissue valves. In June 2015, the first repositionable transcatheter valve, the CoreValve Evolut R received FDA approval. This is the first valve which can be repositioned after initial deployment, so as to reduce valve leakage or other issues.

Approaches to transcatheter aortic valve replacement include retrograde delivery with transfemoral, transaortic, and subclavian access, as well as antegrade delivery with the transapical approach.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers FDA approved transcatheter aortic valve implants/replacements when EITHER of the following criteria are met (A or B).

A. TAVR will be approved If recommended by Intermountain Health Cardiovascular Clinical Program,

OR

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

B. For all other clinicians, TAVR will be approved when **ALL** the following criteria are met:

Criteria for Coverage (Must meet ALL):

1. Device has been approved by the FDA [PMA or 510(k)]

   AND

2. Patient has one of the following conditions (a–c):

   a. Severe native calcific aortic stenosis demonstrated by at least one of the following (i–iii):

      i) An aortic valve area ≤ 1.0cm^2
      ii) A mean aortic valve gradient greater than 40mmHg
      iii) A jet velocity greater than 4.0m/sec

   b. Failure (stenosed, insufficient, or combined) of a surgical bioprosthetic aortic valve

   c. Severe aortic regurgitation with justification from a cardiothoracic surgeon

   AND

3. Patient has been evaluated face-to-face for open heart surgery by at least one cardiologist and one cardiothoracic surgeon, with full documentation, and determined to be a candidate for TAVR.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For this policy, specifically, there are no CMS criteria available; therefore, the Select Health Commercial policy or InterQual criteria apply. Select Health applies these requirements after careful review of the evidence that supports the clinical benefits outweigh the clinical risks. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

TAVR for Aortic Stenosis: A May 2010 Medical Technology Assessment found the published literature available on this topic was limited. Only 1 technology assessment and 4 primary studies were identified. However, it is important to note the 1 technology assessment was published in June 2006 and was performed by a highly respected organization—the National Institute for Health and Clinical Excellence (NICE) out of the U.K. This technology assessment aimed to provide conclusions concerning current evidence of transcatheter aortic valve implantation. Aortic regurgitation and transvalvular gradient both appeared to decrease significantly in postoperative follow-up. Most of the literature focused on the implementation of these 2 devices. These are 2 major indicators/complications associated with diseased aortic valves.

A Medical Technology Assessment performed in June 2012 identified 23 new papers published since the last review in 2010, reflecting data at least 2 years post-procedure. In February 2012, Bleiziffer et al.

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

reported 2-year results in 227 patients who received TAVI/TAVR. Clinical and echocardiographic investigations were performed at 6 months, 1 year, and 2 years. Survival was 88.5% at 30 days, 75.9% at 6 months, 74.5% at 1 year, and 64.4% at 2 years. Patients improved significantly in New York Heart Association class after 6 months (from 3.2 +/- 0.5 to 1.7 +/- 0.7, p < .001) and up to 2 years (1.9 +/- 0.7). Cumulative incidences of myocardial infarction, stroke, and life-threatening or major bleeding were 2.7%, 6.2%, and 16.2% at 2 years, respectively. Moderate or severe prosthetic regurgitation was present in 8% of patients at 2 years. In 6% of patients, the paravalvular or valvular regurgitation grade increased significantly over time. They concluded transcatheter aortic valve implantation may be considered the treatment of choice for aortic valve stenosis in elderly patients with an increased risk for surgery with a heart-lung machine.

A prospective multicenter observational study published in 2011 by D'Onofrio et al. assessed early and 2-year outcomes after TAVI/TAVR in 179 patients. Patients underwent clinical and echocardiographic follow-up visits at hospital discharge, 3 and 6 months after TAVI/TAVR, and every 6 months thereafter. Seventeen severe intraoperative complications occurred in 13 (7.3%) patients. Thirty-day mortality was 3.9% (7 patients). Mean follow-up was 9.2 +/- 6.5 months. Late mortality occurred in 9 patients. Two-year survival was 88% +/- 3%. An intraoperative severe complication was identified as the only significant independent predictor of 1-year mortality. A significant benefit was found when comparing 2-year survival of the second versus the first 50% patients at each center (93% +/- 2% vs. 84% +/- 3 %; p = 0.046). A significant reduction of both mean and peak gradients from the preoperative to the postoperative period, which remained stable during follow-up, was found.

In May 2012, the Centers for Medicare and Medicaid (CMS) issued a national coverage determination (NCD) outlining criteria under which it would provide coverage of TAVR/TAVI. Current evidence demonstrates improved mortality for patients who are ineligible for standard AVR, though, some increased morbidity risks exist especially for early stroke problems. Nonetheless, several studies identify this therapy to meet current standards for cost-effectiveness as defined by QALY's.

Transcatheter aortic valve replacement (TAVR) has emerged as a therapeutic alternative for patients with severe aortic stenosis whose surgical risk is very high or prohibitively high, in part due to findings of the PARTNER trials (inoperable patients: *NEJM JW Cardiology May 2012* and *N Engl J Med* 2012; 366:1696; high-risk patients: *NEJM JW Cardiology May 2012* and *N Engl J Med* 2012; 366:1686). Two manufacturer-sponsored analyses from PARTNER provide insights into the long-term survival patterns of such patients.

Kapadia and colleagues focused on 358 patients with severe aortic stenosis (mean age, 83; mean Society of Thoracic Surgeons estimated risk of 30-day mortality after operative AVR [STS], 11.7%; women, 54%) who were deemed inoperable because of mortality risk or anatomical factors. Individuals were randomized to TAVR or nonoperative standard therapy. At 5 years, mortality was 71.8% in the TAVR group and 93.6% in the standard therapy group. There were six standard-therapy survivors with follow-up data; five received AVR outside the study. Of 49 survivors in the TAVR group, 86% had modest symptom burden (New York Heart Association class I or II symptoms). Prosthetic valve deterioration was not evident in the TAVR group.

Mack and colleagues followed 699 patients with severe aortic stenosis (mean age, 84; mean STS score, 11.7%; women, 43%) who were considered at high but not prohibitive risk for surgery. Individuals were randomized to TAVR or surgical AVR (SAVR). At 5 years, the two groups had statistically similar mortality—67.8% with TAVR and 62.4% with SAVR. No postprocedural events requiring repeat valve replacement were reported. Moderate-to-severe aortic regurgitation 30 days after the procedure occurred in 14% of the TAVR group and 1% of the SAVR group ($P < 0.0001$) and was identified as a risk factor for death.

Aortic Stenosis with Aortic Insufficiency: A literature review completed in November 2016 reviewed the evidence as it relates to TAVR use in aortic insufficiency. This review identified one systematic review and 3 primary studies were identified which met inclusion criteria. In all, 159 patients were studied, of which, 92 (57.9%) received TAVR for aortic insufficiency. The systematic review by Phan et al., though, specific to AI, investigated the use of TAVR in patients with a left ventricular assist device. Though the report showed promise for the procedure, it does not generally address the question of the procedure's clinical utility in most patients with traditional AI symptoms.

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

The 3 primary studies were all published since 2013. They consist of multi-center, prospective, or retrospective patient populations. No long-term data has been published to date on the use of this intervention for the treatment of AI. Only 1 of the 3 (33%, Wilder et al.) compared outcomes to other surgical methods. Survival outcomes from studies by Roy et al. and Testa et al. varied widely even with use of the same device (CoreValve) likely due to differing patient populations and inclusion criteria. Mortality from AI/AR in symptomatic patients is > 10% per year. The assessed studies show greater than double the mortality rate in patients treated with TAVR for AI than previously published outcomes from patients not undergoing surgical management for the treatment of AI. One obvious limitation of this conclusion is the lack of randomized, controlled published data regarding TAVR in patients with primary symptomatic AI.

Though the evidence regarding TAVR in aortic insufficiency is weak, it demonstrates net benefit in high-risk patients who are otherwise not surgical candidates. In addition, the FDA has approved the Edwards Sapien XT for use in aortic insufficiency in patients with a bioprosthetic valve (valve-in-valve procedure).

As experience with TAVR continues to grow, potential candidates for TAVR continue to be explored. Whereas initial candidates were either ineligible for surgery or have serious surgery risk for post-operative morbidity or mortality. A body of literature has been published related to performing TAVR in patients with intermediate surgical risk (STS score $\geq$ 3 and $\leq$ 10). A review of the published literature recently completed identified four systematic reviews and 14 primary studies related to patients with intermediate surgical risk undergoing TAVR. All 4 systematic reviews were published in 2016 and the primary studies have all been published since 2012. These studies represent assessment of > 17,000 intermediate risk patients, of which, > 8,800 received TAVR.

The four systematic reviews by Arora et al., Khan et al., Siemieniuk et al., and Zhou et al., reached similar conclusions that all-cause mortality rates did not differ to a statistically significant degree between SAVR and TAVR-treated intermediate-risk patients. From the primary literature studies by Abdul-Jawad Altisent et al., D'Errigo et al., Fanning et al., Kodali et al., Leon et al., and Thourani et al., it was also suggested the rate of stroke was lower in TAVR than in SAVR.

One concern noted from the studies was related to the need for permanent cardiac pacemaker placement. Nine of the primary studies and the four systematic reviews reported on pacemaker implantation. All the systematic reviews reported an increased incidence of pacemaker implantation in TAVR vs. SAVR patients. This rate varied in the studies, from 2 to 15 times the incidence of pacemaker placement TAVR compared to SAVR.

Conclusions drawn from the recently published data on TAVR for intermediate risk patients from 2012 has illustrated mortality and pacemaker implantation rates in nearly 8,000 patients. Most of the literature compared TAVR to SAVR and illustrated comparable mortality rates at follow-up periods between 1 and 24 months. However, substantially more pacemakers are implanted in TAVR patients than in those receiving standard surgical treatment.

Billing/Coding Information
Covered: For the circumstances outlined above

CPT CODES

33361	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; percutaneous femoral artery approach
33362	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; open femoral artery approach
33363	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; open axillary artery approach
33364	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; open iliac artery approach
33365	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; transaortic approach (eg, median sternotomy, mediastinotomy)

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

33366	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; transapical exposure (eg, left thoracotomy
33367	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; cardiopulmonary bypass support with percutaneous peripheral arterial and venous cannulation (eg, femoral vessels) (List separately in addition to code for primary procedure)
33368	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; cardiopulmonary bypass support with open peripheral arterial and venous cannulation (eg, femoral, iliac, axillary vessels) (List separately in addition to code for primary procedure)
33369	Transcatheter aortic valve replacement (TAVR/TAVI) with prosthetic valve; cardiopulmonary bypass support with central arterial and venous cannulation (eg, aorta, right atrium, pulmonary artery) (List separately in addition to code for primary procedure)
33405	Replacement, aortic valve, with cardiopulmonary bypass; with prosthetic valve other than homograft or stentless valve
93355	Echocardiography, transesophageal (TEE) for guidance of a transcatheter intracardiac or great vessel(s) structural intervention(s) (eg,TAVR, transcatheter pulmonary valve replacement, mitral valve repair, paravalvular regurgitation repair, left atrial appendage occlusion/closure, ventricular septal defect closure) (peri-and intra-procedural), real-time image acquisition and documentation, guidance with quantitative measurements, probe manipulation, interpretation, and report, including diagnostic transesophageal echocardiography and, when performed, administration of ultrasound contrast, Doppler, color flow, and 3D

Not Covered: Considered investigational

93591	Percutaneous transcatheter closure of paravalvular leak; initial occlusion device, aortic valve

HCPCS CODES

No specific codes identified

Key References

1. Al-Attar N, Ghodbane W, Himbert D, et al. (2009). Unexpected complications of transapical aortic valve implantation. Ann Thorac Surg 88.1: 90-4.
2. Amat-Santos, IJ, Rodes-Cabau, J, Urena, M, et al. (2012). Incidence, predictive factors, and prognostic value of new-onset atrial fibrillation following transcatheter aortic valve implantation. J Am Coll Cardiol 59.2: 178-88.
3. Barbanti, M., et al. (2012). "Transcatheter aortic valve implantation in patients with mitral prosthesis." J Am Coll Cardiol 60(18): 1841-1842.
4. Beller, C. J., et al. (2011). "Transcatheter aortic valve implantation after previous mechanical mitral valve replacement: expanding indications?" Heart Surg Forum 14(3): E166-170.
5. Bleiziffer, S, Mazzitelli, D, Opitz, A, et al. (2012). Beyond the short-term: clinical outcome and valve performance 2 years after transcatheter aortic valve implantation in 227 patients. J Thorac Cardiovasc Surg 143.2: 310-7.
6. Bunc, M. (2012). "Successful Tavi in Patients with Previous Mitral Valve Replacement " Scietific Research 3((7)).
7. CardiacHealth.org. (2012) Cardiac Risk STS Calculator. Cardiachealth.org. Available: http://www.cardiachealth.org/app/sts.php. Date Accessed: May 29, 2012.
8. Conradi, L, Seiffert, M, Treede, H, et al. (2012). Transcatheter aortic valve implantation versus surgical aortic valve replacement: a propensity score analysis in patients at high surgical risk. J Thorac Cardiovasc Surg 143.1: 64-71.
9. D'Onofrio, A, Fusari, M, Abbiate, N, et al. (2011). Transapical aortic valve implantation in high-risk patients with severe aortic valve stenosis. Ann Thorac Surg 92.5: 1671-7.
10. Edwards Life Sciences LLC. (2010) Edwards SAPIEN Transcatheter Heart Valve. Available: Edwards SAPIEN Transcatheter Heart Valve. Date Accessed: March 1, 2010.
11. ElBardissi, AW, Shekar, P, Couper, GS, et al. (2011). Minimally invasive aortic valve replacement in octogenarian, high-risk, transcatheter aortic valve implantation candidates. J Thorac Cardiovasc Surg 141.2: 328-35.
12. Erkapic, D, S, DER, Kelava, A, et al. (2012). Risk for permanent pacemaker after transcatheter aortic valve implantation: a comprehensive analysis of the literature. J Cardiovasc Electrophysiol 23.4: 391-7.
13. Ewe, SH, Delgado, V, Ng, AC, et al. (2011). Outcomes after transcatheter aortic valve implantation: transfemoral versus transapical approach. Ann Thorac Surg 92.4: 1244-51.
14. Fairbairn, TA, Meads, DM, Mather, AN, et al. (2012). Serial change in health-related quality of life over 1 year after transcatheter aortic valve implantation: predictors of health outcomes. J Am Coll Cardiol 59.19: 1672-80.
15. Food and Drug Administration (2013). "Edwards SAPIEN Transcatheter Heart Valve (THV)-P100041." Retrieved 11/07, 2013.

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

16. Food and Drug Administration. *Edwards SAPIEN 3 Transcatheter Heart Valve*. 2016 August 18, 2016 [cited 2016 November 23]; Available from: http://www.accessdata.fda.gov/cdrh_docs/pdf14/P140031S010a.pdf.
17. Food and Drug Administration. Edwards SAPIEN XT Transcatheter Heart Valve. 2015 October 9, 2015 [cited 2016 November 23]; Available from: http://www.accessdata.fda.gov/cdrh_docs/pdf13/P130009S034A.pdf.
18. Fraccaro C, Napodano M, Tarantini G, et al. (2009). Expanding the eligibility for transcatheter aortic valve implantation the trans-subclavian retrograde approach using: the III generation CoreValve revalving system. JACC Cardiovasc Interv 2.9: 828-33.
19. Gaasch, WH. (2009) Indications for valve replacement in aortic stenosis in adults. August 28, 2009. Up to Date. Available: http://www.utdol.com/online/content/topic.do?topicKey=valve_hd/3020&selectedTitle=1%7E85&source=search_result#. Date Accessed: March 2, 2010, 2010.
20. Gaasch, WH. (2009) Methods of valve replacement in aortic stenosis in adults. February 2, 2009. Up To Date. Available: http://www.utdol.com/online/content/topic.do?topicKey=valve_hd/21421&selectedTitle=1%7E150&source=search_result. Date Accessed: February 24, 2010, 2010.
21. Gaasch, W.H. *Clinical manifestations and diagnosis of chronic aortic regurgitation in adults*. 2016 April 10 [cited 2016 November 11]; Available from: https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-chronic-aortic-regurgitation-in-adults?source=search_result&search=aortic%20insufficiency&selectedTitle=1~150#H5.
22. Gaasch, W.H. *Natural history and management of chronic aortic regurgitation in adults*. 2016 July 29, 2015 [cited 2016 November 11]; Available from: https://www.uptodate.com/contents/natural-history-and-management-of-chronic-aortic-regurgitation-in-adults?source=search_result&search=aortic%20regurgitation%20treatment&selectedTitle=1~150#H1411845277.
23. Gada, H, Kapadia, SR, Tuzcu, EM, et al. (2012). Markov model for selection of aortic valve replacement versus transcatheter aortic valve implantation (without replacement) in high-risk patients. Am J Cardiol 109.9: 1326-33.
24. Garcia, E., et al. (2011). "[Transcatheter aortic valve implantation in patients with a mechanical mitral valve]." Rev Esp Cardiol 64(11): 1052-1055.
25. Généreux, P., Schwartz, A., Oldemeyer, J.B., Pibarot, P., Cohen, D.J., Blanke, P. … Leon, M.B.; EARLY TAVR Trial Investigators. Transcatheter Aortic-Valve Replacement for Asymptomatic Severe Aortic Stenosis. *N Engl J Med*. 2024 Oct 28. doi: 10.1056/NEJMoa2405880. Epub ahead of print. PMID: 39466903.
26. Gilard, M, Eltchaninoff, H, Iung, B, et al. (2012). Registry of transcatheter aortic-valve implantation in high-risk patients. N Engl J Med 366.18: 1705-15.
27. Grube E, Laborde JC, Gerckens U, et al. (2006). Percutaneous implantation of the CoreValve self-expanding valve prosthesis in high-risk patients with aortic valve disease: the Siegburg first-in-man study. Circulation 114.15: 1616-24.
28. Grube E, Schuler G, Buellesfeld L, et al. (2007). Percutaneous aortic valve replacement for severe aortic stenosis in high-risk patients using the second- and current third-generation self-expanding CoreValve prosthesis: device success and 30-day clinical outcome. J Am Coll Cardiol 50.1: 69-76.
29. Hammermeister K, Sethi GK, Henderson WG, et al. (2000). Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the Veterans Affairs randomized trial. J Am Coll Cardiol 36.4: 1152-8.
30. Hayashida, K, Morice, MC, Chevalier, B, et al. (2012). Sex-related differences in clinical presentation and outcome of transcatheter aortic valve implantation for severe aortic stenosis. J Am Coll Cardiol 59.6: 566-71.
31. Holmes, DR, Jr., Mack, MJ, Kaul, S, et al. (2012). 2012 ACCF/AATS/SCAI/STS expert consensus document on transcatheter aortic valve replacement: developed in collaboration with the American Heart Association, American Society of Echocardiography, European Association for Cardio-Thoracic Surgery, Heart Failure Society of America, Mended Hearts, Society of Cardiovascular Anesthesiologists, Society of Cardiovascular Computed Tomography, and Society for Cardiovascular Magnetic Resonance. Ann Thorac Surg 93.4: 1340-95.
32. Kalavrouziotis, D, Rodes-Cabau, J, Bagur, R, et al. (2011). Transcatheter aortic valve implantation in patients with severe aortic stenosis and small aortic annulus. J Am Coll Cardiol 58.10: 1016-24.
33. Kapadia SR et al. 5-year outcomes of transcatheter aortic valve replacement compared with standard treatment for patients with inoperable aortic stenosis (PARTNER 1): A randomised controlled trial. Lancet 2015 Mar 15; [e-pub]. (http://dx.doi.org/10.1016/S0140-6736(15)60290-2)
34. Kappetein, A.P. PARTNERs in the future of surgical aortic valve replacement. Lancet 2015 Mar 15; [e-pub]. (http://dx.doi.org/10.1016/S0140-6736(15)60568-2)
35. Latsios, G, Gerckens, U, Grube, E. (2009). Transaortic transcatheter aortic valve implantation: A novel approach for the truly "no-access option" patients. Catheter Cardiovasc Interv.
36. Leon, MB, Kodali, S, Williams, M, et al. (2006). Transcatheter aortic valve replacement in patients with critical aortic stenosis: rationale, device descriptions, early clinical experiences, and perspectives. Semin Thorac Cardiovasc Surg 18.2: 165-74.
37. Lichtenstein SV, Cheung A, Ye J, et al. (2006). Transapical transcatheter aortic valve implantation in humans: initial clinical experience. Circulation 114.6: 591-6.
38. Mack MJ et al. 5-year outcomes of transcatheter aortic valve replacement or surgical aortic valve replacement for high surgical risk patients with aortic stenosis (PARTNER 1): A randomised controlled trial. Lancet 2015 Mar 15; [e-pub]. (http://dx.doi.org/10.1016/S0140-6736(15)60308-7
39. Makkar, RR, Fontana, GP, Jilaihawi, H, et al. (2012). Transcatheter aortic-valve replacement for inoperable severe aortic stenosis. N Engl J Med 366.18: 1696-704.
40. Medtronic. (2009) Medtronic CoreValve System 60. Available: http://www.medtronic.com/corevalve/other.html. Date Accessed: April 28, 2010.
41. Medtronic (2015). "Medtronic Announces FDA Approval for New TAVR System, Introduces First and Only Recaturable Heart Valve in U.S.". Retrieved 06/23/2015, 2015, from http://newsroom.medtronic.com/phoenix.zhtml?c=251324&p=irol-newsArticle&ID=2061818.
42. Moat, NE, Ludman, P, de Belder, MA, et al. (2011). Long-term outcomes after transcatheter aortic valve implantation in high-risk patients with severe aortic stenosis: the U.K. TAVI (United Kingdom Transcatheter Aortic Valve Implantation) Registry. J Am Coll Cardiol 58.20: 2130-8.
43. Motloch, LJ, Reda, S, Rottlaender, D, et al. (2012). Postprocedural atrial fibrillation after transcatheter aortic valve implantation versus surgical aortic valve replacement. Ann Thorac Surg 93.1: 124-31.

Select Health

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

44. NICE. (2008) http://www.nice.org.uk/nicemedia/live/11914/41020/41020.pdf. London: National Institute for Health and Clinical Excellence.
45. Nishimura, R.A., et al., *2014 AHA/ACC Guideline for the Management of Patients With Valvular Heart Disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines.* Circulation, 2014. 129(23): p. 2440-92.
46. Otto, CM. (2010). Pathophysiology and clinical features of valvular aortic stenosis in adults. 18.1. Available: http://www.uptodate.com/online/content/topic.do?topicKey=valve_hd/7401&selectedTitle=2%7E150&source=search_result. April 4 Access Date, Access 2010.
47. Otto, CM. (2012) Estimating the mortality risk of valvular surgery. April 11, 2012. Up to Date. Available: http://www.uptodate.com/contents/estimating-the-mortality-risk-of-valvular-surgery?source=search_result&search=sts+score&selectedTitle=1~150. Date Accessed: June 4, 2012.
48. Phan, K., et al., *Percutaneous transcatheter interventions for aortic insufficiency in continuous-flow left ventricular assist device patients: A systematic review and meta-analysis.* ASAIO J, 2016.
49. Piazza N, Grube E, Gerckens U, et al. (2008). Procedural and 30-day outcomes following transcatheter aortic valve implantation using the third generation (18 Fr) corevalve revalving system: results from the multicentre, expanded evaluation registry 1-year following CE mark approval. EuroIntervention 4.2: 242-9.
50. Piazza N, Grube E, Gerckens U, et al. (2010). A clinical protocol for analysis of the structural integrity of the Medtronic CoreValve System(R) frame and its application in patients with 1-year minimum follow-up. EuroIntervention 5.6: 680-6.
51. Reynolds, MR, Magnuson, EA, Lei, Y, et al. (2011). Health-related quality of life after transcatheter aortic valve replacement in inoperable patients with severe aortic stenosis. Circulation 124.18: 1964-72.
52. Roy, D.A., et al., Transcatheter aortic valve implantation for pure severe native aortic valve regurgitation. J Am Coll Cardiol, 2013. 61(15): p. 1577-84.
53. Smith, CR, Leon, MB, Mack, MJ, et al. (2011). Transcatheter versus surgical aortic-valve replacement in high-risk patients. N Engl J Med 364.23: 2187-98.
54. Society for Cardiothoracic Surgery. (2008) A position statement of the British Cardiovascular Intervention Society (BCIS) and the Society for Cardiothoracic Surgery (SCTS). December 18, 2008. Society for Cardiothoracic Surgery in Great Britain and England. 58. Available: http://www.scts.org/documents/PDF/BCISTAVIDec2008.pdf. Date Accessed: March 3, 2010
55. Stortecky, S, Brinks, H, Wenaweser, P, et al. (2011). Transcatheter aortic valve implantation or surgical aortic valve replacement as redo procedure after prior coronary artery bypass grafting. Ann Thorac Surg 92.4:1324-30; discussion 1230-1.
56. Swinkels, BM, Plokker, HW. (2010). Evaluating operative mortality of cardiac surgery: first define operative mortality. Neth Heart J 18.7-8: 344-5.
57. Tchetche D, Dumonteil N, Sauguet A, et al. (2010). Thirty-day outcome and vascular complications after transarterial aortic valve implantation using both Edwards Sapien and Medtronic CoreValve(R) bioprostheses in a mixed population. EuroIntervention 5.6: 659-65.
58. Testa, L., et al., *CoreValve implantation for severe aortic regurgitation: a multicentre registry.* EuroIntervention, 2014. 10(6): p. 739-45.
59. The Society of Thoracic Surgeons. (2012) Explanation of Quality Rating, Composite Score, and Star Ratings. The Society of Thoracic Surgeons. Available: http://www.sts.org/quality-research-patient-safety/sts-public-reporting-online/explanation-quality-rating-composite-sco. Date Accessed: June 1, 2012.
60. TheHeart.org. (2012). PARTNER CAP data show transapical outcomes improve with practice. January 31, 2012. Interventional and Surgery. Available: http://www.theheart.org/article/1348117.do. Date Accessed: June 5, 2012.
61. Unbehaun, A, Pasic, M, Dreysse, S, et al. (2012). Transapical aortic valve implantation: incidence and predictors of paravalvular leakage and transvalvular regurgitation in a series of 358 patients. J Am Coll Cardiol 59.3: 211-21.
62. UnitedHealthcare. *Transcatheter Heart Valve Procedures*. 2016 September 1, 2016 [cited 2016 November 1, 2016]; Available from: https://www.unitedhealthcareonline.com/ccmcontent/ProviderII/UHC/en_US/Assets/ProviderStaticFiles/ProviderStaticFilesPdf/Tools%20and%20Resources/Policies%20and%20Protocols/Medical%20Policies/Medical%20Policies/TranscatheterHeartValve_Procedures.pdf
63. Ussia, GP, Scarabelli, M, Mule, M, et al. (2011). Dual antiplatelet therapy versus aspirin alone in patients undergoing transcatheter aortic valve implantation. Am J Cardiol 108.12: 1772-6.
64. Wang, S.S. *Aortic Regurgitation*. 2016 February 12, 2014 [cited 2016 November 11]; Available from: http://emedicine.medscape.com/article/150490-overview#a7.
65. Watt, M, Mealing, S, Eaton, J, et al. (2012). Cost-effectiveness of transcatheter aortic valve replacement in patients ineligible for conventional aortic valve replacement. Heart 98.5: 370-6.
66. Webb, JG, Chandavimol, M, Thompson, CR, et al. (2006). Percutaneous aortic valve implantation retrograde from the femoral artery. Circulation 113.6: 842-50.
67. Wilder, T.J., et al., *Aortic valve repair for insufficiency in older children offers unpredictable durability that may not be advantageous over a primary Ross operationdagger.* Eur J Cardiothorac Surg, 2016. 49(3): pp. 883-92.
68. Ye, J, Cheung, A, Lichtenstein, SV, et al. (2007). Six-month outcome of transapical transcatheter aortic valve implantation in the initial seven patients. Eur J Cardiothorac Surg 31.1: 16-21.
69. Zierer, A, Wimmer-Greinecker, G, Martens, S, et al. (2008). The transapical approach for aortic valve implantation. J Thorac Cardiovasc Surg 136.4: 948-53.

Intermediate Risk References

70. Abdul-Jawad Altisent, O., et al., Neurological damage after transcatheter aortic valve implantation compared with surgical aortic valve replacement in intermediate risk patients. Clin Res Cardiol, 2016. 105(6): p. 508-17.
71. Arora, S., et al., Transcatheter versus surgical aortic valve replacement in intermediate risk patients: a meta-analysis. Cardiovasc Diagn Ther, 2016. 6(3): p. 241-9.
72. Barbash, I.M., et al., Outcomes of Patients at Estimated Low, Intermediate, and High Risk Undergoing Transcatheter Aortic Valve Implantation for Aortic Stenosis. Am J Cardiol, 2015. 116(12): p. 1916-22.
73. Castrodeza, J., et al., Propensity score matched comparison of transcatheter aortic valve implantation versus conventional surgery in intermediate and low risk aortic stenosis patients: A hint of real-world. Cardiol J, 2016.

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

74. D'Errigo, P., et al., Transcatheter aortic valve implantation versus surgical aortic valve replacement for severe aortic stenosis: results from an intermediate risk propensity-matched population of the Italian OBSERVANT study. Int J Cardiol, 2013. 167(5): p. 1945-52.
75. Fanning, J.P., et al., Neurological Injury in Intermediate-Risk Transcatheter Aortic Valve Implantation. J Am Heart Assoc, 2016. 5(11).
76. Food and Drug Administration. Edwards SAPIEN 3 Transcatheter Heart Valve. 2016 August 18, 2016 [cited 2016 November 23]; Available from: http://www.accessdata.fda.gov/cdrh_docs/pdf14/P140031S010a.pdf.
77. Food and Drug Administration. Edwards SAPIEN XT Transcatheter Heart Valve. 2015 October 9, 2015 [cited 2016 November 23]; Available from: http://www.accessdata.fda.gov/cdrh_docs/pdf13/P130009S034A.pdf.
78. Fraccaro, C., et al., Early and Midterm Outcome of Propensity-Matched Intermediate-Risk Patients Aged >/=80 Years With Aortic Stenosis Undergoing Surgical or Transcatheter Aortic Valve Replacement (from the Italian Multicenter OBSERVANT Study). Am J Cardiol, 2016. 117(9): p. 1494-501.
79. Gaasch, W.H. Clinical manifestations and diagnosis of chronic aortic regurgitation in adults. 2016 April 10 [cited 2016 November 11]; Available from: https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-chronic-aortic-regurgitation-in-adults?source=search_result&search=aortic%20insufficiency&selectedTitle=1~150#H5.
80. Gaasch, W.H. Indications for valve replacement in aortic stenosis in adults. 2009 August 28, 2009 [cited 2010 March 2, 2010]; Available from: http://www.utdol.com/online/content/topic.do?topicKey=valve_hd/3020&selectedTitle=1%7E85&source=search_result#.
81. Gaasch, W.H. Methods of valve replacement in aortic stenosis in adults. [Methods of valve replacement in aortic stenosis in adults] 2009 February 2, 2009 [cited 2010 February 24, 2010]; Available from: http://www.utdol.com/online/content/topic.do?topicKey=valve_hd/21421&selectedTitle=1%7E150&source=search_result.
82. Gaasch, W.H. Natural history and management of chronic aortic regurgitation in adults. 2016 July 29, 2015 [cited 2016 November 11]; Available from: https://www.uptodate.com/contents/natural-history-and-management-of-chronic-aortic-regurgitation-in-adults?source=search_result&search=aortic%20regurgitation%20treatment&selectedTitle=1~150#H1411845277.
83. Groh, M.A., et al., Is Surgical Intervention the Optimal Therapy for the Treatment of Aortic Valve Stenosis for Patients With Intermediate Society of Thoracic Surgeons Risk Score? Ann Thorac Surg, 2016.
84. Hammermeister, K., et al., Outcomes 15 years after valve replacement with a mechanical versus a bioprosthetic valve: final report of the Veterans Affairs randomized trial. J Am Coll Cardiol, 2000. 36(4): p. 1152-8.
85. Khan, A.R., et al., Efficacy and safety of transcatheter aortic valve replacement in intermediate surgical risk patients: A systematic review and meta-analysis. Catheter Cardiovasc Interv, 2016. 88(6): p. 934-944.
86. Kodali, S., et al., Early clinical and echocardiographic outcomes after SAPIEN 3 transcatheter aortic valve replacement in inoperable, high-risk and intermediate-risk patients with aortic stenosis. Eur Heart J, 2016. 37(28): p. 2252-62.
87. Latib, A., et al., Transcatheter vs surgical aortic valve replacement in intermediate-surgical-risk patients with aortic stenosis: a propensity score-matched case-control study. Am Heart J, 2012. 164(6): p. 910-7.
88. Leon, M.B., et al., Transcatheter aortic valve replacement in patients with critical aortic stenosis: rationale, device descriptions, early clinical experiences, and perspectives. Semin Thorac Cardiovasc Surg, 2006. 18(2): p. 165-74.
89. Leon, M.B., et al., Transcatheter or Surgical Aortic-Valve Replacement in Intermediate-Risk Patients. N Engl J Med, 2016. 374(17): p. 1609-20.
90. Muneretto, C., et al., A comparison of conventional surgery, transcatheter aortic valve replacement, and sutureless valves in "real-world" patients with aortic stenosis and intermediate- to high-risk profile. J Thorac Cardiovasc Surg, 2015. 150(6): p. 1570-7; discussion 1577-9.
91. National Guideline Clearinghouse, 2014 AHA/ACC guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014.
92. Osnabrugge, R.L., et al., Costs of transcatheter versus surgical aortic valve replacement in intermediate-risk patients. Ann Thorac Surg, 2012. 94(6): p. 1954-60.
93. Piazza, N., et al., A 3-center comparison of 1-year mortality outcomes between transcatheter aortic valve implantation and surgical aortic valve replacement on the basis of propensity score matching among intermediate-risk surgical patients. JACC Cardiovasc Interv, 2013. 6(5): p. 443-51.
94. Siemieniuk, R.A., et al., Transcatheter versus surgical aortic valve replacement in patients with severe aortic stenosis at low and intermediate risk: systematic review and meta-analysis. BMJ, 2016. 354: p. i5130.
95. Thourani, V.H., et al., Transcatheter aortic valve replacement versus surgical valve replacement in intermediate-risk patients: a propensity score analysis. Lancet, 2016. 387(10034): p. 2218-25.
96. UnitedHealthcare. Transcatheter Heart Valve Procedures. 2016 September 1, 2016 [cited 2016 November 1, 2016]; Available from: https://www.unitedhealthcareonline.com/ccmcontent/ProviderII/UHC/en-US/Assets/ProviderStaticFiles/ProviderStaticFilesPdf/Tools%20and%20Resources/Policies%20and%20Protocols/Medical%20Policies/Medical%20Policies/Transcatheter_Heart_Valve_Procedures.pdf.
97. Van Mieghem, N. The Art of Risk Stratification in TAVI. 2013 [cited 2016 December 13]; Available from: http://www.medscape.com/viewarticle/807418_3.
98. Wenaweser, P., et al., Clinical outcomes of patients with estimated low or intermediate surgical risk undergoing transcatheter aortic valve implantation. Eur Heart J, 2013. 34(25): p. 1894-905.
99. Van Mieghem, N.M. and P.W. Serruys, The art of risk stratification in TAVI. Eur Heart J, 2013. 34(25): p. 1859-61.
100. Wang, S.S. Aortic Regurgitation. 2016 February 12, 2014 [cited 2016 November 11]; Available from: http://emedicine.medscape.com/article/150490-overview#a7.
101. Webb, J.G., et al., Percutaneous aortic valve implantation retrograde from the femoral artery. Circulation, 2006. 113(6): p. 842-50.
102. Yoon SH, Schmidt T, Bleiziffer S, et al. Transcatheter Aortic Valve Replacement in Pure Native Aortic Valve Regurgitation. J Am Coll Cardiol. 2017 Dec 5;70(22):2752-2763. doi: 10.1016/j.jacc.2017.10.006
103. Zhou, Y., et al., Transcatheter versus surgical aortic valve replacement in low to intermediate risk patients: A meta-analysis of randomized and observational studies. Int J Cardiol, 2016. 228: p. 723-728.
104. Zierer, A., et al., The transapical approach for aortic valve implantation. J Thorac Cardiovasc Surg, 2008. 136(4): p. 948-53.

Transcatheter Aortic Valve Implant (TAVI), Transcatheter Aortic Valve Replacement (TAVR), continued

Revision History

Revision Date	Summary of Changes
11/11/24	For Commercial Plan Policy, removed criterion #B-5: "Patient does not have a life expectancy < 12 months due to non-cardiac co-morbid conditions" as a requirement.
12/16/24	For Commercial Plan Policy, removed previous criterion #B-3 as a requirement: "Patient has documented New York Heart Association (NYHA) functional class II or greater."

Select Health

Select Health

MEDICAL POLICY

TRANSCATHETER MITRAL VALVE IMPLANTATION/REPLACEMENT

Policy # 682

Implementation Date: 7/1/24
Review Dates:
Revision Dates:

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Transcatheter mitral valve replacement (TMVR), or transcatheter mitral valve implantation (TMVI), is a minimally invasive intervention aimed to treat mitral regurgitation that would normally require open surgical intervention. During the procedure, a prosthetic valve is delivered via a transeptally or transapically inserted catheter and then deployed over the diseased mitral valve. This type of transcatheter procedure has also been used to place a new valve inside an existing prosthetic valve that is no longer functioning properly in what is referred to as mitral valve-in-valve replacement (MViV).

Less commonly, a transcatheter replacement procedure may be used to treat a calcified mitral valve, and this procedure involves significantly more risk of complication. At this time, TMVI and MViV are intended for patients who are at high risk for conventional open mitral valve repair or replacement.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health considers transcatheter mitral valve-in-valve implantation (TMVI) using an FDA approved device (e.g., Edwards SAPIEN 3 Transcatheter Heart Valve System or Edwards SAPIEN 3 Ultra Transcatheter Heart Valve System) to be medically necessary when either A or B are met:

A. Procedure is recommended and will be performed by Intermountain Health Cardiovascular Clinical Program; **OR**

B. For all other clinicians, **BOTH** of the following criteria must be met:

1. Symptomatic heart disease due to failing (i.e., stenosed, insufficient, or combined) surgical bioprosthetic mitral valve; **AND**

2. There is high or greater risk for open surgical therapy (e.g., predicted 30-day risk of surgical mortality ≥ 8%, based on Society of Thoracic Surgeons [STS] risk score and other clinical co-morbidities unmeasured by the STS risk calculator) as determined by a heart team including a cardiothoracic surgeon.

Transcatheter Mitral Valve Implantation/Replacement, continued

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Billing/Coding Information

CPT CODES

0483T Transcatheter mitral valve implantation/replacement (TMVI) with prosthetic valve; percutaneous approach, including transseptal puncture, when performed

0484T Transcatheter mitral valve implantation/replacement (TMVI) with prosthetic valve; transthoracic exposure (e.g., thoracotomy, transapical)

Key References

1. Alperi A, Avanzas P, Leon V, et al. Current status of transcatheter mitral valve replacement: systematic review and meta-analysis. Front Cardiovasc Med. 2023 May 10; 10:1130212. doi: 10.3389/fcvm.2023.1130212. PMID: 37234369; PMCID: PMC10206247.
2. Bapat V, Rajagopal V, Meduri C, et al. Early Experience With New Transcatheter Mitral Valve Replacement. J Am Coll Cardiol. 2018 Jan 2;71(1):12-21. doi: 10.1016/j.jacc.2017.10.061. Epub 2017 Nov 16. PMID: 29102689.
3. Bonow RO, O'Gara PT, Adams DH, et al. 2019 AATS/ACC/SCAI/STS Expert Consensus Systems of Care Document: Operator and Institutional Recommendations and Requirements for Transcatheter Mitral Valve Intervention: A Joint Report of the American Association for Thoracic Surgery, the American College of Cardiology, the Society for Cardiovascular Angiography and Interventions, and The Society of Thoracic Surgeons. J Am Coll Cardiol. 2020 Jul 7;76(1):96-117. doi: 10.1016/j.jacc.2019.12.002. Epub 2019 Dec 16. Erratum in: J Am Coll Cardiol. 2020 Jul 7;76(1):130. PMID: 31857196.
4. Bonow RO, O'Gara PT, Adams DH, et al. 2020 Focused Update of the 2017 ACC Expert Consensus Decision Pathway on the Management of Mitral Regurgitation: A Report of the American College of Cardiology Solution Set Oversight Committee. J Am Coll Cardiol. 2020 May 5;75(17):2236-2270. doi: 10.1016/j.jacc.2020.02.005. Epub 2020 Feb 14. Erratum in: J Am Coll Cardiol. 2020 Jun 16;75(23):3000. PMID: 32068084
5. Centers for Medicare and Medicaid Services (CMS). Medicare coverage database. https://www.cms.gov/medicare-coveragedatabase/search.aspx.
6. Hayes. Transcatheter mitral valve replacement for mitral regurgitation. Published January 11, 2021. Annual review May 3, 2023. https://evidence.hayesinc.com/.
7. Muller DWM, Farivar RS, Jansz P, et al. Transcatheter Mitral Valve Replacement for Patients With Symptomatic Mitral Regurgitation: A Global Feasibility Trial. J Am Coll Cardiol. 2017 Jan 31;69(4):381-391. doi: 10.1016/j.jacc.2016.10.068. Epub 2016 Dec 28. Erratum in: J Am Coll Cardiol. 2017 Mar 7;69(9):1213. PMID: 28040318.
8. Muller DWM, Sorajja P, Duncan A, et al. 2-Year Outcomes of Transcatheter Mitral Valve Replacement in Patients With Severe Symptomatic Mitral Regurgitation. J Am Coll Cardiol. 2021 Nov 9;78(19):1847-1859. doi: 10.1016/j.jacc.2021.08.060. PMID: 34736561.
9. Otto CM, Nishimura RA, Bonow RO. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021 Feb 2;143(5): e72-e227. doi: 10.1161/CIR.0000000000000923. Epub 2020 Dec 17. Erratum in: Circulation. 2021 Feb 2;143(5): e229. PMID: 33332150.
10. Pislaru S. Surgical procedures for severe chronic mitral regurgitation. Updated November 23, 2020. Reviewed June 2023. http://www.uptodate.com
11. Regueiro A, Ye J, Fam N, et al. 2-Year Outcomes After Transcatheter Mitral Valve Replacement. JACC Cardiovasc Interv. 2017 Aug 28;10(16):1671-1678. doi: 10.1016/j.jcin.2017.05.032. PMID: 28838478.
12. Sorajja P, Moat N, Badhwar V, et al. Initial Feasibility Study of a New Transcatheter Mitral Prosthesis: The First 100 Patients. J Am Coll Cardiol. 2019 Mar 26;73(11):1250-1260. doi: 10.1016/j.jacc.2018.12.066. PMID: 30898200.
13. United States Food and Drug Administration (FDA). SAPIEN 3 and SAPIEN 3 Ultra Transcatheter Heart Valve System. Premarket approval no. P140031. Received November 18, 2020. https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfpma/pma.cfm?id=P140031S125
14. United States National Library of Medicine. http://clinicaltrials.gov

Select Health

Transcatheter Mitral Valve Implantation/Replacement, continued

15. U.S. Food & Drug Administration. FDA Approval. Edwards SAPIEN 3 and SAPIEN 3 Ultra Transcatheter Heart Valve System – P140031/S125. May 13, 2021.
16. Webb JG, Murdoch DJ, Boone RH, et al. Percutaneous Transcatheter Mitral Valve Replacement: First-in-Human Experience With a New Transseptal System. J Am Coll Cardiol. 2019 Mar 26;73(11):1239-1246. doi: 10.1016/j.jacc.2018.12.065. PMID: 30898198.
17. Whisenant, B, Kapadia SR, Eleid MF, et al. One-Year Outcomes of Mitral Valve-in-Valve Using the SAPIEN 3 Transcatheter Heart Valve. JAMA Cardiol. 2020 Nov 1;5(11):1245-1252. doi: 10.1001/jamacardio.2020.2974. PMID: 32745164; PMCID: PMC7391176.
18. Zia Khan, M, Zahid, S, Khan, M. U., et al. Redo Surgical Mitral Valve Replacement Versus Transcatheter Mitral Valve in Valve From the National Inpatient Sample. J Am Heart Assoc. 2021 Sep 7;10(17): e020948. doi: 10.1161/JAHA.121.020948. Epub 2021 Aug 28. PMID: 34459226; PMCID: PMC8649266.

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

TRANSCATHETER PULMONARY VALVE REPLACEMENT

Policy # 483
Implementation Date: 4/11/11
Review Dates: 6/21/12, 6/20/13, 4/17/14, 5/7/15, 4/14/16, 4/27/17, 7/25/18, 4/27/19, 11/11/19, 5/26/21, 9/15/22, 10/17/23, 10/17/24
Revision Dates: 5/21/16, 2/13/20, 6/17/21

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

The pulmonary valve is made up of 3 thin pieces of tissue called leaflets that are arranged in a circle, much like a 3-piece pie. With each heartbeat, the valve opens in the direction of blood flow, into the pulmonary artery and continuing to the lungs, and then closes to prevent blood from flowing backward into the right ventricle of the heart. In pulmonary valve stenosis, 1 or more of the leaflets may be defective or too thick, or the leaflets may not separate from each other properly. If this happens, the valve doesn't open correctly, restricting blood flow. Pulmonary valve stenosis usually occurs when the pulmonary valve doesn't grow properly during fetal development. It's not known what causes the valve to develop abnormally. Adults occasionally have the condition as a complication of another illness, but most of the time pulmonary valve stenosis develops before birth.

As the condition advances, patients may develop symptoms of shortness of breath or fluid retention. In these instances, medications may be prescribed to control shortness of breath, reduce the heart's workload, or regulate the heart's rhythm. For most people, medication alone cannot slow the progression of pulmonary valve disease. Once severe or moderately severe stenosis develops, patients will often require surgical intervention. The 2006 ACC/AHA guidelines recommend balloon valvotomy in symptomatic patients with a peak systolic gradient > 30 mmHg and in asymptomatic patients with peak systolic gradient > 40 mmHg (moderate-to-severe disease). Standard surgery involves open sternotomy with inherent risks to cardiovascular, pulmonary, and infectious complications.

A newer approach has been developed which avoids the necessity of open-heart surgery. Transcatheter valve implantation involves placement of an artificial valve in the affected valve via a catheter inserted through a vein such as the femoral vein or jugular vein. Currently, only two valves have obtained FDA approval, the Medtronic Melody Transcatheter Pulmonary Valve (Medtronic, Inc., Santa Ana, CA) and the Edwards SAPIEN XT Transcatheter Heart Valve (Edwards Lifesciences Corporation Irvine, CA). With both these valves, the heart valve is first crimped down onto the catheter's balloon and then is fished through a vein in the groin and into the right side of the heart where it is placed into position within the pulmonary valve. The small balloon is then inflated to open the valve into position, the catheter is removed from the body, and then immediately becomes the new pulmonary valve.

Harmony TPV is the first FDA-approved transcatheter valve system specifically designed to treat severe pulmonary regurgitation in patients with a native or surgically-repaired right ventricular outflow tract (RVOT); offering patients a minimally invasive treatment option.

The Harmony TPV was designed in an effort to offer a treatment alternative for patients with Congenital Heart Disease (CHD), specifically the 80 percent of CHD patients born with right ventricular outflow tract anomalies who undergo a surgical repair early in life. For these patients, the Harmony TPV provides a less invasive option to restore normal valve function later in life. The minimally invasive TPV therapy

Transcatheter Pulmonary Valve Replacement, continued

builds off of the proven Melody TPV technology, the first transcatheter heart valve available anywhere in the world, which has been implanted in more than 10,000 patients worldwide.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers Transcatheter Pulmonary Valve Replacement with an FDA approved device when either A or B are met:

A. Will be approved If recommended by Intermountain Health Cardiovascular Clinical Program,

OR

B. For all other clinicians, will be approved when either of the following criteria are met:

1. Select Health covers Transcatheter Pulmonary Valve Replacement with an FDA approved device (e.g., Harmony valve) for pediatric and adult patients who meet the following conditions:
   - i) Severe pulmonary regurgitation (i.e., severe pulmonary regurgitation as determined by echocardiography and/or pulmonary regurgitant fraction ≥ 30% as determined by cardiac magnetic resonance imaging)
   - ii) Who have a native or surgically-repaired right ventricular outflow tract and are clinically indicated for surgical pulmonary valve replacement; or
2. Select Health covers Transcatheter Pulmonary Valve Replacement with an FDA approved device (e.g., Melody Transcatheter Pulmonary Valve or Sapien XT) for the management of pediatric and adult patients with prior surgical valve replacement when the following criteria are met:

   i) Dysfunctional RVOT conduits with a clinical indication for intervention, ***and*** either of the following:

   a) ≥ Moderate pulmonary stenosis, **or**

   b) ≥ Moderate pulmonary regurgitation

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For this policy, specifically, there are no CMS criteria available; therefore, the Select Health Commercial policy or InterQual criteria apply. Select Health applies these requirements after careful review of the evidence that supports the clinical benefits outweigh the clinical risks. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit

Transcatheter Pulmonary Valve Replacement, continued

their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

A Medical Technology Assessment performed in March 2011 identified 3 systematic reviews and 8 peer-reviewed journal articles related to percutaneous transcatheter pulmonary valve implantation. Notable, is the fact that the 3 systematic reviews were completed by well-respected organizations: Hayes, NICE, and the Australia and New Zealand Horizon Scanning Network.

Though the Hayes systematic review completed in 2010 was a "Prognosis Notes" rather than a "Directory Report," this reflects the limited evidence currently available on the Melody device; it provided insight into the current state of the evidence, specifically regarding the Melody device. It reported: "PPVI with the Melody Valve system is feasible and may delay open heart surgery in selected patients with failing RVOT conduits, but the long-term efficacy of this intervention to reduce the lifetime risk for exposure to multiple pulmonary valve surgeries is still being evaluated." It also noted, importantly, that long-term durability remains unknown and the appropriate patient selection criteria remain undefined.

The NICE guidance, though completed in 2007, holds remarkably similar findings to that of Hayes' "Prognosis Notes." It notes most of the evidence was derived from a small number of patients, but short-term efficacy was good. It too, noted little evidence related to long-term efficacy; safety was also noted to not be a concern.

The Australia and New Zealand Horizon Scanning Network compared bare-metal stenting (BMS) to percutaneous pulmonary valve implantation (PPVI). Though use of BMS to treat pulmonary stenosis is not commonly performed in the US, this review noted no major complications for BMS or PPVI in the perioperative period, and a complication rate of 9% over the median 28.4-month follow-up period. Additionally, it observed the mean pulmonary artery diastolic pressure increased significantly after PPVI compared to BMS, indicating that pulmonary valvular competence was restored (9 mmHg before BMS vs. 11 mmHg after PPVI; $p = 0.048$). Pulmonary regurgitation was virtually eliminated following PPVI as indicated by measurements of pulmonary regurgitation fraction (41.4% after BMS vs. 3.6% after PPVI; $p < 0.001$). Right ventricular end diastolic volume was also significantly lower (98.3 mL/m^2 vs. 85.3 mL/m^2, $p = 0.021$) and there was a significant improvement in effective right ventricular stroke volume after PPVI, compared with the post-BMS state (32.6 mL/m^2 vs. 41.0 mL/m^2; $p = 0.004$). All indications were for correction of the underlying hemodynamic issues related to the pulmonary valve stenosis. Again, a lack of long-term outcomes was noted.

Seven of these were considered major complications: device instability in 5 patients, which included dislodgement of the device (n = 2) and homograft rupture (n = 3); compression of the left main coronary artery (n = 1); and obstruction of the origin of the right pulmonary artery (n = 1). Five of the patients with major complications required surgical RVOT revision. During the follow-up period, 5 patients were diagnosed with endocarditis, a median 4.9 months after PPVI, which led to valve removal in 3 patients. A stent fracture led to stent embolization in the right pulmonary valve in 1 patient, requiring surgical removal of the Melody valve.

BMS achieved significant reduction in mean right ventricular systolic pressure, mean pulmonary artery to right ventricular pullback gradient, and the mean ratio of right ventricular to systemic pressure. However, PPVI did not produce any statistically significant changes in these measurements.

The primary studies, as could be imagined (as they are the basis for the systematic reviews), demonstrate the same outcomes. They show adequate safety and good short-term efficacy. These studies all suffer from the lack of randomization and short-term endpoints, and for the most part, are small in size. One large case series study indicated that PPVI is a feasible procedure (in a select group of patients) and that most patients who underwent PPVI avoided surgical RVOT revision. Nevertheless, the proportion of patients who required re-do procedures (surgical or transcatheter) was quite substantial and increased over time. The results also indicated that patient outcomes improved substantially with operator experience.

In conclusion, current evidence supports transcatheter pulmonary valve implantation to be safe and efficacious, at least in the short-term. Long-term evidence is lacking related to efficacy and durability of the procedure and the optimal patient candidates are poorly defined.

Transcatheter Pulmonary Valve Replacement, continued

Billing/Coding Information

Covered: For the indications outlined above

CPT CODES

33477 Transcatheter pulmonary valve implantation, percutaneous approach, including pre-stenting of the valve delivery site, when performed

HCPCS CODES

No specific codes identified

Key References

1. Australia and New Zealand Horizon Scanning Network (ANZHSN). (2009) Horizon scanning technology prioritising summary: Percutaneous pulmonary valve implantation. Australian Safety and Efficacy Register of New Interventional Procedures - Surgical. November 2009
2. Bittl, JA. (2010) Natural history and treatment of pulmonic stenosis. 18.3. Last Update: March 23, 2010. UpToDate. Available: http://www.uptodate.com/contents/natural-history-and-treatment-of-pulmonic-stenosis?source=preview&selectedTitle=2%7E42&anchor=H9#H9. Date Accessed: March 12, 2011.
3. Boone, RH, Webb, JG, Horlick, E, et al. (2010). Transcatheter pulmonary valve implantation using the Edwards SAPIEN transcatheter heart valve. Catheter Cardiovasc Interv 75.2: 286-94.
4. Food and Drug Administration (FDA). (2010) Medtronic Melody® Transcatheter Pulmonary Valve - H080002 New Humanitarian Device Approval. Last Update: January 29, 2010. US Department of Health & Human Services. Available: http://www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm199258.htm. Date Accessed: February 4, 2011.
5. Interventional Procedures Programme. (2007) IPG237 Percutaneous pulmonary valve implantation for right ventricular outflow tract dysfunction: guidance. The National Institute for Health and Clinical Excellence (NICE). November 2007
6. Lurz, P, Coats, L, Khambadkone, S, et al. (2008). Percutaneous pulmonary valve implantation: impact of evolving technology and learning curve on clinical outcome. Circulation 117.15: 1964-72.
7. Mayo Clinic Staff. (2011) Pulmonary Valve Stenosis. Last Update: December 11, 2009. Mayo Clinic. Available: http://www.mayoclinic.com/health/pulmonary-valve-stenosis/DS00610. Date Accessed: February 14, 2011.
8. McElhinney, DB, Hellenbrand, WE, Zahn, EM, et al. (2010). Short- and medium-term outcomes after transcatheter pulmonary valve placement in the expanded multicenter US melody valve trial. Circulation 122.5: 507-16.
9. Medtronic. (2011) Melody. Last Update. Medtronic. Available: http://www.medtronic.com/melody/probable-benefits-and-risks.html. Date Accessed: February 14, 2011,
10. Moiduddin, N, Asoh, K, Slorach, C, et al. (2009). Effect of transcatheter pulmonary valve implantation on short-term right ventricular function as determined by two-dimensional speckle tracking strain and strain rate imaging. Am J Cardiol 104.6: 862-7.
11. Raikou, M, McGuire, A, Lurz, P, et al. (2011). An assessment of the cost of percutaneous pulmonary valve implantation (PPVI) versus surgical pulmonary valve replacement (PVR) in patients with right ventricular outflow tract dysfunction. J Med Econ 14.1: 47-52.
12. Ruzyllo, W, Demkow, M, Wlodarska, EK, et al. (2009). [POL-PAVTI--Polish report on transcatheter pulmonary artery valve implantation of Melody-Medtronic prosthesis in the first 14 patients in Poland]. Kardiol Pol 67.10: 1155-61.
13. U.S. Food and Drug Administration. FDA News Release. FDA Approves First in the World Device to Treat Patients with Congenital Heart Disease. March 26, 2021. Available: https://www.fda.gov/news-events/press-announcements/fda-approves-first-world-device-treat-patients-congenital-heart-disease?utm_medium=email&utm_source=govdelivery
14. Vezmar, M, Chaturvedi, R, Lee, KJ, et al. (2010). Percutaneous pulmonary valve implantation in the young 2-year follow-up. JACC Cardiovasc Interv 3.4: 439-48.
15. Zahn, EM, Hellenbrand, WE, Lock, JE, et al. (2009). Implantation of the melody transcatheter pulmonary valve in patients with a dysfunctional right ventricular outflow tract conduit early results from the U.S. Clinical trial. J Am Coll Cardiol 54.18: 1722-9.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Transcatheter Pulmonary Valve Replacement, continued

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

Select Health

MEDICAL POLICY

TRANSTHORACIC ELECTRICAL BIOIMPEDANCE (TEB) TESTING

Policy # 335

Implementation Date:12/21/06

Review Dates: 12/20/07, 12/18/08, 12/19/09, 12/16/10, 12/15/11, 7/18/13, 6/19/14, 6/11/15, 6/16/16, 6/15/17, 7/20/18, 6/13/19, 6/18/20, 6/17/21, 5/19/22, 6/15/23, 6/20/24

Revision Dates: 1/17/14

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Transthoracic electrical bioimpedance (TEB), also referred to as plethysmography or bioimpedance cardiography, has been investigated as a non-invasive method for the determination of cardiac output. TEB relies on the conductivity of blood and the fact that resistance to electrical current in the thorax varies in relation to the amount of blood in the aorta. Blood pumped into the aorta causes a decrease in electrical impedance (resistance) that is inversely proportional to the volume of blood pumped.

Transthoracic electrical bioimpedance measures cardiac output by introducing a low voltage alternating current between 2 sets of electrodes placed on the skin over the thorax. The outer sensors, attached to the neck and chest, transmit a high-frequency, low-amplitude electric current through the thoracic cavity. The inner sensors, placed adjacent to the first set, detect impedance of the electric current in the thoracic cavity. The difference between the initial voltage and that which the device senses moving through the thorax provides a measure of electrical impedance. The magnitude of the decrease in impedance in conjunction with electrocardiographic results allows stroke volume to be estimated, which can be used to calculate cardiac output.

Multiple uses for TEB have been proposed: these include non-invasive diagnosis or monitoring of hemodynamics in patients with suspected or known cardiovascular disease, differentiation of cardiogenic from pulmonary causes of acute dyspnea, optimization of atrioventricular interval for patients with A/V sequential cardiac pacemakers, patients with need of determination for intravenous inotropic therapy, early identification of rejection in post-heart transplant myocardial biopsy patients, cardiac patients with a need for fluid management (excluding patients on dialysis and with cirrhosis of the liver), and management of hypertension.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health does NOT cover transthoracic electrical bioimpedance (TEB) testing. This testing is not found to be medically necessary to manage patients with various cardiac conditions in an outpatient setting.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage,

please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

In a 2003 Hayes Review, Thoracic Electrical Bioimpedance was given a rating of 'C,' reflecting a technology with potential but unproven benefit. The review concluded that the literature suggests that TEB provides accurate measurement of cardiac output in properly selected patients, but that the appropriate clinical role of cardiac output measurement in patient management and its impact on clinical outcomes were poorly defined. Similarly, the Agency for Healthcare Research and Quality's (AHRQ) 2002 review concluded that TEB may have potential value in patient care. However, the accuracy of TEB relative to other measures of hemodynamic parameters could not be evaluated because limited literature and that a focus on clinical outcomes of TEB measurement was needed to evaluate its role in clinical care. The literature published since 2003 is fairly consistent with the observations made in these two reviews, and TEB measurements of hemodynamic parameters also correlate fairly well with those from other invasive or noninvasive techniques, though one study by Leslie et al., found poor agreement between TEB and thermodilution on measures of cardiac output.

A few studies provide some data to address the question of the clinical role of TEB and the impact of measuring cardiac output on changing patient outcomes. Several of these have application to outpatient uses. In Packer et al. for example, 212 patients with chronic heart failure attributable to an ischemic or non-ischemic cause underwent clinical assessment and TEB every 2 weeks for 26 weeks. Three clinical variables (patient visual analog rating of symptoms, NYHA functional class, and systolic blood pressure) and three TEB parameters (thoracic fluid content index, velocity index, and left ventricular ejection time) were independently associated with the occurrence of a heart failure event within 14 days following their measurement. A composite ICG score based on these 3 variables grouped patients into low- (0–3), intermediate- (4–6), and high-risk categories (7–10). Patients in the high-risk group had an 8.4% event rate compared with vs. 3.5% and 1.0% for intermediate- and low-risk patients, respectively. High-risk patients accounted for 41.6% of heart-failure events. While the study suggests that TEB may provide some prognostic information pertinent to treatment planning in this population, the authors caution that TEB parameters should not be used to titrate therapeutic agents or to monitor therapy. Moreover, they noted that the study could not determine whether TEB contributed any unique information to the clinical data already available to clinicians. They indicate that large-scale trials are needed to evaluate whether treatment guided by TEB would have any impact on clinical outcomes.

Peacock et al. examined the use of TEB with patients presenting with dyspnea to an emergency department. After conducting their routine history and physical, and indicating their working diagnosis, physicians reviewed each patient's TEB data and reconsidered their working diagnosis. Of the 89 patients evaluated, the initial diagnosis for 12 (13%) was changed after the physician had reviewed the TEB data. Physicians made changes to the medication plan in 35 patients (39%) after the initial assessment and review of TEB.

A 2004 study by Sharman et al. enrolled 21 patients with uncontrolled hypertension on a treatment protocol that was guided by TEB measurements of hemodynamic parameters. After 215 ± 85 days, average blood pressure had declined from approximately 157/78 mmHg to 142/77 mmHg. Moreover, 57% of participants achieved sustained blood pressure control. A prospective randomized controlled trial in 2006 by Smith et al. also evaluated use of TEB to inform treatment in patients with essential hypertension. Patients (n = 164) underwent a 2-week anti-hypertensive medication washout period and were randomized to a standard care or TEB-informed treatment group. Physicians treating patients in the standard group prescribed anti-hypertensives according to published guidelines, usual practice patterns, and patient characteristics. Physicians treating patients in the TEB-informed group were also provided

Transthoracic Electrical Bioimpedance (TEB) Testing, continued

TEB measurements about their patients and were encouraged to use a hemodynamic treatment algorithm to guide decisions about pharmacologic agents and dosing. Compared with standard care, patients in the TEB group were more likely to achieve blood pressure reduction at or below the target goal of < 140/90 mmHg (77% vs. 57%). A greater percentage of TEB patients also achieved reductions below 130/85mmHg (55% vs. 27%).

These studies suggest some novel clinical applications for TEB in the outpatient setting and that TEB may impact physicians' treatment decisions. However, small sample sizes and lack of replication suggest that these findings should be considered preliminary. Future research is needed to define specific TEB-informed treatment protocols in addition to replicating these findings in larger patient samples.

Billing/Coding Information

Not Covered: Investigational/Experimental/Unproven for this indication

CPT CODES

93701 Bioimpedance-derived physiologic cardiovascular analysis

HCPCS CODES

No specific codes identified

Key References

1. Abdelhammed AI, Smith RD, Levy P, Smits GJ, Ferrario CM. "Noninvasive hemodynamic profiles in hypertensive subjects." Am J Hypertens 18.2 Pt 2 (2005): 51S-59S.
2. Bhalla V, Isakson S, Bhalla MA, et al. "Diagnostic ability of B-type natriuretic peptide and impedance cardiography: testing to identify left ventricular dysfunction in hypertensive patients." Am J Hypertens 18.2 Pt 2 (2005): 73S-81S.
3. Bougault V, Lonsdorfer-Wolf E, Charloux A, Richard R, Geny B, Oswald-Mammosser M. "Does thoracic bioimpedance accurately determine cardiac output in COPD patients during maximal or intermittent exercise?" Chest 127.4 (2005): 1122-31.
4. Braun MU, Schnabel A, Rauwolf T, Schulze M, Strasser RH. "Impedance cardiography as a noninvasive technique for atrioventricular interval optimization in cardiac resynchronization therapy." J Interv Card Electrophysiol 13.3 (2005): 223-9.
5. Braunwald E. "Disorders of the Cardiovascular System." Harrison's Principles of Internal Medicine. Eds. Fauci A, Braunwald E, Isselbacher K, et al. 14th ed. New York: McGraw Hill, 1998.
6. Cardiodynamics Website. CardioDynamics International Corporation. Available: http://www.cardiodynamics.com/index.html. Date Accessed: November 7, 2006.
7. Centers for Medicare and Medicaid Services. Decision Memo for Electrical Bioimpedance for Cardiac Output Monitoring. 2003. Available: http://www.cms.hhs.gov/mcd/viewdecisionmemo.asp?id=23. Date Accessed: November 7, 2006.
8. Colucci WS. Overview of the therapy of heart failure due to systolic dysfunction. 2006. UpToDate. Available: http://www.utdol.com/utd/content/topic.do?topicKey=hrt_fail/6883. Date Accessed: November 6, 2006.
9. DeMarzo AP, Calvin JE, Kelly RF, Stamos TD. "Using impedance cardiography to assess left ventricular systolic function via postural change in patients with heart failure." Prog Cardiovasc Nurs 20.4 (2005): 163-7.
10. "Effect of prophylactic amiodarone on mortality after acute myocardial infarction and in congestive heart failure: meta-analysis of individual data from 6500 patients in randomised trials. Amiodarone Trials Meta-Analysis Investigators." Lancet 350.9089 (1997): 1417-24.
11. Goedhart AD, Kupper N, Willemsen G, Boomsma DI, de Geus EJ. "Temporal stability of ambulatory stroke volume and cardiac output measured by impedance cardiography." Biol Psychol 72.1 (2006): 110-7.
12. Hayes Directory. Electrical Bioimpedance for the Measurement of Cardiac Output. 2003. Winifred S. Hayes, Inc. Available: https://www.hayesinc.com/subscribers/displaySubscriberArticle.do?articleId=1896&targetList=searchArticles.do&query=bioimpedance&icdQuery=&sd1=asearchRelevance&sd2=dtransformdatesort&sd3=atransformdoctype&sd4=atransformsort. Date Accessed: November 7, 2006.
13. Hunt SA. "ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure)." J Am Coll Cardiol 46.6 (2005): e1-82.
14. Jordan HS, Ioannidis JPA, Goudas LC, et al. Thoracic Electrical Bioimpedance. 2002. Agency for Health Care Research and Quality and Centers for Medicare and Medicaid Services. Available: http://www.cms.hhs.gov/mcd/viewtechassess.asp?id=23. Date Accessed: November 7, 2006.
15. Karakitsos DN, Patrianakos AP, Paraskevopoulos A, et al. "Impedance cardiography derived cardiac output in hemodialysis patients: a study of reproducibility and comparison with echocardiography." Int J Artif Organs 29.6 (2006): 564-72.
16. Leslie SJ, McKee S, Newby DE, Webb DJ, Denvir MA. "Non-invasive measurement of cardiac output in patients with chronic heart failure." Blood Press Monit 9.5 (2004): 277-80.
17. Packer M, Abraham WT, Mehra MR, et al. "Utility of impedance cardiography for the identification of short-term risk of clinical decompensation in stable patients with chronic heart failure." J Am Coll Cardiol 47.11 (2006): 2245-52.
18. Parry MJ, McFetridge-Durdle J. "Ambulatory impedance cardiography: a systematic review." Nurs Res 55.4 (2006): 283-91.
19. Peacock WF, Summers RL, Vogel J, Emerman CE. "Impact of impedance cardiography on diagnosis and therapy of emergent dyspnea: the ED-IMPACT trial." Acad Emerg Med 13.4 (2006): 365-71.
20. Santos JF, Parreira L, Madeira J, Fonseca N, Soares LN, Ines L. "Non invasive hemodynamic monitorization for AV interval optimization in patients with ventricular resynchronization therapy." Rev Port Cardiol 22.9 (2003): 1091-8.

Transthoracic Electrical Bioimpedance (TEB) Testing, continued

21. Scherhag A, Kaden JJ, Kentschke E, Sueselbeck T, Borggrefe M. "Comparison of impedance cardiography and thermodilution-derived measurements of stroke volume and cardiac output at rest and during exercise testing." Cardiovasc Drugs Ther 19.2 (2005): 141-7.
22. Scherhag A, Pfleger S, Garbsch E, Buss J, Sueselbeck T, Borggrefe M. "Automated impedance cardiography for detecting ischemic left ventricular dysfunction during exercise testing." Kidney Blood Press Res 28.2 (2005): 77-84.
23. Shochat M, Charach G, Meyler S, et al. "Internal thoracic impedance monitoring: a novel method for the preclinical detection of acute heart failure." Cardiovasc Revasc Med 7.1 (2006): 41-5.
24. Sharman DL, Gomes CP, Rutherford JP. "Improvement in blood pressure control with impedance cardiography-guided pharmacologic decision making." Congest Heart Fail 10.1 (2004): 54-8.
25. Smith RD, Levy P, Ferrario CM. "Value of noninvasive hemodynamics to achieve blood pressure control in hypertensive subjects." Hypertension 47.4 (2006): 771-7.
26. Springfield CL, Sebat F, Johnson D, Lengle S, Sebat C. "Utility of impedance cardiography to determine cardiac vs. noncardiac cause of dyspnea in the emergency department." Congest Heart Fail 10.2 Suppl 2 (2004): 14-6.
27. Stout CL, Van de Water JM, Thompson WM, et al. "Impedance cardiography: can it replace thermodilution and the pulmonary artery catheter?" Am Surg 72.8 (2006): 728-32; discussion 733-4.
28. Tordi N, Mourot L, Matusheski B, Hughson RL. "Measurements of cardiac output during constant exercises: comparison of two non-invasive techniques." Int J Sports Med 25.2 (2004): 145-9.
29. Treister N, Wagner K, Jansen PR. "Reproducibility of impedance cardiography parameters in outpatients with clinically stable coronary artery disease." Am J Hypertens 18.2 Pt 2 (2005): 44S-50S.
30. Van De Water JM, Miller TW, Vogel RL, Mount BE, Dalton ML. "Impedance cardiography: the next vital sign technology?" Chest 123.6 (2003): 2028-33.
31. Ventura HO, Taler SJ, Strobeck JE. "Hypertension as a hemodynamic disease: the role of impedance cardiography in diagnostic, prognostic, and therapeutic decision making." Am J Hypertens 18.2 Pt 2 (2005): 26S-43S.
32. Vijayaraghavan K, Crum S, Cherukuri S, Barnett-Avery L. "Association of impedance cardiography parameters with changes in functional and quality-of-life measures in patients with chronic heart failure." Congest Heart Fail 10.2 Suppl 2 (2004): 22-7.
33. Wynne JL, Ovadje LO, Akridge CM, Sheppard SW, Vogel RL, Van de Water JM. "Impedance cardiography: a potential monitor for hemodialysis." J Surg Res 133.1 (2006): 55-60.
34. Yu CM, Wang L, Chau E, et al. "Intrathoracic impedance monitoring in patients with heart failure: correlation with fluid status and feasibility of early warning preceding hospitalization." Circulation 112.6 (2005): 841-8.
35. Yung GL, Fedullo PF, Kinninger K, Johnson W, Channick RN. "Comparison of impedance cardiography to direct Fick and thermodilution cardiac output determination in pulmonary arterial hypertension." Congest Heart Fail 10.2 Suppl 2 (2004): 7-10.

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

TRICUSPID VALVE IMPLANTATION

Policy # 684
Implementation Date: 6/14/24
Review Dates:
Revision Dates:10/17/24

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Tricuspid valve disease is a condition in which the valve between the two right heart chambers (right ventricle and right atrium) does not function properly. Tricuspid valve disease often occurs with other heart valve problems. Tricuspid regurgitation (TR) is a commonly encountered manifestation of valvular heart disease. Many patients with TR have mild disease that is classified as nonpathological or a normal variant. These patients can remain asymptomatic for some time. Moderate-to-severe TR is usually considered pathological and is associated with poor prognosis. The prevalence of moderate-to-severe TR in the United States has been reported to be greater than 1.6 million. With severe TR, one-year mortality increases and may reach greater than 36%. Surgical repair of TR is generally reserved for patients with advanced disease. These patients are often high-risk candidates for open surgical procedures, making the percutaneous or transcatheter minimally invasive approach attractive for this population. The current standard of care is open surgical valve replacement or repair surgery (Otto 2023; Hayes 2023).

Transcatheter heart valve replacement and repair are relatively new interventional procedures involving the insertion of an artificial heart valve or repair device using a catheter, rather than through open heart surgery, or surgical valve replacement. The point of entry is typically either the femoral vein (antegrade) or femoral artery (retrograde), or directly through the myocardium via the apical region of the heart. For valve replacement surgery, an expandable prosthetic heart valve is pressed onto a catheter and then deployed at the site of the diseased native valve.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers transcatheter tricuspid valve implantation when either A or B are met.

A. Procedure is recommended and will be performed by Intermountain Health Cardiovascular Clinical Program; **OR**

B. For all other clinicians, Select Health will cover transcatheter tricuspid valve implantation with an FDA approved valve for patients who meet the following criteria:

   1. For prior failed tricuspid valve replacement, patients with severe symptomatic tricuspid regurgitation despite guideline directed medical therapy; or

   2. For native tricuspid valve, patients with primary or secondary tricuspid regurgitation that is moderate-to-severe or severe.

SELECT HEALTH ADVANTAGE (MEDICARE/CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Billing/Coding Information

CPT CODES

0545T Transcatheter tricuspid valve annulus reconstruction with implantation of adjustable annulus reconstruction device, percutaneous approach

0646T Transcatheter tricuspid valve implantation/replacement (TTVI) with prosthetic valve, percutaneous approach, including right heart catheterization, temporary pacemaker insertion, and selective right ventricular or right atrial angiography, when performed

Key References

1. FDA Executive Summary. TriClip G4 System. February 13, 2024. https://www.fda.gov/media/176091/download
2. Sorajjo, P., et al. Transcatheter Repair for Patients with Tricuspid Regurgitation. *The New England Journal of Medicine*. May 18, 2023. 388:20.

Revision History

Revision Date	Summary of Changes
10/17/24	For Commercial Plan Policy, incorporated coverage criteria for both prior failed tricuspid valve implantation and native tricuspid valve: "1. For prior failed tricuspid valve replacement, patients with severe symptomatic tricuspid regurgitation despite guideline directed medical therapy; or 2. For native tricuspid valve, patients with primary or secondary tricuspid regurgitation that is moderate-to-severe or severe." Also, added coverage for CPT code 0545T with criteria.

Select Health

Select Health

MEDICAL POLICY

VENTRICULAR ASSIST DEVICES

Policy # 187

Implementation Date:7/98

Review Dates: 1/4/00, 2/27/01, 4/16/02, 10/23/03, 11/18/04, 3/25/05, 4/20/06, 10/18/07, 10/22/09, 10/22/10, 2/16/12, 4/25/13, 2/20/14, 3/19/15, 2/11/16, 2/16/17, 2/15/18, 2/5/19, 2/11/20, 2/18/21, 3/21/22, 3/23/23, 1/30/24, 2/10/25

Revision Dates: 8/22/06, 10/30/06, 10/23/08, 2/24/25

Description

A ventricular assist device (VAD) is a mechanical pump that relieves the native ventricle by assisting in pumping blood through the body. The VAD requires a control system and a power source that are maintained outside the body. The VAD pump may be placed internally or externally but the control system and power source are maintained outside the body. Sometimes called, "bridge-to-transplant" (BTT), VADs were originally intended for use in patients awaiting heart transplant who required additional assistance for survival. As VAD technology has improved and the supply of transplantable hearts remains limited, VADs are sometimes used for long-term,"destination therapy" (DT) in severe heart-failure patients.

The first FDA approval of a VAD for this indication was in 1994. A variety of devices have received approval from the U.S. Food and Drug Administration (FDA), encompassing both biventricular and left ventricular devices, as well as devices that are intended to be used in the hospital setting alone and those that can be used as an outpatient. Devices that can be used in an outpatient setting while the patient awaits a heart transplantation, as destination therapy or bridge to recovery, include Abbott HeartMate II LVAD, Abbott HeartMate 3 LVAD and Medtronic HeartWare LVAD (HVAD). In these systems, the device is surgically placed entirely within the thoracic cavity except for the HeartMate II. The LVAD is placed under the diaphragm. All devices are connected to a controller and power sources by a percutaneous drive line.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers all FDA approved* ventricular assist devices, when either A or B are met:

A. Ventricular assist devices will be approved if recommended by Intermountain Health Cardiovascular Clinical Program; or

B. For all other clinicians, one of the following criteria must be met:

1. Members with ventricular dysfunction secondary to post cardiotomy syndrome, post myocardial infarct ventricular dysfunction, and chronic ischemic and non-ischemic cardiomyopathy who have undergone advance heart failure evaluation and treatment that may include but is not limited to inotropic therapy or temporary mechanical support; or

Ventricular Assist Devices, continued

2. As a bridge to transplant for members who are awaiting heart transplantation; or

3. As destination therapy for members with severe (NYHA Class IV) heart failure, and who are ineligible for heart transplantation due to age or co-morbidities.

**FDA approved is defined as devices that have been granted approval through a Pre-Marketing Approval (PMA) process or have gained FDA approval as Investigational Exempt Devices (IDE) and are categorized as Category "B" devices by the FDA.*

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information
There are two types of Mechanical Circulatory Support (MCS) devices: Short-term and intermediate to long-term devices.

1) Short-Term devices:

These devices are designed to provide hemodynamic support for a wide range of clinical conditions, ranging from prophylactic insertion for high-risk invasive coronary artery procedures to the management of cardiogenic shock, acute decompensated heart failure, or cardiopulmonary arrest. Each of these devices provides circulatory support by performing work for a failing left or right ventricle or both and are used as bridge to recovery, transplantation and intermediate to long-term MCS or facilitating very high risk percutaneous coronary artery intervention, valve procedure and electrophysiologic procedures.

a) Intra-Aortic Balloon Pump (IABP): Uses the counterpulsation system to displace the blood to the proximal aorta by inflation during diastole and reduce the afterload during systole by rapid balloon deflation. It can provide up to 0.5 L/min flow and be inserted via femoral or axillary arteries.

b) Percutaneous Short-Term Left Ventricular Assist Devices

i) **Impella (Abiomed)**: Intravascular micro-axial continuous flow pump that is inserted via femoral or axillary artery and positioned across the aortic valve. Currently, two versions are available for left side support: Impella (CP & 5.5) with flow up to 4.3L/min with the CP and up to 6L/min with the 5.5.

ii) **TandemHeart (TandemLife)**: Centrifugal continuous pump inserted in the left atrium using transseptal approach from the femoral vein (max flow 4.0L/min).

c) Percutaneous Short-Term Right Ventricular Assist Devices:

i) **Impella RP Flex (Abiomed):** Like other Impella devices, it is inserted in an anterograde fashion via the femoral vein, crossing the tricuspid and pulmonary valves (4.0 L/min)

ii) **Protek Duo (TandemLife):** A dual Lumen cannula, draining right atrium and superior vena cava blood via proximal vents into an extracorporeal centrifugal pump, which is then delivered back directly to the Pulmonary artery. It is inserted via the right internal jugular vein

Ventricular Assist Devices, continued

and crosses the tricuspid and pulmonary valve and connected to an extracorporeal centrifugal pump. (Flow 4.0 L/min).

2) Intermediate to Long-Term MCS Devices

These devices are designed to support the patient while waiting for heart transplantation (Bridge to Transplantation) or as permanent therapy (Destination Therapy). Currently there is one LVAD that the FDA has approved for Bridge to Transplantation (BT) and Destination Therapy (DT):

a) Third Generation LVADs**:** These are continuous, centrifugal flow devices. These are durable LVADs and have similar basic design to the second generation LVADs however they are placed in the pericardium and attached to the LV apex with a sewing ring.

i. HeartMate 3 LVAD (Abbott): is a fully magnetically levitated continuous centrifugal flow pump that uses magnetic fields to float the device's rotor, creating a contact-free, friction-free environment. The pump is designed for implant in the pericardial space and can provide 2.5 - 10 liters/minute of blood flow**.** *It is currently the only FDA approved LVAD in production*.

3) Total Artificial Heart:

a) The SynCardia temporary Total Artificial Heart (TAH) is intended as a cardiac replacement device for bridging to transplantation for patients with severe biventricular failure, intractable arrythmias or complex congenital heart disease.
b) The artificial heart consists of two ventricles, each with two Medtronic-Hall tilting disc valves. The ventricles are orthotopically positioned and attached to the patient's remnant atria and great vessels with atrial sewing cuffs and vascular graft material.
c) The TAH is driven by metered pulses of compressed air delivered by a C2 hospital driver that adjusts the pumping rate, driveline inflation pressures and percent systole. A flexing diaphragm is found in each ventricle to pump the blood from the ventricle when the diaphragms are inflated.
d) The driveline from the base of each ventricle exits below the sternum and connects to an 8-foot driveline extension that goes to a C2 driver (console).
e) The TAH can pump up to 9.5 L/min of flow through both ventricles. With careful control of the console, the patient can ambulate around the hospital and hospital grounds for 1 to 2 hours on the C2 driver and battery supplies.
f) TAH patients are not permitted to leave the hospital while on the larger C2 console; however, the portable, 13.5-pound Freedom Driver is available for use outside the hospital. Participation in a cardiac rehabilitation program is possible while on the TAH. The 70 cc TAH can only be used in patients with a body surface area (BSA) of 1.7 m^2 or greater and an A-P distance from posterior sternum to anterior vertebral body at T-10 on CT scan of ≥ 10 cm.

Billing/Coding Information

Covered: For the conditions outlined above

CPT CODES

0451T	Insertion or replacement of a permanently implantable aortic counterpulsation ventricular assist system, endovascular approach, and programming of sensing and therapeutic parameters; complete system (counterpulsation device, vascular graft, implantable vascular hemostatic seal, mechano-electrical skin interface and subcutaneous electrodes)
0452T	; aortic counterpulsation device and vascular hemostatic seal
0455T	Removal of permanently implantable aortic counterpulsation ventricular assist system; complete system (aortic counterpulsation device, vascular hemostatic seal, mechano-electrical skin interface and electrodes)
0456T	; aortic counterpulsation device and vascular hemostatic seal
0458T	; subcutaneous electrode

Ventricular Assist Devices, continued

0459T	Relocation of skin pocket with replacement of implanted aortic counterpulsation ventricular assist device, mechano-electrical skin interface and electrodes
0460T	Repositioning of previously implanted aortic counterpulsation ventricular assist device; subcutaneous electrode
0461T	; aortic counterpulsation device
0463T	Interrogation device evaluation (in person) with analysis, review and report, includes connection, recording and disconnection per patient encounter, implantable aortic counterpulsation ventricular assist system, per day
33975	Insertion of ventricular assist device; extracorporeal, single ventricle
33976	; extracorporeal, biventricular
33977	Removal of ventricular assist device; extracorporeal, single ventricle
33978	; extracorporeal, biventricular
33979	Insertion of ventricular assist device, implantable intracorporeal, single ventricle
33980	Removal of ventricular assist device, implantable intracorporeal, single ventricle
33981	Replacement of extracorporeal ventricular assist device, single or biventricular, pump(s), single or each pump
33990	Insertion of ventricular assist device, percutaneous including radiological supervision and interpretation; arterial access only
33991	Insertion of ventricular assist device, percutaneous including radiological supervision and interpretation; both arterial and venous access, with transseptal puncture
33992	Removal of percutaneous ventricular assist device at separate and distinct session from insertion
33993	Repositioning of percutaneous ventricular assist device with imaging guidance at separate and distinct session from insertion
33999	Unlisted procedure, cardiac surgery
92970	Cardioassist-method of circulatory assist; internal
92971	; external
93750	Interrogation of ventricular assist device (VAD), in person, with physician or other qualified health care professional analysis of device parameters (eg, drivelines, alarms, power surges), review of device function (eg, flow and volume status, septum status, recovery), with programming, if performed, and report

HCPCS CODES

Q0480	Driver for use with pneumatic ventricular assist device, replacement only
Q0481	Microprocessor control unit for use with pneumatic ventricular assist device, replacement only
Q0482	Microprocessor control unit for use with electric/pneumatic combination ventricular assist device, replacement only
Q0483	Monitor/display module for use with pneumatic ventricular assist device, replacement only
Q0484	Monitor/display module for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0485	Monitor control cable for use with pneumatic ventricular assist device, replacement only
Q0486	Monitor control cable for use with electric/pneumatic ventricular assist device, replacement only

Ventricular Assist Devices, continued

Q0487	Leads (pneumatic/electrical) for use with any type electric/pneumatic ventricular assist device, replacement only
Q0488	Power pack base for use with pneumatic ventricular assist device, replacement only
Q0489	Power pack base for use with electric/pneumatic ventricular assist device, replacement only
Q0490	Emergency power source for use with pneumatic ventricular assist device, replacement only
Q0491	Emergency power source for use with electric/pneumatic ventricular assist device, replacement only
Q0492	Emergency power supply cable for use with pneumatic ventricular assist device, replacement only
Q0493	Emergency power supply cable for use with electric/pneumatic ventricular assist device, replacement only
Q0494	Emergency hand pump for use with electric/pneumatic ventricular assist device, replacement only
Q0495	Battery power pack charger for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0496	Battery for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0497	Battery clip for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0498	Holster for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0499	Belt/vest for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0500	Filters for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0502	Mobility cart for pneumatic ventricular assist device, replacement only
Q0503	Battery for pneumatic ventricular assist device, replacement only, each
Q0506	Battery, lithium-ion, for use with electric or electric/pneumatic ventricular assist device, replacement only
Q0507	Miscellaneous supply or accessory for use with an external ventricular assist device
Q0508	Miscellaneous supply or accessory for use with an implanted ventricular assist device

Revision History

Revision Date	Summary of Changes
2/24/25	For Commercial Plan Policy, added qualifying option of ventricular assist devices being approved if recommended by Intermountain Health Cardiovascular Clinical Program.

Key References

1. Aaronson KD, Slaughter MS, Miller LW HeartWare Ventricular Assist Device (HVAD) Bridge to Transplant ADVANCE Trial Investigators. Use of an intrapericardial, continuous-flow, centrifugal pump in patients awaiting heart transplantation. Circulation. 2012 Jun;125(25):3191-200.
2. Copeland JG, Smith RG, Arabia FA et al. Cardiac replacement with a total artificial heart as a bridge to transplantation. CardioWest Total Artificial Heart Investigators N Engl J Med. 2004;351(9):859.
3. Copeland JG, Copeland H, Gustafson M. Experience with more than 100 total artificial heart implants.J Thorac Cardiovasc Surg. 2012 Mar;143(3):727-34.

Ventricular Assist Devices, continued

4. Elbadawi A, Elgendy IY, Mahmoud K, et al. Temporal trends and outcomes of mechanical complications in patients with acute myocardial infarction. J Am Coll Cardiol Intv 2019; 12:1825–36.
5. Jurmann MJ, Siniawski H, Erb M, Drews T, Hetzer R. Initial experience with miniature axial flow ventricular assist devices for postcardiotomy heart failure. Ann Thorac Surg. 2004;77(5):1642.
6. Kapur NK, Esposito ML, Bader Y, et al. Mechanical circulatory support devices for acute right ventricular failure. Circulation 2017; 136:314–26.
7. Kirklin JK, Pagani FD, Goldstein DJ et al, American Association for thoracic Surgery/International Society for Heart and Lung Transplantation guidelines on selected topics in mechanical circulatory support.
8. Mehra MR, Naka Y, Uriel N, Goldstein DJ et al. A Fully Magnetically Levitated Circulatory Pump for Advanced Heart Failure. MOMENTUM 3 Investigators. N Engl J Med. 2017;376(5):440.
9. Meyer A, Slaughter M. The total artificial heart. Panminerva Med. 2011;53(3):141.
10. Miller LW, Pagani FD, Russell SD et al HeartMate II Clinical Investigators. Use of a continuous-flow device in patients awaiting heart transplantation. N Engl J Med. 2007;357(9):885.
11. Molina EJ, Shah P, Kiernan MS et al. The Society of Thoracic Surgeons Intermacs 2020 Annual Report. Ann Thorac Surg 2021; 111:778-92.
12. Rihal CS, Naidu SS, Givertz MMet al, 2015 SCAI/ACC/HFSA/STS Clinical Expert Consensus Statement on the Use of Percutaneous Mechanical Circulatory Support Devices in Cardiovascular Care: Endorsed by the American Heart Assocation, the Cardiological Society of India, and Sociedad Latino Americana de Cardiologia Intervencion; Affirmation of Value by the Canadian Association of Interventional Cardiology-Association Canadienne de Cardiologie d'intervention.Cardiovascular Angiography and Interventions (SCAI), Heart Failure Society of America (HFSA), Society of Thoracic Surgeons (STS), American Heart Association (AHA), and American College of Cardiology (ACC). J Am Coll Cardiol. 2015;65(19).
13. Rogers JG, Pagani FD, Tatooles AJ, Bhat G, Slaughter MS et al. Intrapericardial Left Ventricular Assist Device for Advanced Heart Failure. N Engl J Med. 2017;376(5):451.
14. Siegenthaler MP, Brehm K, Strecker T, Hanke T, Nötzold A, Olschewski M, Weyand M, Sievers H, Beyersdorf F. The Impella Recover microaxial left ventricular assist device reduces mortality for postcardiotomy failure: a three-center experience. J Thorac Cardiovasc Surg. 2004;127(3):812.
15. Thiele H, Lauer B, Hambrecht R, Boudriot E, Cohen HA, Schuler G. Reversal of cardiogenic shock by percutaneous left atrial-to-femoral arterial bypass assistance. Circulation. 2001;104(24):2917.

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

Ventricular Assist Devices, continued

19. Wessex Institute for Health Research and Development, Development and Evaluation Committee. Left ventricular assist devices (LVADs) for end stage heart failure. Southampton, UK: Wessex Institute; 1999.
20. Westaby S. Ventricular assist devices as destination therapy. Surg Clin North Am. 2004;84(1):91-123.
21. www.clinicaltrials.gov. search term "ventricular assist device." Accessed 5/4/2006.
22. www.clinicaltrials.gov. search term "ventricular assist device." Accessed 5/4/2006.
23. 2001 Heart and stroke statistical update. Dallas: American Heart Association, 2000.

Select Health

MEDICAL POLICY

VERTICAL AUTOPROFILING (VAP) CHOLESTEROL TEST

Policy # 298
Implementation Date:2/15/06
Review Dates: 5/17/07, 4/24/08, 4/23/09, 2/18/10, 5/19/11, 6/21/12, 6/20/13, 4/17/14, 5/7/15, 4/14/16, 4/27/17, 7/20/18, 4/27/19, 4/15/20, 12/23/20, 7/5/21, 7/21/22, 8/17/23, 8/8/24
Revision Dates: 12/28/20

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Advantage (Medicare/CMS), and Select Health Community Care (Medicaid/CHIP) plans. Refer to the "Policy" section for more information.

Description

Coronary heart disease (CHD) affects about 14 million men and women in the United States. Atherosclerosis is an important risk factor in the development of CHD. Routine cholesterol screening, which measures HDL, LDL (calculated), and triglycerides is a major component of CHD risk management. The Vertical Auto Profile (VAP) cholesterol test from Atherocare not only includes these standard tests, but also, measures atherogenic remnant lipids (VLDL and IDL), Lp(a), LDL pattern density, and HDL subtypes. This additional testing purports to improve CHD risk stratification by measuring these additional risk factors along with the traditional cholesterol panel. By measuring these lipid factors, the test also provides a screen for metabolic syndrome, a constellation of metabolic risk factors that increase risk for CHD.

The VAP test is the only commercially available test that measures possible cardiovascular risk factors Lp(a), LDL particle size, lipoprotein remnants, and HDL subfractions, at the same time. Alternatively, providers can order each test individually. Additionally, many other tests are available to assess cardiovascular risk. Tests covered by Select Health include fasting lipid profiles, PLAC testing, and hs-CRP. Other risk factors for CHD can be assessed through a variety of methods including the Framingham Risk Assessment tool.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Select Health does NOT cover vertical autoprofiling (VAP) cholesterol testing for assessment of cardiovascular risk. This test is not medically necessary, as additional information obtained from VAP testing does not change treatment or clinical outcome.

SELECT HEALTH MEDICARE

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

Vertical Autoprofiling (VAP) Cholestrol Test, continued

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Coverage is determined by the State of Utah Medicaid program; if Utah State Medicaid has no published coverage position and InterQual criteria are not available, the Select Health Commercial criteria will apply. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information
The extant literature is somewhat equivocal on the prognostic value of the risk factors the VAP test adds to the traditional lipid panel:

Remnant lipoproteins: Fukushima et al.'s 2004 2-year follow-up study of 240 diabetes patients with or without CAD found remnant lipoproteins to be a strong independent risk factor for CAD. Inoue et al. reported similar findings in their 2004 study of 188 CAD patients and 68 controls. In contrast, Imke et al.'s study of 1,156 Japanese-American men over 7 years found that remnant lipoproteins levels did not improve the prediction of future CHD incidence beyond that already provided by total triglycerides.

Lp(a): Ariyo et al. studied 5,888 adults over 7.5 years (median) and concluded that Lp(a) level is an independent risk factor of future vascular events in men, but not in women. Danesh et al.'s meta-analysis of 27 studies supported the association between CHD and Lp(a) but called for additional research to determine the extent to which this relationship is causal. Moliterno et al.'s study of 140 African American subjects concluded that Lp(a) is not an independent risk factor for CAD in that population.

LDL particle size: Berneis et al. studied LDL size in 38 type II diabetics and concluded that, among other lipid parameters, LDL particle size is the strongest predictor of CHD and atherosclerosis. Mykkanen et al., in contrast, studied 86 patients who were MI-free over 3.5 years and observed that LDL particle size was not an independent risk factor of CHD events once controlling for diabetes status. St-Pierre et al.'s study of 2,072 men over 13 years demonstrated that accumulation of small LDL particles was primarily responsible for the risk attributable to LDL size.

HDL subtypes: Lamarche et al.'s 1990 study of 1,169 men followed over 11 years found a statistical association between HDL2 and CHD but concluded that that in the clinical setting, measurement of HDL subtypes provides no additional information about CHD risk over traditional lipid risk markers. Sweetnam et al. followed 4,860 men between 3−5 years and observed an inverse association between HDL2 and HDL3 cholesterol and the incidence of CHD. They further noted that the predicting adding HDL subtype measurement to traditional HDL did not improve predication of CHD risk.

Only 2 studies located for this literature review examined the impact of treating these risk factors on future atherosclerosis or CHD events. Campos et al. administered pravastatin or placebo to 837 MI survivors and matched controls with similarly elevated LDL and tracked outcomes over 5 years. In patients taking placebo, large LDL predicted coronary events, but this association was not present in patients taking pravastatin. However, the authors concluded that identifying LDL size 5, not to be very useful clinically since elevated LDL cholesterol and large LDL are both effectively treated in the same manner. Miller et al. prospectively followed 213 men enrolled in a cardiac risk reduction program that included lipid-altering therapies with counseling and training in the modification of diet, exercise, and other lifestyle factors. After four years, patients with higher dense LDL levels showed significant benefit from the program while subjects with higher buoyant (less dense) LDL had no benefit from the intervention.

ATP-III identifies lipoprotein remnants, Lp(a), LDL particle size, and HDL subtypes as "emerging risk factors", suggesting that their association with CHD and the impact of modifying them on CHD risk is not well understood. Additional prospective trials are needed to more fully explain the relationship between these risk factors and atherosclerosis and future CHD events. Research is particularly needed to determine whether adding these emerging risk factors to traditional lipid panels adds any value to the prediction of CHD. The interaction between these emerging risk factors and other lipid and non-lipid risk factors in producing CHD risk must also be examined. Finally, research is needed to determine whether measuring these risk factors would have any unique impact on treatment decisions, whether treatments can be sufficiently tailored for specific lipid profiles, and whether patients with particular lipid parameters respond differently to lipid lowering therapies.

Vertical Autoprofiling (VAP) Cholestrol Test, continued

A June 2012 literature review identified the 2010 ACCF/AHA Guideline for Assessment of Cardiovascular Risk in Asymptomatic Adults and does not recommend lipid/lipoprotein subfraction. Similarly, the 2011 National Lipid Association Expert Panel did not recommend HDL and LDL subfraction measurements for clinical assessment. However, Apo-B or LDL particle measurement and LPa are endorsed by NLA reasonable for many subjects at intermediate risk, those with a positive family history, and those with recurrent events, and selectively in those with coronary heart disease and CHD equivalents. These recommendations follow recent studies and metaanalyses that indicate that LDL particle number (measured by NML or as Apo-B) may be a letter indicator of CHD risk than LDLc or non-HDLc.

Billing/Coding Information

Not Covered: Investigational/Experimental/Unproven for this indication (when used in this combination)

CPT CODES

3011F	Lipid panel results documented and reviewed (must include total cholesterol, HDL-C, triglycerides and calculated LDL-C) (CAD)
80050	General health panel This panel must include the following: Comprehensive metabolic panel (80053) Blood count, complete (CBC), automated and automated differential WBC count (85025 or 85027 and 85004) OR Blood count, complete (CBC), automated (85027) and appropriate manual differential WBC count (85007 or 85009) Thyroid stimulating hormone (TSH) (84443)
80061	Lipid panel. This panel must include the following: Cholesterol, serum, total (82465) Lipoprotein, direct measurement, high density cholesterol (HDL cholesterol) (83718) Triglycerides (84478)
82465	Cholesterol, serum or whole blood, total
83698	Lipoprotein-associated phospholipase A2 (Lp-PLA2)
83701	Lipoprotein, blood; high-resolution fractionation and quantitation of lipoproteins including lipoprotein subclasses when performed (e. electrophoresis, ultracentrifugation)
83718	Lipoprotein, direct measurement; high density cholesterol (HDL cholesterol)
83719	Lipoprotein, direct measurement; VLDL cholesterol
83721	Lipoprotein, direct measurement; LDL cholesterol
84478	Triglycerides

HCPCS CODES

No specific codes identified

Key References

1. Adams J, Apple F. Cardiology patient page. New blood tests for detecting heart disease. Circulation. 2004; 109(3): E12-4.
2. Alaupovic P, Mack WJ, Knight-Gibson C, Hodis HN. The role of triglyceride-rich lipoprotein families in the progression of atherosclerotic lesions as determined by sequential coronary angiography from a controlled clinical trial. Arterioscler Thromb Vasc Biol. 1997; 17(4):715-22.
3. Ariyo AA, Thach C, Tracy R. Lp(a) lipoprotein, vascular disease, and mortality in the elderly. N Engl J Med. 2003; 349(22):2108-15.
4. Atherotech. The VAP™ Cholesterol Test as a Replacement for the Traditional Lipid Profile. 2005.
5. Atherotech Website. http://www.atherotech.com. 2005.
6. Alaupovic P, Mack WJ, Knight-Gibson C, Hodis HN. The role of triglyceride-rich lipoprotein families in the progression of atherosclerotic lesions as determined by sequential coronary angiography from a controlled clinical trial. Arterioscler Thromb Vasc Biol. 1997; 17(4):715-22.
7. Berneis K, Jeanneret C, Muser J, Felix B, Miserez AR. Low-density lipoprotein size and subclasses are markers of clinically apparent and non-apparent atherosclerosis in type 2 diabetes. Metabolism. 2005; 54(2):227-34.
8. Budde T, Fechtrup C, Bosenberg E, et al. Plasma Lp(a) levels correlate with number, severity, and length-extension of coronary lesions in male patients undergoing coronary arteriography for clinically suspected coronary atherosclerosis. Arterioscler Thromb. 1994; 14(11):1730-6.
9. Campos H, Moye LA, Glasser SP, Stampfer MJ, Sacks FM. Low-density lipoprotein size, pravastatin treatment, and coronary events. Jama. 2001; 286(12):1468-74.
10. Coronary heart disease overview. http://www.emedicinehealth.com/Articles/10951-1.asp. 2005.

Select Health

Vertical Autoprofiling (VAP) Cholestrol Test, continued

11. Cromwell WC, Otvos JD, Keyes MJ, et al. (2011). LDL Particle Number and Risk of Future Cardiovascular Disease in the Framingham Offspring Study - Implications for LDL Management. J Clin Lipidol. 2007 Dec;1(6):583-92.
12. Danesh J, Collins R, Peto R. Lipoprotein(a) and coronary heart disease. Meta-analysis of prospective studies. Circulation. 2000; 102(10):1082-5.
13. Davidson MH, Ballantyne CM, Jacobson TA, et al. (2011). Clinical utility of inflammatory markers and advanced lipoprotein testing: advice from an expert panel of lipid specialists. J Clin Lipidol. Sep-Oct;5(5):338-67.
14. Evans RW, Shpilberg O, Shaten BJ, Ali S, Kamboh MI, Kuller LH. Prospective association of lipoprotein(a) concentrations and apo(a) size with coronary heart disease among men in the Multiple Risk Factor Intervention Trial. J Clin Epidemiol. 2001; 54(1):51-7.
15. Fukushima H, Sugiyama S, Honda O, et al. Prognostic value of remnant-like lipoprotein particle levels in patients with coronary artery disease and type II diabetes mellitus. J Am Coll Cardiol. 2004; 43(12):2219-24.
16. Gardner CD, Fortmann SP, Krauss RM. Association of small low-density lipoprotein particles with the incidence of coronary artery disease in men and women. Jama. 1996; 276(11):875-81.
17. Hayes Technology Directory. High-density lipoprotein subclass testing for screening, diagnosis, and management of dyslipidemia and cardiovascular disease. 2000.
18. Greenland P, Alpert JS, Beller GA, et al. (2010). 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. Dec 14;56(25): e50-103.
19. Imhof A, Rothenbacher D, Khuseyinova N, et al. Plasma lipoprotein Lp(a), markers of haemostasis and inflammation, and risk and severity of coronary heart disease. Eur J Cardiovasc Prev Rehabil. 2003; 10(5):362-70.
20. Imke C, Rodriguez BL, Grove JS, et al. Are remnant-like particles independent predictors of coronary heart disease incidence? The Honolulu Heart study. Arterioscler Thromb Vasc Biol. 2005; 25(8):1718-22.
21. Inoue T, Uchida T, Kamishirado H, et al. Remnant-like lipoprotein particles as risk factors for coronary artery disease in elderly patients. Horm Metab Res. 2004; 36(5):298-302.
22. Kulkarni KR, Garber DW, Jones MK, Segrest JP. Identification and cholesterol quantification of low density lipoprotein subclasses in young adults by VAP-II methodology. J Lipid Res. 1995; 36(11):2291-302.
23. Kulkarni KR, Marcovina SM, Krauss RM, Garber DW, Glasscock AM, Segrest JP. Quantification of HDL2 and HDL3 cholesterol by the Vertical Auto Profile-II (VAP-II) methodology. J Lipid Res. 1997; 38(11):2353-64.
24. Kulkarni KR, Markovitz JH, Nanda NC, Segrest JP. Increased prevalence of smaller and denser LDL particles in Asian Indians. Arterioscler Thromb Vasc Biol. 1999; 19(11):2749-55.
25. Lamarche B, Moorjani S, Cantin B, Dagenais GR, Lupien PJ, Despres JP. Associations of HDL2 and HDL3 subfractions with ischemic heart disease in men. Prospective results from the Quebec Cardiovascular Study. Arterioscler Thromb Vasc Biol. 1997; 17(6):1098-105.
26. Luc G, Bard JM, Arveiler D, et al. Lipoprotein (a) as a predictor of coronary heart disease: the PRIME Study. Atherosclerosis. 2002; 163(2):377-84.
27. Medline Plus. Coronary Heart Disease. Medical Encyclopedia. 2005; http://www.nlm.nih.gov/medlineplus/ency
28. Merck Manual Online. Arteriosclerosis. 17 ed. Vol. http://www.merck.com. (Berkow M, ed.).
29. Miller BD, Alderman EL, Haskell WL, Fair JM, Krauss RM. Predominance of dense low-density lipoprotein particles predicts angiographic benefit of therapy in the Stanford Coronary Risk Intervention Project. Circulation. 1996; 94(9):2146-53.
30. Moliterno DJ, Jokinen EV, Miserez AR, et al. No association between plasma lipoprotein(a) concentrations and the presence or absence of coronary atherosclerosis in African-Americans. Arterioscler Thromb Vasc Biol. 1995; 15(7):850-5.
31. Mykkanen L, Kuusisto J, Haffner SM, Laakso M, Austin MA. LDL size and risk of coronary heart disease in elderly men and women. Arterioscler Thromb Vasc Biol. 1999; 19(11):2742-8.
32. National Cholesterol Education Program. (National Institutes of Health). Detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). 2002.
33. Nishino M, Malloy MJ, Naya-Vigne J, Russell J, Kane JP, Redberg RF. Lack of association of lipoprotein(a) levels with coronary calcium deposits in asymptomatic postmenopausal women. J Am Coll Cardiol. 2000; 35(2):314-20.
34. Nowicka G, Jarosz A. LpAI in HDL subfractions: serum levels in men and women with coronary heart disease and changes under hypolipemic therapy. Clin Chim Acta. 2001; 306(1-2):43-9.
35. Rallidis LS, Pitsavos C, Panagiotakos DB, Sinos L, Stefanadis C, Kremastinos DT. Non-high density lipoprotein cholesterol is the best discriminator of myocardial infarction in young individuals. Atherosclerosis. 2005; 179(2):305-9.
36. Rosenson RS. Lipoprotein classification; metabolism; and role in atherosclerosis. UpToDate Online. 2005; http://www.utdol.com/.
37. Rosenson RS. HDL metabolism and approach to the patient with low HDL-cholesterol. UpToDate Online. 2005; http://www.utdol.com/.
38. Sacks FM, Alaupovic P, Moye LA, et al. VLDL, apolipoproteins B, CIII, and E, and risk of recurrent coronary events in the Cholesterol and Recurrent Events (CARE) trial. Circulation. 2000; 102(16):1886-92.
39. Segrest JP. Lipids: classifications and understandings. 2005.
40. Seman LJ, DeLuca C, Jenner JL, et al. Lipoprotein(a)-cholesterol and coronary heart disease in the Framingham Heart Study. Clin Chem. 1999; 45(7):1039-46.
41. Sherrard B, Simpson H, Cameron J, Wahi S, Jennings G, Dart A. LDL particle size in subjects with previously unsuspected coronary heart disease: relationship with other cardiovascular risk markers. Atherosclerosis. 1996; 126(2):277-87.
42. Sniderman AD, Williams K, Contois JH. (2011). A meta-analysis of low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B as markers of cardiovascular risk. Circ Cardiovasc Qual Outcomes. May;4(3):337-45. Epub 2011 Apr 12.
43. St-Pierre AC, Cantin B, Dagenais GR, et al. Low-density lipoprotein subfractions and the long-term risk of ischemic heart disease in men: 13-year follow-up data from the Quebec Cardiovascular Study. Arterioscler Thromb Vasc Biol. 2005; 25(3):553-9.
44. Sweetnam PM, Bolton CH, Yarnell JW, et al. Associations of the HDL2 and HDL3 cholesterol subfractions with the development of ischemic heart disease in British men. The Caerphilly and Speedwell Collaborative Heart Disease Studies. Circulation. 1994; 90(2):769-74.

Select Health

Vertical Autoprofiling (VAP) Cholestrol Test, continued

45. Tzou WS, Douglas PS, Srinivasan SR, Chen W, Berenson G, Stein JH. Advanced lipoprotein testing does not improve identification of subclinical atherosclerosis in young adults: the Bogalusa Heart Study. Ann Intern Med. 2005; 142(9):742-50.
46. Wilson W, Culleton M. Overview of the risk factors for cardiovascular disease. UpToDate Online. 2005; http://www.utdol.com.
47. Yu S, Yarnell JW, Sweetnam P, Bolton CH. High density lipoprotein subfractions and the risk of coronary heart disease: 9-years follow-up in the Caerphilly Study. Atherosclerosis. 2003; 166(2):331-8.

Disclaimer
This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Select Health

MEDICAL POLICY

WEARABLE CARDIOVERTER-DEFIBRILLATOR

Policy # 406

Implementation Date: 8/1/08

Review Dates:6/11/09, 9/15/11, 11/29/12, 2/20/14, 3/19/15, 2/16/17, 2/15/18, 2/5/19, 2/17/20, 12/23/20, 4/18/22, 4/12/23, 2/15/24, 3/23/25

Revision Dates: 6/17/10, 2/29/16, 2/15/18, 3/2/22

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

The term "sudden cardiac death" (SCD) is used to describe cardiac arrest with cessation of cardiac function, whether resuscitation or spontaneous reversion occurs. This definition of SCD is misleading because not all affected individuals die, and the use of SCD in this sense has been challenged.

The implantable cardioverter defibrillator (ICD) is considered a cornerstone of modern cardiology practice for reducing the incidence of sudden cardiac death related to ventricular fibrillation (VF) or ventricular tachycardia (VT). However, several factors limit the prophylactic implantation of the ICD, mainly the inability of invasive and noninvasive laboratory investigations to predict sudden death accurately in a patient population bearing the risk of dying suddenly. As a result, a substantial portion of the at-risk population does not receive adequate preventive therapy and others with lower risk will have the ICD implanted. A potential solution to this problem could be the use of an external, wearable cardioverter defibrillator (vest) that has defibrillation features like those of the ICD (i.e., no operator required to defibrillate), providing protection to the patient until it is determined that the implantation of the ICD is warranted, or another strategy pursued (e.g., heart transplant). A device used a "bridge" to ICD implantation must be capable of reliably terminating episodes of VF/VT, have a highly sensitive and specific algorithm for the detection of VT/VF, and be user-friendly, thereby, ensuring patient compliance.

The vest-like device consists of a chest garment with electrode belt, monitor/defibrillator, patient base station, and physician programming console. The belt is strapped under the heart directly onto the patient's skin to continuously sense electrical activity. If a life-threatening arrhythmia is detected and no response to device alarms is noted, the patient is presumed to be unconscious, and the device delivers a shock to restore normal rhythm. Additional treatment may be delivered if the initial shock is not effective. The device weighs approximately 3 pounds. It is worn continuously, but it is not waterproof. For proper use, patients are trained to connect the device to an external modem and send data over the phone to the physician's computer for medical evaluation.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers wearable cardioverter-defibrillators when either of the following criteria are met (A or B).

A. Wearable cardioverter-defibrillators will be approved If recommended by Intermountain Health Cardiovascular Clinical Program,

Wearable Cardioverter-Defibrillator, continued

OR

B. For all other clinicians, wearable cardioverter-defibrillators will be approved when at least one of the following criteria are met (1–4):

Criteria for coverage include:

1. The patient is at high risk for sudden cardiac death and meets criteria for implantable cardioverter defibrillator (ICD) placement, but is not currently a suitable candidate for ICD placement because of one of the following:
   - Awaiting heart transplantation
   - Awaiting ICD reimplantation following infection-related removal of ICD
   - Systemic infectious process or other temporary medical condition precludes implantation
   - ICD lead malfunction with anticipated lead revision

   OR
2. As a bridge to ICD risk stratification and possible implantation for patients:
   - Had ventricular tachycardia (VT) or ventricular fibrillation (VF) within 48 hours of a myocardial infarction (MI), **or**
   - The first 40 days after myocardial infarction (MI) in patients with an ejection fraction (EF) < 35%, **or**
   - Within 3 months of CABG or PCI with ejection fraction (EF) < 35%, **or**
   - With non-ischemic cardiomyopathy and EF of < 35%

   OR
3. Newly diagnosed non-ischemic/idiopathic dilated cardiomyopathy (EF ≤ 35) with ICD implantation deferred to titrate therapy with at least one of the following risk markers as listed below:
   - Left or right bundle branch block
   - Marked impairment or left ventricular function (EF < 20)
   - Left ventricular end-diastolic dimension < 7.0 cm
   - Consistent right ventricular dysfunction
   - Non-sustained ventricular arrythmias
   - No obvious reversable causes (e.g., ETOH on thyroid induced cardiomyopathy)

   OR
4. Recent myocardial infarction (< 40 days) with severe left ventricular dysfunction (EF < 35) and additional markers of early mortality risk with at least one additional marker of risk:
   - Left or right bundle branch block
   - History of chronic ischemic heart failure (EF ≤ 40) with severe left ventricular dysfunction
   - Sustained ventricular tachycardia induced during electrophysiology study
   - Frequent premature ventricular ectopy and/or non-sustained ventricular tachycardia during telemetry monitoring
   - T wave alternans
   - Late potentials on signal average ECOG

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage,

Wearable Cardioverter-Defibrillator, continued

please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

The U.S. Food and Drug Administration (FDA) approved the Lifecor Wearable Cardioverter Defibrillator 2000 system via 510(k) approval in December 2001, based on clinical data submitted to the FDA by the manufacturer, which has subsequently been published in the peer-reviewed literature, and referred to as the BIROAD and WEARIT studies. The trials consisted of prospective, non-randomized studies, which compared the outcomes of the WCD with historical controls of patients suffering sudden cardiac arrest, who called 911 emergency services. While this study demonstrated that the WCD could detect arrhythmias and appropriately deliver a counter shock, its long-term efficacy will depend on patient compliance, and from a practical perspective, the WCD cannot be continuously worn. For example, the BIROAD and WEARIT studies included 289 patients; there were 12 deaths reported, and 50% occurred in patients either not wearing the device or wearing it inappropriately. Additionally, 68 of the 289 patients discontinued wearing the device due to comfort issues or adverse reactions. Therefore, an implantable cardiac defibrillator (ICD) is considered the gold standard, and, as such, a WCD would be considered an alternative to an ICD only in the small subset of patients who have co-morbidities or other contraindications for an ICD. Patients with an infected ICD requiring removal may benefit from a WCD worn during the limited interim period until an ICD can be re-implanted. Additionally, a small subset of patients awaiting heart transplantation may be considered at high risk for arrhythmia but are not candidates for an ICD due to co-morbidities. A WCD may be considered an alternative to an ICD in these patients while they are on the heart transplant waiting list.

There has been interest in offering WCDs to patients in the immediate post myocardial infarction (MI) period, when patients are considered at high risk of arrhythmia. However, the DINAMIT trial demonstrated that an ICD is not indicated during this period, thus, the WCD cannot be considered an alternative to an ICD in this setting. The DINAMIT trial randomized 674 patients to receive either an ICD or no ICD within 40 days of a myocardial infarction. All patients had reduced ejection fractions (ejection fraction ≤ 35%) and impaired cardiac autonomic function. There was no difference in overall mortality between the two groups. While the nonrandomized BIROAD study investigated patients treated with a WCD in the immediate post MI period, the results of the large randomized DINAMIT study provide a higher level of evidence, which may be extrapolated to WCD.

A 2009 technology assessment published by the California Technology Assessment Forum (CTAF), concluded that the use of a wearable cardioverter defibrillator (WCD) for patients at risk for sudden cardiac arrest and who are not candidates for or refuse an ICD does not meet the CTAF criteria. The assessment included uncontrolled case series by Auricchio (1998, n = 15), and Reek (2003, n = 12) that evaluated the ability of the device to detect and terminate tachyarrhythmias induced in the controlled setting of the electrophysiology laboratory. The author concluded that the limited scientific evidence, consisting of one pivotal trial with a precursor device and a small number of events, does not permit conclusions regarding the effectiveness of the WCD regarding health outcomes. A multicenter cohort study evaluating the impact of the WCD on mortality and quality of life in patients who meet criteria for, but are unable or unwilling to have an ICD, is needed before definitive conclusions can be made regarding safety and effectiveness. For patients who do not meet criteria for an ICD, but who are considered to be at increased risk of SCD (e.g., post-acute MI with reduced EF), a randomized controlled trial with mortality data is recommended before the safety and efficacy of the device can be evaluated for use in clinical practice.

Wearable Cardioverter-Defibrillator, continued

Billing/Coding Information

Covered: For the conditions outlined above

CPT CODES

93292	Interrogation device evaluation (in person) with analysis, review and report by a physician or other qualified health care professional, includes connection, recording and disconnection per patient encounter; wearable defibrillator system
93745	Initial set-up and programming by a physician of wearable cardioverter-defibrillator, includes initial programming of system, establishing baseline electronic ECG, transmission of data to data repository, patient instruction in wearing system and patient reporting of problems or events

HCPCS CODES

K0606	Automatic external defibrillator, with integrated electrocardiogram analysis, garment type
K0607	Replacement battery for automated external defibrillator, garment type only, each
K0608	Replacement garment for use with automated external defibrillator, each
K0609	Replacement electrodes for use with automated external defibrillator, garment type only, each

Key References

1. Al-Khatib, S. M., W. G. Stevenson, M. J. Ackerman, W. J. Bryant, D. J. Callans, A. B. Curtis, B. J. Deal, T. Dickfeld, M. E. Field, G. C. Fonarow, A. M. Gillis, M. A. Hlatky, C. B. Granger, S. C. Hammill, J. A. Joglar, G. N. Kay, D. D. Matlock, R. J. Myerburg and R. L. Page (2017). "2017 AHA/ACC/HRS Guideline for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society." Heart Rhythm.
2. Auricchio A, Klein H, Geller CJ, et al. (1998). Clinical efficacy of the wearable cardioverter defibrillator in acutely terminating episodes of ventricular fibrillation. Am J Cardiol. 1998; 81(10):1253-1256.
3. Beauregard LA. (2004). Personal security: clinical applications of the wearable defibrillator. Pacing Clin Electrophysiol. 27(1):1-3.
4. Bigger JT. (1997). Prophylactic use of implanted cardiac defibrillators in patients at high risk for ventricular arrhythmias after coronary artery bypass surgery. The CABG-PATCH Trial Investigators. N Engl J Med. 337(22):1569-1575.
5. Boehmer JP. (2003). Device therapy for heart failure. Am J Cardiol. 91(suppl):53D-59D.
6. Feldman AM, Klein H, Tchou P, et al. (2004). Use of a wearable defibrillator in terminating tachyarrhythmias in patients at high risk for sudden death: Results of the WEARIT/BIROAD. Pacing Clin Electrophysiol. 27(1):4-9.
7. Feldman MD. (2009). Wearable cardioverter defibrillator for patients at risk for sudden cardiac arrest. California Technology Assessment Forum (CTAF). March 11. Available: http://www.ctaf.org/content/assessment/detail/987. Date Accessed: June 29, 2010.
8. Hohnloser SH, Kuck KH, Dorian P, et al. (2004). Prophylactic use of an implantable cardioverter-defibrillator after acute myocardial infarction. N Engl J Med. 351(24):2481-2488.
9. Podrid, PJ, Arnsdorf, MF, Cheng, J, et al. (2008). Acute therapy of sudden cardiac arrest. UpToDate. Available: http://www.utdol.com/online/content/topic.do?topicKey=carrhyth/49765&selectedTitle=2~150&source=search_result. Accessed July 21, 2008.
10. Reek S, Geller JC, Meltendorf U, et al. (2003). Clinical efficacy of a wearable defibrillator in acutely terminating episodes of ventricular fibrillation using biphasic shocks. Pacing Clin Electrophysiol. 26(10):2016-2022.
11. Reek S, Meltendorf U, Geller JC, et al. (2002). The wearable cardioverter defibrillator (WCD) for the prevention of sudden cardiac death - a single center experience. Z Kardiol. 91(12):1044-1052 (abstract available in English; article in German).

Disclaimer

This document is for informational purposes only and should not be relied on in the diagnosis and care of individual patients. Medical and Coding/Reimbursement policies do not constitute medical advice, plan preauthorization, certification, an explanation of benefits, or a contract. Members should consult with appropriate healthcare providers to obtain needed medical advice, care, and treatment. Benefits and eligibility are determined before medical guidelines and payment guidelines are applied. Benefits are determined by the member's individual benefit plan that is in effect at the time services are rendered.

The codes for treatments and procedures applicable to this policy are included for informational purposes. Inclusion or exclusion of a procedure, diagnosis or device code(s) does not constitute or imply member coverage or provider reimbursement policy. Please refer to the member's contract benefits in effect at the time of service to determine coverage or non-coverage of these services as it applies to an individual member.

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.

Wearable Cardioverter-Defibrillator, continued

Select Health

MEDICAL POLICY

WIRELESS CARDIAC MONITORING (E.G., CARDIOMEMS)

Policy # 642

Implementation Date:9/28/20
Review Dates: 1/20/22, 2/16/23, 2/15/24, 2/10/25
Revision Dates:

Disclaimer:
1. Policies are subject to change without notice.
2. Policies outline coverage determinations for Select Health Commercial, Select Health Medicare (CMS), and Select Health Community Care (Medicaid) plans. Refer to the "Policy" section for more information.

Description

Heart failure (HF) is a chronic condition that develops over time due to circumstances that overwork and damage the heart. The primary causes include coronary heart disease, high blood pressure, and diabetes. HF is characterized by the inability of the heart to pump blood efficiently. HF is estimated to affect approximately 5.7 million people in the United States. Currently, there is no cure for HF. It can affect both children and adults, although, it most commonly occurs in adults 65 years of age or older.

The CardioMEMS (CM) System (Abbott) is a wireless implantable hemodynamic monitor (IHM), for use at home in patients with HF. The CM-IHM system includes an implantable pulmonary artery pressure (PAP) sensor, a transvenous catheter delivery system, a patient home-monitoring electronic system, and a secure Internet-accessible database that allows clinicians to access patient data. The CM-IHM system provides measurement of the systolic, diastolic, and mean PAP, intending to allow for adjustment of HF medical therapy based on pressure trends and specified pressure goals. Once implanted, the patient will take a wireless reading from their PAP sensor once a day from home. Pressure data from this daily reading is then transmitted to a secure website for the physician and clinical team to review and make any necessary adjustments to HF treatment.

COMMERCIAL PLAN POLICY AND CHIP (CHILDREN'S HEALTH INSURANCE PROGRAM)

Application of coverage criteria is dependent upon an individual's benefit coverage at the time of the request.

Select Health covers the CardioMEMS wireless cardiac monitor for heart failure patients, if either A or B are documented:

A. Recommended by Intermountain Health Cardiovascular Clinical Program, **OR**

B. All the following criteria are met:

1. Left-sided heart failure diagnosis (preserved or reduced ejection fraction) for at least 3 months
2. History of ≥ 1 heart failure hospitalizations/events in the past year
3. HFrEF (heart failure with reduced ejection fraction) patients must be on optimal heart failure medications (GDMT [guideline-directed medical therapy] at best-tolerated doses)
4. NYHA (New York Heart Association) class III heart failure symptoms

Wireless Cardiac Monitoring (e.g., Cardiomems), continued

5. The patient is an outpatient, and actively being monitored by a cardiologist specializing in heart failure with the capability of daily monitoring of the CardioMEMS data
6. The patient agrees to the patient training and home measurement schedule

Absolute Contraindications to CardioMEMS:

a. Non-adherence with medication use, and/or laboratory follow-up recommendations
b. The patient has refused CRT (cardiac resynchronization therapy)
c. Inability to take dual antiplatelet therapy or anticoagulation for one-month post-implantation
d. Patient cannot tolerate a right heart catheterization
e. Mechanical tricuspid or pulmonic valve
f. BMI > 35 with chest circumference (at axillary level) > 165cm

Relative Contraindications to CardioMEMS:

g. Patients with an active but treatable infection
h. Patients with a history of recurrent (> 1) pulmonary embolism or deep vein thrombosis
i. Patients with a GFR < 25 ml/min who are non-responsive to diuretic therapy.
j. Patients with ESRD on dialysis
k. Patients with congenital heart disease
l. Patients with known coagulation disorders
m. Patients with a hypersensitivity or allergy to aspirin, and/or clopidogrel
n. CRT implantation in the past 3 months (as these patients may clinically improve post-CRT)
o. Recent pacemaker, ICD, or CRT implantation (risk of lead dislodgement and would need to discuss timing with electrophysiology)
p. Tricuspid valve clip (would need to be discussed with structural heart team)

SELECT HEALTH MEDICARE (CMS)

Coverage is determined by the Centers for Medicare and Medicaid Services (CMS); if a coverage determination has not been adopted by CMS, and InterQual criteria are not available, the Select Health Commercial policy applies. For the most up-to-date Medicare policies and coverage, please visit their search website http://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx?from2=search1.asp& or the manual website

SELECT HEALTH COMMUNITY CARE (MEDICAID)

Select Health Community Care policies typically align with State of Utah Medicaid policy, including use of InterQual. There may be situations where NCD/LCD criteria or Select Health commercial policies are used. For the most up-to-date Medicaid policies and coverage, please visit their website http://health.utah.gov/medicaid/manuals/directory.php or the Utah Medicaid code Look-Up tool

Summary of Medical Information

CardioMEMS pulmonary artery monitoring device has been approved by the US Food and Drug Administration to monitor pulmonary artery pressure and heart rate in patients with NYHA class III HF who have been hospitalized during the previous year. Further study is needed to determine the efficacy and safety of this device. The 2016 ESC HF (European Society of Cardiology Heart Failure) guidelines included only a very weak recommendation stating that this device may be considered in symptomatic patients with HF with previous HF hospitalization.

Wireless Cardiac Monitoring (e.g., Cardiomems), continued

The CardioMEMS Heart Sensor Allows Monitoring of Pressure to Improve Outcomes In NYHA Class III Heart Failure Patients (CHAMPION) randomized single-blind trial of 550 patients found that transmission of pulmonary artery pressure data from the device reduced HF-related hospitalizations at six months (31 versus 44 percent, HR 0.70, 95% CI 0.60−0.84). There was a 1.5 percent rate of device- or system-related complications. An exploratory subgroup analysis found that device-guided management reduced HF-related hospitalization in patients with preserved LVEF (left ventricular ejection fraction) (LVEF ≥ 40 percent or LVEF ≥ 50 percent), as well as in patients with LVEF < 40 percent. Another exploratory analysis found that device-guided management reduced respiratory hospitalization rates as well as HF hospitalization rates in the entire cohort, as well as in a subgroup of 187 patients with chronic obstructive pulmonary disease.

A later analysis reported sustained reduction in HF-related hospitalization in the device-guided management group compared with the control at 18-month average follow-up (46 versus 68 percent, HR 0.67, 95% CI 0.55 to 0.80). During a subsequent open access period (mean duration 13 months), pulmonary artery pressure information was made available to guide therapy in the former control group; the rate of admission was reduced compared with that in the control group during the randomized access period (36 versus 68 percent; HR 0.52, 95% CI 0.40 to 0.69). The rate of device- or system-related complications was 1 percent, and the rate of procedure-related adverse events was 1 percent.

However, the efficacy of the CardioMEMS device is uncertain given concerns raised about potential bias introduced in the conduct of the CHAMPION trial (including interaction between the trial sponsor and clinical investigators on certain treatment group subjects) and the analysis of data.

Billing/Coding Information

CPT CODES

33289	Transcatheter implantation of wireless pulmonary artery pressure sensor for long-term hemodynamic monitoring, including deployment and calibration of the sensor, right heart catheterization, selective pulmonary catheterization, radiological supervision and interpretation, and pulmonary artery angiography, when performed
93264	Remote monitoring of a wireless pulmonary artery pressure sensor for up to 30 days, including at least weekly downloads of pulmonary artery pressure recordings, interpretation(s), trend analysis, and report(s) by a physician or other qualified health care

HCPCS CODES

C2624	Implantable wireless pulmonary artery pressure sensor with delivery catheter, including all system components

Key References

1. Colucci, W. S. (2019). Investigational and emerging strategies of heart failure. *UpToDate*. Retrieved from: https://www.uptodate.com/contents/investigational-and-emerging-strategies-for-management-of-heart-failure/print?search=cardiomems&source=search_result&selectedTitle=2~2&usage_type=default&display_rank=2
2. Hayes Brief. (2019). CardioMEMS Implantable Hemodynamic Monitor (Abbott) for Managing Patients with Heart Failure.

Select Health

Wireless Cardiac Monitoring (e.g., Cardiomems), continued

Select Health® makes no representations and accepts no liability with respect to the content of any external information cited or relied upon in this policy. Select Health updates its Coverage Policies regularly, and reserves the right to amend these policies without notice to healthcare providers or Select Health members.

Members may contact Customer Service at the phone number listed on their member identification card to discuss their benefits more specifically. Providers with questions about this Coverage Policy may call Select Health Provider Relations at (801) 442-3692.