



Ultrasonographic Measurement of Carotid Artery Intima-Media Thickness as an Assessment of Atherosclerosis

Effective: April 1, 2026

Next Review: December 2026

Last Review: February 2026

IMPORTANT REMINDER

Medical Policies are developed to provide guidance for members and providers regarding coverage in accordance with contract terms. Benefit determinations are based in all cases on the applicable contract language. To the extent there may be any conflict between the Medical Policy and contract language, the contract language takes precedence.

PLEASE NOTE: Contracts exclude from coverage, among other things, services or procedures that are considered investigational or cosmetic. Providers may bill members for services or procedures that are considered investigational or cosmetic. Providers are encouraged to inform members before rendering such services that the members are likely to be financially responsible for the cost of these services.

DESCRIPTION

The carotid artery intima-media thickness (CIMT) is used as a marker of subclinical atherosclerosis and its measurement has been proposed as method to screen for cardiovascular risk.

MEDICAL POLICY CRITERIA

Ultrasonographic measurement of the carotid artery intima-media thickness is considered **investigational** for screening, diagnosis, and management of atherosclerotic disease.

NOTE: A summary of the supporting rationale for the policy criteria is at the end of the policy.

CROSS REFERENCES

1. [Computed Tomography to Detect Coronary Artery Calcifications](#), Radiology, Policy No. 06

BACKGROUND

Coronary heart disease accounts for 27% of all deaths in the United States.^[1] Established

major risk factors for coronary heart disease (CHD) have been identified by the National Cholesterol Education Program (NCEP) Expert Panel and include elevated serum levels of low-density lipoprotein (LDL) cholesterol and total cholesterol, and low serum levels of high-density lipoprotein (HDL) cholesterol. Other risk factors include a history of cigarette smoking, hypertension, family history of premature CHD, and age. Pathology studies have demonstrated that levels of traditional risk factors are associated with the extent and severity of atherosclerosis. However, at every level of risk factor exposure, there is substantial variation in the amount of atherosclerosis, presumably related to genetic susceptibility and the influence of other risk factors. Therefore, there has been interest in identifying a technique that can improve the ability to diagnose those at risk of developing CHD, as well as measure disease progression, particularly for those at intermediate risk.

Ultrasonographic measurement of carotid intima-medial (also called intimal-medial or intima-media) thickness (CIMT) refers to the use of B-mode ultrasound to determine the thickness of the two innermost layers of the carotid artery wall, the intima and the media. Ultrasonographic measurement of CIMT has been investigated as a proxy for progression of atherosclerosis and is proposed for use in identifying and monitoring subclinical CHD.

REGULATORY STATUS

In February 2003, SonoCalc® (SonoMetric Health, LLC) was cleared for marketing by the FDA through the 510(k) process. The FDA determined that this software was substantially equivalent to image display products from existing ultrasound systems. Subsequently, several other devices have been approved through the 510(k) process.

Note: this policy does not address carotid artery ultrasound for the evaluation of a cerebrovascular condition suspected on the basis of abnormal signs or symptoms, which is considered a standard of care.

EVIDENCE SUMMARY

Currently, screening and monitoring for coronary artery disease in clinically asymptomatic individuals is achieved through administration of standard risk assessment measures (including family history and non-invasive testing). Measurement of carotid intima-medial (or intimal-media) thickness (CIMT) is primarily meant to assess risk for future disease, and therefore can be evaluated as a prognostic measure. Within this context, assessment of the proposed use of ultrasonographic measurement of carotid intima-media thickness (CIMT) must fulfill three parameters:

- 1) Establish technical feasibility, typically assessed with two types of studies, those that compare test measurements with a gold standard and those that compare results taken with the same device on different occasions (test-retest). Normally conducted in the pre-clinical setting, the focus of this parameter is on test reproducibility and establishment of the test protocol.
- 2) Demonstrate diagnostic performance (sensitivity, specificity, positive and negative predictive values) of the test compared with the gold standard.
- 3) Evaluate clinical outcomes based on the performance of the test versus the standard of care. While in some cases, new diagnostic tests can be adequately evaluated using technical and diagnostic performance, when a test identifies a new or different group of

patients with a disease, randomized trials are needed to demonstrate the impact of the test on net health outcomes.

DIAGNOSTIC UTILITY (ANALYTICAL AND CLINICAL VALIDITY)

The current literature consists of several systematic reviews, meta-analyses, and case series related to technical feasibility, and large longitudinal cohort studies conducted in the research setting.

Systematic Reviews

Tschiderer (2023) published a meta-analysis of 20 studies from the Proof-ATHERO (Prospective Studies of Atherosclerosis) consortium (n=21,494 participants) to investigate the association between CIMT and carotid plaque development.^[2] Mean participant age was 56 years, and mean baseline CIMT was 0.71 mm at the beginning of the studies. Median follow-up was 5.9 years. 8,278 participants developed first-ever carotid plaques. The meta-analysis suggested an association between CCA-IMT and long-term risk of developing a first-ever carotid plaque. The age-, sex-, and trial arm–adjusted odds ratio [OR] for carotid plaque per higher baseline CIMT was 1.40 (95% confidence interval [CI], 1.31 to 1.50; $I^2=63.9\%$). When further adjusted for ethnicity, smoking, diabetes, body mass index, systolic blood pressure, low- and high-density lipoprotein cholesterol, and lipid-lowering and anti-hypertensive medication, the OR carotid plaques associated with CIMT was 1.34 (95% CI, 1.24 to 1.45; $I^2=59.4\%$; n=14 studies; 16 297 participants; 6381 incident plaques). This meta-analysis is limited by heterogeneity in imaging methods, definitions of measured CIMT and carotid plaques, and uncertainty in the time point of plaque development across studies.

Wang (2022) performed a systematic review of 26 studies available up until October 30, 2021, with 1,370 participants.^[3] Compared with control participants, those who engaged in exercise showed a decline in CIMT. An exercise duration of greater than six months was associated with a 0.02 mm reduction in CIMT.

Van Bergen (2022) conducted a systematic review for 42 studies (6,143 participants) addressing CIMT in Familial hypercholesterolemia (FH) patients and controls.^[4] They concluded that increase was smaller in treated vs untreated FH patients, when compared to controls. The authors suggest that more robust earlier treatment initiation and achieving treatment targets could be beneficial to reduce cardiovascular risk in patients with FH.

Three systematic reviews^[5-7] with meta-analyses^[8-12] analyzed the ability of CIMT measurement to identify coronary artery disease in asymptomatic patients and predict first-time myocardial infarction (MI) or first-time stroke. The inclusion criteria for the studies included in these reviews varied. However, the results consistently reported that, while CIMT is a predictor of cardiovascular risk, the addition of CIMT measurement did not significantly improve risk prediction over conventional cardiovascular risk factors. In addition, most of the reviewed studies were conducted in the research setting and therefore cannot be used to draw conclusions on the applicability of CIMT measurement in the clinical setting for asymptomatic patients at large.

Randomized Controlled Trials

There are no RCTs evaluating the analytical or clinical validity of ultrasonographic measurement of CIMT.

Nonrandomized Studies

Using data from the Atherosclerosis Risk in Communities (ARIC) Study, Caughey (2018) examined the link between common carotid artery intima-media thickness (CCA-IMT) and silent brain infarctions.^[13] Stroke-free participants (641 black and 702 white) underwent MRI brain imaging and carotid ultrasound. Silent brain infarctions, defined as asymptomatic brain lesions greater than or equal to three mm, were identified in 156 patients. These were associated with elevated CCA-IMT in black patients, but not white patients (prevalence ratio [PR] 1.60, 95% confidence interval [CI] 1.02 to 2.51, and PR 0.85, 95% CI 0.35 to 2.04, respectively).

Geisel (2017) reported on a prospective cohort study of 3,108 patients without cardiovascular disease on entrance to the study.^[14] All patients were evaluated by CIMT, coronary artery calcification, and ankle-brachial index. During a mean follow-up time of 10 years, 223 individuals suffered a major cardiovascular event (coronary event, stroke, CV death). All three methods served to help predict adverse cardiovascular event. While CIMT was found to be higher in those who experienced an adverse cardiovascular event than those who did not (0.76 ± 0.17 vs 0.69 ± 0.15), it did not lead to a significant improvement in predicting cardiac risk for patients with an intermediate Framingham Risk Score.

Villines (2017) published a prospective cohort study of 3,801 African American patients who were free of cardiovascular disease at baseline.^[15] Over a median follow-up time of nine years, there were 171 new cases of cardiovascular disease and 339 deaths. The incidence of cardiovascular events was related to changes in CIMT, and participants in the highest CIMT quartile had the largest crude incident rates of cardiovascular disease for both men and women. However, risk reclassification improved only slightly when adding CIMT to a model which required only traditional risk factors for cardiovascular disease.

A prospective cohort study by Moreo (2015) assessed the value of adding CIMT to other potentially predictive parameters to enhance the prediction of coronary artery disease (CAD) in 247 patients with CAD and 184 patients without CAD.^[16] The predictive parameters assessed in CAD vs non-CAD patients included blood pressure, CIMT, carotid pulse wave velocity (cPWV), semiquantitative score of cardiac calcifications, global myocardial longitudinal strain (GLS), and rest Doppler flow velocity on the left anterior descending (LAD) coronary artery. The patients with CAD had significantly higher blood pressure, cIMT, cPWV, score of calcium, and LAD velocity than non-CAD patients. All ultrasound parameters significantly predicted CAD. Stepwise logistic regression concluded that the only combined predictors of CAD were score of calcium, cIMT, and LAD velocity.

The BioImage study enrolled 5,808 asymptomatic individuals from the United States to compare three-dimensional carotid ultrasound with CT scans of the coronary arteries in their ability predict atherothrombotic events.^[17] Carotid ultrasound was used to calculate carotid plaque burden (cPB), and CT scans were used to evaluate coronary artery calcification (CAC). After a median of 2.7 years of follow-up, both cPB and CAC were found to be independent predictors of major cardiovascular events, defined as cardiovascular death, MI and ischemic stroke, with hazard ratios of 2.36 (95% CI 1.13 to 4.92) and 2.99 (95% CI 1.48 to 6.05), respectively for individuals in the highest tertile. Both cPB and CAC score led to significant net reclassification compared with conventional risk factors, with net reclassification indices of 0.23 and 0.25, respectively.

More recent studies reported that including carotid plaques in CIMT increased the predictive value of cardiovascular risk over CIMT assessed only in plaque-free sites.^[18-21] However, the

meta-analysis by Lorenz found no difference in the main results between studies that included CIMT with carotid plaque and plaque-free CIMT.^[8] The systematic review by Peters found adding carotid plaque to the traditional CIMT model increased the c-statistic from 0.01 to 0.06.^[5]

An observational study among 320 Spanish patients compared CIMT measurements with traditional risk assessment measures (age, hypertension and systolic blood pressure).^[22] Although CHD risk was reclassified for 18% of participants based on CIMT, implications for clinical management and effect on health outcomes were not reported.

In a community-based cohort in Taiwan, CIMT and extracranial carotid artery plaque score were measured in 1,398 participants.^[23] In this study, the five-year individual change in CIMT was not associated with cardiovascular events. The development of new plaques was associated with increased risk, but this was attenuated after adjusting for cardiovascular risk factors.

A 2016 study evaluated the relationship between CIMT and cerebral microbleed (CMB) in 1,243 participants from the Framingham Offspring Study. Participants had carotid ultrasound information available from two exam periods, 1995-1998 and 2005-2008, prior to brain imaging with MRI.^[24] Baseline carotid stenosis, baseline intima-media thickness, and CIMT progression at both internal and common carotid locations were tested for associations with CMB. While carotid stenosis greater than or equal to 25% was associated with the presence of CMB (odds ratio [OR] 2.20, 95% CI 1.10 to 4.40), baseline CIMT was not associated with CMB. Additionally, progression of common carotid intima-media thickness in individuals on hypertension treatment was associated with a lower risk of CMB.

Polak (2014) reported 7.8 years follow-up of 6,255 individuals free of CAD, stroke, and atrial fibrillation at baseline.^[25] Subjects were from a multiethnic community based-cohort with mean age of 62.2 years at baseline. The aim of the study was to determine whether CIMT and common carotid artery diameter were predictors of ischemic stroke. There were 115 first-time ischemic strokes during the follow-up period. The authors reported that common carotid artery diameter was independently associated with first-time incident ischemic stroke but CIMT was not.

In the Atherosclerosis Risk in Communities (ARIC) study, a large observation study conducted in the research setting, the authors evaluated risk factors associated with increased CIMT in 15,800 subjects.^[26] CIMT had a graded relationship with increasing quartiles of plasma total cholesterol, LDL cholesterol, and triglycerides. CIMT was also correlated with the incidence of coronary heart disease (CHD) in a subgroup of patients enrolled in the trial after 4 to 7 years of follow-up.^[27] The researchers defined and compared extreme carotid IMT (0.1 mm or greater) to non-extreme IMT (less than 0.1 mm) and found a relationship between CIMT and CHD events. Nevertheless, this definition of extreme IMT has yet to be tested in the clinical setting.

A 2014 retrospective analysis of 184 children and adolescents reported excellent reproducibility of CIMT measurements when the same methodology was applied.^[28] However, there was significant variation throughout the cardiac cycle. The authors concluded that standardized CIMT measurements that use electrocardiographic timing are needed for this patient population.

Technical feasibility was addressed in a 2010 study on inter-reader differences in measuring CIMT.^[29] Among five readers with six months to six years of experience reading CIMT images,

significant differences were seen in the measurement of 26 CIMT images, whose final measurements ranged from 0.57 to 0.78 mm. This range corresponds to as much as a 21-year vascular age discrepancy in the same image, a high degree of error. The authors suggest improved training of CIMT readers, or the development of an IMT edge-reader before this technology is adopted in the clinical setting.

Several other studies have used CIMT measurements as outcome measures.^[30-41] Due to limitations such as the lack of a shared diagnostic CIMT measurement protocol, lack of head-to-head comparisons with gold standard diagnostic tests for CHD, and unknown impact of CIMT measurement on clinical decision-making and primary health outcomes, these studies do not add to the understanding of the net effect of this testing on the diagnosis and treatment of CHD.

CLINICAL UTILITY

Randomized Controlled Trials

There are no RCTs investigating the clinical utility of measuring CIMT for cardiac risk stratification.

Nonrandomized Study

In a study by Johnson (2011), 355 patients, aged 40 years with one or more cardiovascular disease risk factor, received carotid ultrasound screenings to prospectively determine whether abnormal results would change physician and patient behaviors.^[42] Results were considered abnormal in 266 patients (CIMT greater than the 75th percentile or the presence of carotid plaque). Self-reported questionnaires were completed before the carotid ultrasound, immediately after the ultrasound and 30 days later to determine behavioral changes. Physician behavior in prescribing aspirin and cholesterol medication changed significantly ($p < 0.001$ and $p < 0.001$, respectively) after identification of abnormal carotid ultrasound results. Abnormal ultrasound results predicted reduced dietary sodium (OR 1.45, $p = 0.002$) and increased fiber intake (OR 1.55, $p = 0.022$) in patients but no other significant changes. Health outcomes were not evaluated in this study and the short-term follow-up limits interpretation of results.

SECTION SUMMARY

Evidence from large, prospective cohort studies has established that CIMT is an independent risk factor for cardiovascular disease. The evidence on reclassification of cardiovascular risk offers a potential indirect chain of evidence to improve outcomes. If CIMT were able to reclassify patients into risk categories that have different treatment approaches, then clinical management changes may occur that lead to improved outcomes. However, there is no direct evidence on the clinical utility of measuring CIMT for cardiac risk stratification, and systematic reviews have concluded that the ability of CIMT to reclassify patients into clinically relevant categories is modest and may not be clinically important. The uncertainty around the ability to reclassify patients into clinically relevant categories with CIMT limits the potential for CIMT to improve health outcomes.

PRACTICE GUIDELINE SUMMARY

**AMERICAN COLLEGE OF CARDIOLOGY AND THE AMERICAN HEART ASSOCIATION
(ACC/AHA)**

The 2013 update of the ACCF/AHA evidence-based clinical practice guideline for the assessment of cardiovascular risk recommends against CIMT measurement in asymptomatic patients (Class III recommendation; Level of evidence B, defined as a recommendation that the procedure is not useful/effective and may be harmful based on evidence from a single RCT or nonrandomized studies).^[43] This is a reversal of the 2010 version of this guideline^[44] which indicated that CIMT measurement might be reasonable in certain patients. This change was based on new evidence reviewed during the update.

AMERICAN ASSOCIATION OF CLINICAL ENDOCRINOLOGISTS AND THE AMERICAN COLLEGE OF ENDOCRINOLOGY

The American Association of Clinical Endocrinologists and American College of Endocrinology published 2017 guidelines stating that CIMT could be applied as a risk stratification tool in determining the need for more aggressive preventive strategies against cardiovascular disease (Grade B; BEL 2)—but that it should not be performed routinely.^[45]

AMERICAN COLLEGE OF PREVENTIVE MEDICINE (ACPM)

In a 2011 position statement, the ACPM recommends CHD risk assessment using the Framingham Risk Score to guide risk-based therapy. ACPM does not recommend routine screening of the general adult population using electrocardiogram, exercise-stress testing, computed tomography scanning, ankle-brachial index, carotid intima medial thickness, or emerging risk factors, including high-sensitivity C-reactive protein (hs-CRP).^[46]

U.S. PREVENTIVE SERVICES TASK FORCE (USPSTF)

Based on the systematic review^[7] conducted for the USPSTF, the Task Force “concludes that the current evidence is insufficient to assess the balance of benefits and harms of using...[CIMT]...to screen asymptomatic men and women with no history of CHD to prevent CHD events.”^[47] The USPSTF identifies the following research need: “The predictive value...of carotid IMT...should be examined in conjunction with traditional Framingham risk factors for predicting CHD events and death.”

In 2018, the USPSTF published a recommendation statement on using nontraditional risk factors to assess the risk of CVD; CIMT was not mentioned in this recommendation.^[48]

SUMMARY

There is not enough research to show that the measurement of carotid artery intima-media thickness (CIMT) provides information that can improve health outcomes for individuals at risk for cardiovascular disease. There are no clinical guidelines based on research that recommend CIMT measurement for people with any condition. Therefore, measurement of CIMT for screening, diagnosis, and management of cardiovascular disease is considered investigational.

REFERENCES

1. Minino AM, Heron MP, Murphy SL, et al. Deaths: final data for 2004. *Natl Vital Stat Rep*. 2007;55(19):1-119. PMID: 17867520

2. Tschiderer L, Seekircher L, Izzo R, et al. Association of Intima-Media Thickness Measured at the Common Carotid Artery With Incident Carotid Plaque: Individual Participant Data Meta-Analysis of 20 Prospective Studies. *Journal of the American Heart Association*. 2023;12(12):e027657. PMID: 37301757
3. Wang Y, Wu H, Sun J, et al. Effect of Exercise on Carotid Artery Intima-Media Thickness in Adults: A Systematic Review and Meta-Analysis. *J Phys Act Health*. 2022;19(12):855-67. PMID: 36257606
4. van Bergen En Henegouwen K, Hutten BA, Luirink IK, et al. Intima-media thickness in treated and untreated patients with and without familial hypercholesterolemia: A systematic review and meta-analysis. *J Clin Lipidol*. 2022;16(2):128-42. PMID: 35184975
5. Peters SA, den Ruijter HM, Bots ML, et al. Improvements in risk stratification for the occurrence of cardiovascular disease by imaging subclinical atherosclerosis: a systematic review. *Heart*. 2012;98(3):177-84. PMID: 22095617
6. Mookadam F, Moustafa SE, Lester SJ, et al. Subclinical atherosclerosis: evolving role of carotid intima-media thickness. *Prev Cardiol*. 2010;13(4):186-97. PMID: 20860643
7. Helfand M, Buckley DI, Freeman M, et al. Emerging risk factors for coronary heart disease: a summary of systematic reviews conducted for the U.S. Preventive Services Task Force. *Ann Intern Med*. 2009;151(7):496-507. PMID: 19805772
8. Lorenz MW, Polak JF, Kavousi M, et al. Carotid intima-media thickness progression to predict cardiovascular events in the general population (the PROG-IMT collaborative project): a meta-analysis of individual participant data. *Lancet*. 2012;379(9831):2053-62. PMID: 22541275
9. Den Ruijter HM, Peters SA, Anderson TJ, et al. Common carotid intima-media thickness measurements in cardiovascular risk prediction: a meta-analysis. *JAMA*. 2012;308:796-803. PMID: 22910757
10. van den Oord SC, Sijbrands EJ, ten Kate GL, et al. Carotid intima-media thickness for cardiovascular risk assessment: systematic review and meta-analysis. *Atherosclerosis*. 2013;228(1):1-11. PMID: 23395523
11. Willeit P, Thompson SG, Agewall S, et al. Inflammatory markers and extent and progression of early atherosclerosis: Meta-analysis of individual-participant-data from 20 prospective studies of the PROG-IMT collaboration. *European journal of preventive cardiology*. 2014. PMID: 25416041
12. Bots ML, Groenewegen KA, Anderson TJ, et al. Common carotid intima-media thickness measurements do not improve cardiovascular risk prediction in individuals with elevated blood pressure: the USE-IMT collaboration. *Hypertension*. 2014;63:1173-81. PMID: 24614213
13. Caughey MC, Qiao Y, Windham BG, et al. Carotid Intima-Media Thickness and Silent Brain Infarctions in a Biracial Cohort: The Atherosclerosis Risk in Communities (ARIC) Study. *American journal of hypertension*. 2018;31(8):869-75. PMID: 29425278
14. Geisel MH, Bauer M, Hennig F, et al. Comparison of coronary artery calcification, carotid intima-media thickness and ankle-brachial index for predicting 10-year incident cardiovascular events in the general population. *European heart journal*. 2017;38(23):1815-22. PMID: 28379333
15. Villines TC, Hsu LL, Blackshear C, et al. Relation of Carotid Intima-Media Thickness to Cardiovascular Events in Black Americans (From the Jackson Heart Study). *Am J Cardiol*. 2017;120(9):1528-32. PMID: 28844515
16. Moreo A, Gaibazzi N, Faggiano P, et al. Multiparametric carotid and cardiac ultrasound compared with clinical risk scores for the prediction of angiographic coronary artery

- disease: a multicenter prospective study. *Journal of hypertension*. 2015;33(6):1291-300. PMID: 25715090
17. Baber U, Mehran R, Sartori S, et al. Prevalence, impact, and predictive value of detecting subclinical coronary and carotid atherosclerosis in asymptomatic adults: the BioImage study. *J Am Coll Cardiol*. 2015;65(11):1065-74. PMID: 25790876
 18. Plichart M, Celermajer DS, Zureik M, et al. Carotid intima-media thickness in plaque-free site, carotid plaques and coronary heart disease risk prediction in older adults. The Three-City Study. *Atherosclerosis*. 2011;219(2):917-24. PMID: 22005196
 19. Keo HH, Baumgartner I, Hirsch AT, et al. Carotid plaque and intima-media thickness and the incidence of ischemic events in patients with atherosclerotic vascular disease. *Vasc Med*. 2011;16(5):323-30. PMID: 21908682
 20. Nambi V, Chambless L, He M, et al. Common carotid artery intima-media thickness is as good as carotid intima-media thickness of all carotid artery segments in improving prediction of coronary heart disease risk in the Atherosclerosis Risk in Communities (ARIC) study. *European heart journal*. 2012;33(2):183-90. PMID: 21666250
 21. Xie W, Liang L, Zhao L, et al. Combination of carotid intima-media thickness and plaque for better predicting risk of ischaemic cardiovascular events. *Heart*. 2011;97(16):1326-31. PMID: 21653216
 22. Aguilar-Shea AL, Gallardo-Mayo C, Garrido-Elustondo S, et al. Carotid intima-media thickness as a screening tool in cardiovascular primary prevention. *Eur J Clin Invest*. 2011;41(5):521-6. PMID: 21155766
 23. Chen PC, Jeng JS, Hsu HC, et al. Carotid Atherosclerosis Progression and Risk of Cardiovascular Events in a Community in Taiwan. *Scientific reports*. 2016;6:25733. PMID: 27169625
 24. Romero JR, Preis SR, Beiser A, et al. Carotid Atherosclerosis and Cerebral Microbleeds: The Framingham Heart Study. *Journal of the American Heart Association*. 2016;5(3):e002377. PMID: 26994127
 25. Polak JF, Sacco RL, Post WS, et al. Incident stroke is associated with common carotid artery diameter and not common carotid artery intima-media thickness. *Stroke*. 2014;45:1442-6. PMID: 24643408
 26. Dobs AS, Nieto FJ, Szklo M, et al. Risk factors for popliteal and carotid wall thicknesses in the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Epidemiol*. 1999;150(10):1055-67. PMID: 10568620
 27. Chambless LE, Heiss G, Folsom AR, et al. Association of coronary heart disease incidence with carotid arterial wall thickness and major risk factors: the Atherosclerosis Risk in Communities (ARIC) Study, 1987-1993. *Am J Epidemiol*. 1997;146(6):483-94. PMID: 9290509
 28. Selamet Tierney ES, Gauvreau K, Jaff MR, et al. Carotid Artery Intima-Media Thickness Measurements in the Youth: Reproducibility and Technical Considerations. *J Am Soc Echocardiogr*. 2014. PMID: 25459501
 29. Polak JF, Funk LC, O'Leary DH. Inter-reader differences in common carotid artery intima-media thickness: implications for cardiovascular risk assessment and vascular age determination. *J Ultrasound Med*. 2011;30(7):915-20. PMID: 21705724
 30. van der Meer IM, Bots ML, Hofman A, et al. Predictive value of noninvasive measures of atherosclerosis for incident myocardial infarction: the Rotterdam Study. *Circulation*. 2004;109(9):1089-94. PMID: 14993130
 31. Paramsothy P, Knopp RH, Bertoni AG, et al. Association of combinations of lipid parameters with carotid intima-media thickness and coronary artery calcium in the

- MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol*. 2010;56(13):1034-41. PMID: 20846602
32. Blaha MJ, Rivera JJ, Budoff MJ, et al. Association between obesity, high-sensitivity C-reactive protein ≥ 2 mg/L, and subclinical atherosclerosis: implications of JUPITER from the Multi-Ethnic Study of Atherosclerosis. *Arterioscler Thromb Vasc Biol*. 2011;31(6):1430-8. PMID: 21474823
 33. Blankstein R, Budoff MJ, Shaw LJ, et al. Predictors of coronary heart disease events among asymptomatic persons with low low-density lipoprotein cholesterol MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol*. 2011;58(4):364-74. PMID: 21757113
 34. Camhi SM, Katzmarzyk PT, Broyles ST, et al. Subclinical atherosclerosis and metabolic risk: role of body mass index and waist circumference. *Metab Syndr Relat Disord*. 2011;9(2):119-25. PMID: 21133775
 35. Green D, Foiles N, Chan C, et al. An association between clotting factor VII and carotid intima-media thickness: the CARDIA study. *Stroke*. 2010;41(7):1417-22. PMID: 20466994
 36. Probstfield JL, Margitic SE, Byington RP, et al. Results of the primary outcome measure and clinical events from the Asymptomatic Carotid Artery Progression Study. *Am J Cardiol*. 1995;76(9):47C-53C. PMID: 7572686
 37. Byington RP, Evans GW, Espeland MA, et al. Effects of lovastatin and warfarin on early carotid atherosclerosis: sex-specific analyses. Asymptomatic Carotid Artery Progression Study (ACAPS) Research Group. *Circulation*. 1999;100(3):e14-7. PMID: 10411862
 38. Hodis HN, Mack WJ, LaBree L, et al. Reduction in carotid arterial wall thickness using lovastatin and dietary therapy: a randomized controlled clinical trial. *Ann Intern Med*. 1996;124(6):548-56. PMID: 8597317
 39. Raiko JR, Magnussen CG, Kivimaki M, et al. Cardiovascular risk scores in the prediction of subclinical atherosclerosis in young adults: evidence from the cardiovascular risk in a young Finns study. *Eur J Cardiovasc Prev Rehabil*. 2010;17(5):549-55. PMID: 20354441
 40. Bots ML, Palmer MK, Dogan S, et al. Intensive lipid lowering may reduce progression of carotid atherosclerosis within 12 months of treatment: the METEOR study. *J Intern Med*. 2009;265(6):698-707. PMID: 19298496
 41. O'Leary DH, Polak JF, Kronmal RA, et al. Carotid-artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. Cardiovascular Health Study Collaborative Research Group. *The New England journal of medicine*. 1999;340(1):14-22. PMID: 9878640
 42. Johnson HM, Turke TL, Grossklaus M, et al. Effects of an office-based carotid ultrasound screening intervention. *J Am Soc Echocardiogr*. 2011;24(7):738-47. PMID: 21477989
 43. Goff DC, Jr., Lloyd-Jones DM, Bennett G, et al. 2013 ACC/AHA guideline on the assessment of cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;129(25 Suppl 2):S49-73. PMID: 24222018
 44. Greenland P, Alpert JS, Beller GA, et al. 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*. 2010;56(25):e50-103. PMID: 21144964
 45. Jellinger PS, Handelsman Y, Rosenblit PD, et al. American Association of Clinical Endocrinologists and American College of Endocrinology Guidelines for Management of

Dyslipidemia and Prevention of Cardiovascular Disease - Executive Summary.

Endocrine practice : official journal of the American College of Endocrinology and the American Association of Clinical Endocrinologists. 2017;23(4):479-97. PMID: 28156151

46. Lim LS, Haq N, Mahmood S, et al. Atherosclerotic cardiovascular disease screening in adults: American College Of Preventive Medicine position statement on preventive practice. *Am J Prev Med*. 2011;40(3):381.e1-10. PMID: 21335273
47. Using nontraditional risk factors in coronary heart disease risk assessment: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2009;151(7):474-82. PMID: 19805770
48. Curry SJ, Krist AH, Owens DK, et al. Risk Assessment for Cardiovascular Disease With Nontraditional Risk Factors: US Preventive Services Task Force Recommendation Statement. *Jama*. 2018;320(3):272-80. PMID: 29998297

CODES

NOTE: CPT 93880 (duplex scan of extracranial arteries; complete bilateral study) should not be used to identify carotid intima-media thickness studies.

Codes	Number	Description
CPT	93895	Quantitative carotid intima media thickness and carotid atheroma evaluation, bilateral
HCPCS	None	

Date of Origin: April 2002