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Non-Invasive Measurement of Central Blood Pressure (cBP)

Table of Contents

Related Policies (if applicable)
None

Disclaimer

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

Coverage

Non-invasive measurement of central blood pressure (cBP) **is considered experimental, investigational and/or unproven.**

Policy Guidelines

None.

Description

Pressure measured with a cuff and sphygmomanometer in the brachial artery is accepted as an important predictor of future cardiovascular risk. However, systolic pressure varies throughout the arterial tree, such that central aortic blood pressure (cBP), is actually lower than corresponding brachial values, although this difference is highly variable between individuals. Some evidence suggests that central pressure is better related to future cardiovascular events

than is brachial pressure. Moreover, anti-hypertensive drugs can exert differential effects on brachial and central pressure. (1)

Several devices and techniques, each purporting to estimate cBP, have entered commercial use. These devices may allow the noninvasive recording of the arterial waveform and the generation of a proximal aorta pressure profile. The devices when clinically validated in catheterization laboratories and when accurately calibrated, have been shown to be within 1 mm Hg to 2 mm Hg of the actual pressure in the proximal aorta. Pulse waveforms can be obtained either using a tonometer (handheld or stationary), which captures the radial artery waveform, or by oscillometric methods, which use a cuff encircling the limb. Both methods produce a waveform, either from the brachial (oscillometric) or radial (tonometric) arteries, which is usually subjected to a general transfer algorithm to produce a central pressure profile. A typical duration of waveform capture is on the order of 10 seconds. (2)

How is central BP different from conventional BP?

Conventional BP is measured in the upper arm, which is a 'peripheral' artery. Peripheral BP is usually higher than central BP as it includes the increased pressure associated with more and smaller arteries in the arm. The degree to which the peripheral BP is higher than central BP is determined by the stiffness of the arteries.

Central aortic blood pressure (cBP) is the pressure in the aorta, which is the large artery into which the heart pumps blood from the heart throughout the body and represents the pressure to which the vital organs (e.g., heart, kidneys, brain) are exposed. The term 'central blood pressure' usually refers to the pressure in the aorta nearest to the heart. Higher cBP means that the heart must work harder to do its job. This can eventually lead to heart failure. As central blood pressure also determines the pressure in the blood vessels feeding the brain, if central pressure is too high, it may cause aneurysms and strokes. (3)

It is believed that cBP is more precise than blood pressure obtained from the arm with a traditional blood pressure cuff. These findings, including measurement of arterial pulse wave velocity are purported to predict the risk of heart disease or stroke more accurately. Additionally, cBP has been shown to strongly relate to vascular disease and outcomes than that of the traditional upper arm blood pressure. It also can distinguish between the effects of different hypertension medications when upper arm blood pressure and pulse wave velocity do not (1, 4).

The SphygmoCor® products use noninvasive tonometry and computerized calculations to obtain central blood pressure and pulse wave velocity from the radial or carotid arteries. The technology behind these products is said to be centered on an algorithm that derives the pressure wave at the ascending aorta from an external measurement taken at the radial artery. (5, 6)

Regulatory Status

- In August 2007, the SphygmoCor® CvMS (cardiovascular management system), was cleared by the U.S. Food and Drug Administration (FDA; K070795). (7)
- In November 2012, the SphygmoCor® XCEL System (K122129; AtCor Medical) was cleared by the FDA. (8)
- In March 2018, the PhysioWave Cardiovascular Analyzer (K172431) was FDA approved under the 510(k) pathway. (9) The PhysioWave is intended to obtain pulse wave velocity (PWV) and pulse rate through a combination of impedance plethysmography and weight measurements in adults 18 years of age and older. The PhysioWave also measures body weight and calculates BMI.

Refer to <https://www.fda.gov> for additional FDA approved devices.

Rationale

Sharman et al. noted that several methodological issues remain to be addressed before measurement of central pressure is fully integrated into clinical decision making and of practical benefit for patients. (10) Firstly, although a number of simple-to-use reliable devices are now on the market, a standard approach to validation of new devices is required. This approach can sometimes produce higher central pressure estimates than the measured brachial cuff pressure, which may seem unphysiological, but is due to the brachial cuff giving a falsely low estimate of brachial systolic pressure. The alternative approach calculates central pressure relative to the measured brachial cuff pressure, which tends to under-estimate the 'true' aortic pressure but may be more intuitive. The authors also commented that there is a need to adopt a standard method for calibrating peripheral waveforms, using either systolic/diastolic or mean arterial pressure (MAP)/diastolic pressure, and to better understand the impact of brachial-radial and aortic-carotid amplification.

The related study found that noninvasive central BP information helped in the management of patients with hypertension. Significantly less antihypertensive medication was used to maintain appropriate BP control, and quality of life was improved to the same degree as best-practice usual care. Despite significant withdrawal of medication, there was a trend toward lower left ventricular mass (LVM) than those treated according to usual care, which was unexpected and merits further investigation because this could be a clue toward helping to understand why intensive BP lowering may increase risk in some patients. The maintenance of good out-of-office BP control using less antihypertensive medication suggests that central BP monitoring may be especially valuable in patient populations where there may be a risk of promoting harmful outcomes by pursuing low BP targets, such as in the elderly where fall risk may be increased. Overall, the findings provide extra impetus to undertake large, hardened point trials on the efficacy of central BP assessment in hypertension management. (10)

Arm cuff blood pressure (BP) may overestimate cardiovascular risk. Central aortic BP predicts mortality and could be a better method for patient management. The study sought to determine the usefulness of central BP to guide hypertension management. This was a

prospective, open-label, blinded–end point study in 286 patients with hypertension randomized to treatment decisions guided by best-practice usual care (n=142; using office, home, and 24-hour ambulatory BP) or, in addition, by central BP intervention (n=144; using SphygmoCor). Therapy was reviewed every 3 months for 12 months, and recommendations were provided to each patient and his/her doctor on antihypertensive medication titration. Outcome measures were as follows: medication quantity (daily defined dose), quality of life, and LVM (3-dimensional echocardiography). There was 92% compliance with recommendations on medication titration; quality of life improved in both groups (post hoc $P<0.05$). For usual care, there was no change in daily defined dose (all $P>0.10$), but with intervention there was a significant stepwise decrease in daily defined dose from baseline to 3 months ($P=0.008$) and each subsequent visit (all $P<0.001$). Intervention was associated with cessation of medication in 23 (16%) patients versus 3 (2%) in usual care ($P<0.001$). Despite this, there were no differences between groups in LVM, 24-hour ambulatory BP, home systolic BP, or aortic stiffness (all $P>0.05$). The study concluded that guidance of hypertension management with central BP results in a significantly different therapeutic pathway than conventional cuff BP, with less use of medication to achieve BP control and no adverse effects on LVM, aortic stiffness, or quality of life. (10)

In a 2014 clinical update review the European Heart Journal (11) noted: "There is now a substantial body of evidence that antihypertensive drugs, and particularly beta-blockers, exert differential effects on brachial and central pressure. As a result, the pharmaceutical industry is becoming increasingly convinced that basing treatment decisions on central, rather than brachial pressure, is likely to have important implications for the future diagnosis and management of hypertension. However, cuff measurements of brachial systolic and diastolic pressure continue to remain the accepted surrogates by drug regulatory authorities. This means that new therapies will continue to be assessed on the basis of brachial measurements, which may ultimately serve as a potential barrier to novel drug development. Therefore, appropriately powered clinical trials demonstrating that preferential lowering of central pressure improves outcome, will ultimately be required before central pressure becomes an accepted surrogate of cardiovascular risk. Nitrovasodilating drugs may be particularly useful in this respect. Before such trials are completed, smaller studies based on established surrogates for cardiovascular disease, such as carotid intima media thickness (IMT) and LVM will be important in providing proof of principle that reduction in central rather than brachial pressure is a more effective therapeutic strategy."

Narayan et al. (12) performed a systematic meta-analysis of studies reporting cBP between 2000 and 2012. Studies were included if both central and brachial blood pressure (cBP and bBP) were reported. Studies were categorized by technique and according to the prevalent disease state with the bBP - cBP difference calculated. Random-effects modeling (inverse variance weighted approach) was used to estimate the pooled mean difference associated with each technique. Of the 164 eligible studies, the SphygmoCor device was most commonly reported (110 studies); with direct carotid applanation second-most utilized (31 studies). In 30 included invasive cohorts, the measured cBP did not differ significantly from the oscillometric bBP recorded [mean difference 4.19 mmHg, 95% confidence interval (CI) -4.13 to 12.51], whereas

mean differences of 12.77 mmHg (95% CI 11.93, 13.60) and 8.83 mmHg (95% CI 7.86, 9.79) were obtained with the SphygmoCor and carotid applanation estimates of cBP, respectively (both $P < 0.05$). Conversely, the reported mean cBP-to-bBP differences measured across various disease states with SphygmoCor did not differ significantly. This meta-analysis suggests that noninvasive cBP estimation is device/technique dependent. Consequently, caution is advisable in applying these devices and techniques across clinical studies.

Townsend and colleagues were part of a panel of clinical researchers and clinicians who study and clinically use pulse wave analysis. (2) This panel was assembled to discuss strategies for using pulse wave analysis in the clinical encounter. The article presents an approach to the clinical application of pulse waveform analysis, how to interpret central pressure waveforms, and how to use existing knowledge about the pharmacodynamic effect of antihypertensive drug classes in combination with brachial and central pressure profiles in clinical practice. The discussion in the article was supplemented by case-based examples provided by panel members, which the authors hope will provoke discussion on how to understand and incorporate pulse wave analysis into clinical practice. The intent was not to recommend replacing brachial pressure with central pressure in the management of BP. The authors go on to note: “such a replacement is not yet supported by sufficient evidence from randomized clinical trials. Our intent was to determine how information from the central pressure and the analysis of the central pressure waveform provides additional information to physicians managing BP beyond current brachial BP goals.”

Rinaldi et al. (13) noted in a 2015 article that emerging evidence now suggest that central pressure may predict cardiovascular diseases better than brachial BP; moreover, it may differently respond to certain antihypertensive drugs. The potential effects beyond peripheral BP control may be due to specific protective properties of different antihypertensive drugs in affecting central aortic pressure and arterial stiffness. Although data on direct cardiovascular benefit impact of cBP treatment in randomized clinical trials are still lacking, it is likely that the improvement of quality of care and the individualized assessment of the hypertension-associated cardiovascular risk are achievable with the use of central hemodynamics. Therefore, basing antihypertensive treatment guidance on central pressures rather than on peripheral blood pressure may be the key for future antihypertensive strategies.

Borlaug et al. (14) sought to determine whether aggressive titration of vasoactive medicines beyond goal-directed heart failure medical therapy (GDMT) based upon aortic pressure improves exercise capacity and cardiovascular structure-function. Subjects with chronic heart failure (HF) (n=50) underwent cardiopulmonary exercise testing, echocardiography, and arterial tonometry to measure aortic pressure and augmentation index and were then randomized to aortic pressure-guided treatment (active, n=23) or conventional therapy (control, n=27). Subjects returned for 6 monthly visits wherein GDMT was first optimized. Additional vasoactive therapies were then sequentially added with the goal to reduce aortic augmentation index to 0% (active) or if brachial pressure remained elevated (control). Subjects randomized to active treatment experienced greater improvement in peak oxygen consumption compared with controls (1.37±3.76 versus −0.65±2.21 mL min−1 kg−1, P=0.025) though reductions in aortic

augmentation index were similar (−7±9% versus −5±6%, P=0.46). Forward stroke volume increased while arterial elastance and left ventricular volumes decreased in all participants, with no between-group difference. Subjects randomized to active treatment were more likely to receive additional vasoactive therapies including nitrates, aldosterone antagonists and hydralazine, with no increased risk of hypotension or worsening renal function. Maximization of goal-directed medical therapy in heart failure patients may enhance afterload reduction and lead to reverse remodeling, while additional medicine titration based upon aortic pressure data improves exercise capacity in patients with heart failure.

In 2016, Cheng et al. (15) evaluated the prognostic value and clinical utilities of pulse wave analysis (PWA) derived mechanical biomarkers in two independent population-based cohorts. PWA on central arterial pressure waveforms were obtained from subjects without a prior history of cardiovascular diseases. The two studies were the Kinmen study (1272 individuals, a median follow-up of 19.8 years); and the Cardiovascular Disease Risk Factors Two-Township Study (CVDFACTS) (2221 individuals, median follow-up of 10 years). In the Kinmen study, right carotid artery pressure waveforms, which have been demonstrated to closely resemble central aortic pressure waveforms, were registered noninvasively with a tonometer. In the CVDFACTS study, central aortic pressure waveforms were obtained with a SphygmoCor device using radial arterial pressure waveforms. The associations between all mechanical biomarkers derived from pulse wave analysis and cardiovascular mortality were then examined in the multivariate Cox proportional hazards models that took into account cardiovascular risk factors including age, sex, systolic BP, body mass index, fasting glucose, triglycerides, low-density-lipoprotein cholesterol, and high-density-lipoprotein cholesterol, and smoking. Only systolic rate constant (SC) and diastolic rate constant (DC) of reservoir pressure could independently and consistently predict cardiovascular mortality in both cohorts. Cardiovascular mortality was higher in the Kinmen study due to higher hypertension prevalence and more male participants. During a median follow-up of 19.8 years, 315 (26.9%) deaths occurred (84 of cardiovascular origin). In the CVDFACTS study, a total of 171 deaths occurred (34 of cardiovascular origin) during a median follow-up of 10 years. Increased brachial systolic BP, pulse pressure, backward wave amplitudes (Pb), and augmentation index (AI) were significantly associated with increased cardiovascular mortality in both studies. Biomarkers derived from reservoir pressure-wave analysis were positively associated with cardiovascular mortality in the Kinmen study, and in the CVDFACTS study, only peak of reservoir pressure and DC remained significant in predicting cardiovascular mortality. The authors concluded that these findings suggested that mechanical biomarkers derived from pulse wave analysis could not only independently predict the long-term cardiovascular risks beyond the traditional risk factors, but also provide more accurate risk stratification by incorporating these mechanical biomarkers into the risk prediction models. It is not clear how this information will affect patient management and outcomes.

It has been hypothesized that the central aortic blood pressure (cBP) waveform may be used for non-invasive estimation of the intracranial pressure (ICP) waveform. Simultaneous invasive ICP and radial artery BP waveforms were measured in 29 individuals with idiopathic normal pressure hydrocephalus (iNPH). The central aortic BP waveforms were estimated from the radial artery BP waveforms using the SphygmoCor system. For each individual, a transfer

function estimate between the central aortic BP and the invasive ICP waveforms was found (Intra-patient approach). The patient specific transfer functions were further utilized to find individual ICP estimates for each patient. A time domain analysis of the estimated ICP compared to the invasive ICP signals found that the estimates correctly predicted the most important clinical parameter mean wave amplitude (MWA) in about 2 of 29 cases. This indicates that the method has some potential, but that there are large uncertainties. For the method to have significant clinical value it should be possible to estimate ICP signals without first measuring invasive ICP signals. This was achieved by using the transfer function estimate that gave the best cross-correlation between the estimated ICP and measured ICP on the total cohort of 29 individuals. The resulting ICP estimates correctly predicted the MWA parameter within the necessary range in 8 out of the 29 cases. However, they did not reproduce the invasive MWA threshold. The quality of the results were too varied thus these results are inadequate for central aortic BP-derived non-invasive ICP estimates to be used in the clinical setting. However, the method does show some promise regarding utility of the central aortic BP waveform to predict the ICP waveform. The assumption of a linear system linking central aortic BP to ICP seems to be too simplistic and the model should be expanded to incorporate more of the complexity of the system. Further studies should therefore be performed to determine the future clinical possibilities of this approach. (16)

Grillo et al. (2018) explored the consistency of aortic pulse wave velocity (PWV) as an indirect index of arterial stiffness and an independent cardiovascular risk factor. (17) Since studies providing a comparative estimate of the reproducibility of PWV across different noninvasive devices are lacking, the authors aimed to fill this gap using 6 different devices (Complior Analyse, PulsePen-ETT, PulsePen-ET, SphygmoCor Px/Vx, BPLab, and Mobil-O-Graph). These devices were evaluated in 102 high cardiovascular risk patients hospitalized for suspected coronary artery disease (72 males, 65 ± 13 years). PWV was measured in a single session twice, at 15-minute intervals, and its reproducibility was assessed though coefficient of variation (CV), coefficient of repeatability, and intraclass correlation coefficient. The CV of PWV, measured with any of these devices, was <10%. Repeatability was higher with cuff- based methods (BPLab: CV = 5.5% and Mobil-O-Graph: CV = 3.4%) than with devices measuring carotid-femoral PWV (Complior: CV = 8.2%; PulsePen-TT: CV = 8.0%; PulsePen- ETT: CV = 5.8%; and SphygmoCor: CV = 9.5%). In the latter group, PWV repeatability was lower in subjects with higher carotid-femoral PWV. The differences in PWV between repeated measurements, except for the Mobil-O-Graph, did not depend on short-term variations of mean blood pressure or heart rate. This study shows that the short-term repeatability of PWV measures is good but not homogenous across different devices and at different PWV values.

Motau et al. (2018) tried to determine the extent to which relations between modifiable risk factors and aortic function translate into increases in central aortic pulse pressure. (18) In 1232 black South Africans, they determined risk factors and aortic function from carotid-femoral PWV and aortic central pulse pressure, forward wave pressures (Pf) and reflected (backward-Pb) wave pressures (applanation tonometry and SphygmoCor software). With adjustments for alternative risk factors and distending pressure (mean arterial pressure [MAP]), diabetes mellitus (treatment or HbA1c >6.5%, n=151) was associated with an increased PWV (7.10±2.09

versus 6.17±2.00 m/sec, p<0.0001), and Pf (26±8 versus 24±8 mm Hg, p<0.005), but neither brachial PP (46±14 versus 45±13, p=0.19), central aortic pulse pressure (36±12 versus 35±11 mm Hg, p=0.48), nor Pb (17±6 versus 17±6 mm Hg, p=0.83). Moreover, independent of alternative risk factors and MAP, uncontrolled hypertension (office BP>140/90 mm Hg, n=433), was associated with an increased Pf (26±12 versus 24±10 mm Hg, p<0.01), but not with changes in brachial PP (45±19 versus 44±17, p=0.75), PPc (35±16 8 versus 35±15 mm Hg, p=0.93) or Pb (18±8 versus 17±8 mm Hg, p=0.46). The authors concluded that neither brachial nor aortic pulse pressures are adequate indexes of relation between the modifiable conventional risk factors, uncontrolled hypertension or diabetes mellitus, and risk-related aortic functional changes.

Milan et al. (2019) reviewed different validation studies of PWV estimation techniques and assessed their conformity to the Artery Society Guidelines and the American Heart Association recommendations. (19) Several devices had been developed and validated to noninvasively measure arterial stiffness, using applanation tonometry (SphygmoCor, PulsePen), piezoelectric mechanotransducers (Complior), cuff-based oscillometry (Arteriograph, Vicorder and Mobil-O-Graph), photodiode sensors (pOpmètre) and devices assessing brachial-ankle PWV and cardiac-ankle PWV. Ultrasound technique and MRI remain confined to clinical research. In Arteriograph, MRI, ultrasound and SphygmoCor Xcel validation studies sample size was smaller than the minimum suggested by the guidelines. High discrepancies between devices were shown in distance estimation: in 2 studies (Arteriograph, Complior) path length was estimated in conformity to the guidelines. Transit time was calculated using the intersecting tangent method, but in 2 studies (Vicorder, pOpmètre) best agreement was found using the maximum of the second derivative. Six studies reached the accuracy level 'excellent' defined in the Artery guidelines. The authors concluded that the method to assess transit time and path length needs validation in larger populations. Further studies are required in different risk population(s) to implement clinical applicability of every device.

Summary of Evidence

Evidence is required to clarify the interchangeability of central blood pressure (cBP) measurements between noninvasive devices and the influence of disease states on central to brachial pulse pressure amplification. The clinical evidence from multi-center clinical trials demonstrating the use of this technology alters patient management and improves clinical outcomes is lacking. Additional research involving larger, well-designed controlled studies are needed to establish the role of cBP, evaluate how the use of cBP alters patient management and the impact of health outcomes.

Coding

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

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

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

CPT Codes	93050
HCPCS Codes	None

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

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Centers for Medicare and Medicaid Services (CMS)

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

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

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

Policy History/Revision

Date	Description of Change
02/15/2025	Reviewed. No changes.
03/15/2024	Document updated with literature review. No change in Coverage. Added reference 1-3, 5-9, 17-19. Others updated, some removed.
03/15/2023	Reviewed. No changes.

05/15/2022	Document updated with literature review. Coverage unchanged. Reference 1 added.
02/15/2021	Reviewed. No changes.
05/15/2020	Document updated with literature review. Coverage unchanged. Reference 8 added.
07/15/2018	Reviewed. No changes.
10/15/2017	Document updated with literature review. Coverage unchanged.
12/01/2016	Reviewed. No changes.
05/01/2016	New medical document. Non-invasive measurement of central blood pressure is considered experimental, investigational and/or unproven.